

BAILEY & LOVE'S SHORT PRACTICE OF SURGERY

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CONTENTS

	PAGE
PREFACE	vii
1. ACUTE INFECTIONS AND WOUNDS	I
2. CHRONIC INFECTIONS, VENEREAL DISEASES, PARASITES	22
3. ULCERATION AND GANGRENE	36
4. TUMOURS AND CYSTS	49
5. HÆMORRHAGE, BLOOD TRANSFUSION, SHOCK	65
6. FLUID, ELECTROLYTE AND ACID-BASE BALANCE	82
7. SKIN, BURNS, SKIN GRAFTING	98
8. ARTERIES AND VEINS	117
9. LYMPHATICS AND LYMPH NODES	151
10. INFECTIONS OF THE HAND AND FOOT	160
11. INJURIES TO BONES	176
12. DISEASES OF BONES	230
13. INJURIES TO JOINTS	265
14. DISEASES OF JOINTS	279
15. MUSCLES, TENDONS AND BURSÆ	305
16. DEFORMITIES	320
17. HEAD	351
18. SPINE	402
19. NERVES	428
20. FACE, LIPS, PALATE, EAR	451
21. TEETH, GUMS, JAWS, NOSE, ORBIT	464
22. MOUTH, TONGUE, CHEEK, SALIVARY GLANDS	486
23. NECK	518
24. THYROID AND THYROGLOSSAL TRACT	537
25. PARATHYROID GLANDS, THYMUS, ADRENAL GLANDS	568
26. BREAST	587
27. PHARYNX	617

CONTENTS

xi

CHAPTER	PAGE
28. LARYNX	633
29. THORAX	644
30. HEART AND PERICARDIUM	686
31. ŒSOPHAGUS	708
32. STOMACH AND DUODENUM	728
33. SPLEEN	773
34. LIVER	791
35. GALL-BLADDER AND BILE DUCTS	818
36. PANCREAS	846
37. PERITONEUM	867
38. INTESTINES	892
39. INTESTINAL OBSTRUCTION	926
40. VERMIFORM APPENDIX	953
41. RECTUM AND ANAL CANAL	975
42. UMBILICUS AND THE ABDOMINAL WALL (EXCLUDING HERNIA)	1023
43. HERNIA	1031
44. URINARY SYMPTOMS. INVESTIGATION OF THE URINARY TRACT. ANURIA	1061
45. KIDNEYS AND URETERS	1074
46. URINARY BLADDER	1121
47. PROSTATE AND SEMINAL VESICLES	1160
48. URETHRA AND PENIS	1183
49. TESTES AND SCROTUM	1213
INDEX	1241

A SHORT PRACTICE OF SURGERY

CHAPTER I

ACUTE INFECTIONS AND WOUNDS

INFECTION is the invasion of the body by pathogenic micro-organisms. For this to happen, a sufficient number of pathogens must enter the tissues, and the resistance of the patient must be low enough to allow them to multiply and invade. General factors which lower resistance include uncontrolled diabetes, steroid therapy, agranulocytosis, and hypogammaglobulinæmia. Local factors of importance are hæmatoma formation, crushing of tissues in injury or at operation, bone injury and foreign bodies.

PRINCIPAL ORGANISMS OF SEPSIS

These are the *Staphylococci*, *Streptococci*, *Escherichia coli*, *Proteus*, and *Pseudomonas pyocyanea*.

GRAM-POSITIVE COCCI

Staphylococcus aureus

Gram-positive cocci in clusters; golden colonies; forms coagulase which clots plasma.



FIG. 1

Habitat and Spread

Lives in nostrils of 30 per cent. of population (nasal carriers), and sometimes in skin. Present in infected discharges. **Spread.** By indirect contact, on hands, etc. Airborne spread also by garment dust and skin squames.

Principal Infections

Boils, styes, carbuncles, septic hands, breast abscess, wound infection. Ac. osteomyelitis, septicæmia, post-operative pneumonia and enterocolitis. Mainly local infections.

Antibiotic Sensitivity

(a) **Outside Hospital.** 60 per cent. are penicillin sensitive. The rest form penicillinase and are resistant. (b) **In Hospital.** 80–90 per cent. are penicillin resistant, also to streptomycin, tetracycline and erythromycin. Most penicillin-resistant strains are sensitive to cloxacillin and methicillin.

Streptococcus (fig. 2)

Gram-positive cocci in chains. Many varieties; some are hæmolytic. Group A hæmolytic streptococci (*Str. pyogenes*) are highly pathogenic.

Str. faecalis and anaerobic streptococci live in bowel.

Group A strep. live in pharynx of 5–10 per cent. of population.

Spread. By indirect contact, respiratory droplets and dust.

Group A strep. cause—tonsillitis, otitis, sinusitis, scarlet fever. Dangerous in wounds, puerperal sepsis, cellulitis, erysipelas (rare nowadays). Infections tend to spread along cellular planes.

Group A strep. are penicillin sensitive; many are resistant to tetracycline.



FIG. 2

INTESTINAL GRAM-NEGATIVE BACILLI (fig. 3)


<i>Escherichia coli</i>	<i>Habitat and Spread</i>	<i>Principal Infections</i>	<i>Antibiotic Sensitivity</i>
Gram-negative rods, motile.	Normal inhabitant of large bowel.	Wounds especially in or near colon, burns, urinary tract. Contains endotoxins which cause pyrexia, rigors, and shock if large numbers enter the blood stream (Catheter fever); also causes shock in peritonitis.	
<i>Pseudomonas pyocyanea</i>	Likes to colonise moist superficial lesions which form its principal habitat.	Contains endotoxins. An opportunist which attacks damaged tissues and moist surfaces such as burns, ears, urinary tract, tracheostomy wounds. In severely ill patients it may be deadly.	All are penicillin resistant—some are sensitive to ampicillin (not <i>Ps. pyocyanea</i>). Resistance to other drugs varies—hospital cross-infecting strains become resistant to nearly all drugs. Polymyxin useful against <i>Ps. pyocyanea</i> .
<i>Proteus</i>	Widely distributed in man's environment.	Contains endotoxins. Splits urea into NH_3 and CO_2 making urine alkaline so that phosphatic deposits occur.	

FIG. 3

Pneumococci are Gram-positive diplococci which cause lobar pneumonia and, together with other organisms, bronchopneumonia. Pneumococci may also cause otitis media, sinusitis, and meningitis. In young girls they occasionally cause acute peritonitis, probably entering via the Fallopian tube (Chapter 37). Pneumococci are invariably sensitive to penicillin.

Hæmophilus influenzae is a small Gram-negative bacillus. Although often present in the healthy upper respiratory tract, *H. influenzae* is the commonest pathogen in purulent bronchitis, and, as such, frequently predominates in the sputum of elderly patients with post-operative chest infection. It does *not* cause influenza. *H. influenzae* is resistant to penicillin, but sensitive to streptomycin, tetracycline and ampicillin.

Salmonella typhi and *S. paratyphi* cause enteric fever in which the blood-stream is invaded. Following enteric fever the organisms may persist in the biliary passages, the intestinal lymphoid tissue, or the kidneys; and the patient is then a 'carrier.' *Salmonellæ* occasionally cause septicæmia, osteitis, and other suppurative lesions.

ANTIBACTERIAL THERAPY

Antibacterial drugs are chemicals which damage bacteria without harming the tissues. They consist of two types (1) **chemotherapeutic agents** (synthetic drugs) such as the sulphonamides and (2) the **antibiotics** which are the products of other microbes. For many years attempts were made to produce a therapeutically useful antibiotic, but it was not till 1941 that Florey produced a reliable and stable preparation of penicillin suitable for clinical purposes. Penicillin had been originally discovered

by Fleming in 1928. Innumerable antibiotics have since been isolated and their numbers steadily increase, but only a comparatively few are useful chemotherapeutically due to the toxic properties of the majority.

CHEMOTHERAPEUTIC AGENTS

Sulphonamides inhibit the growth of many bacteria, but are partially neutralised by substances present in pus, blood, and the tissues. Sulphonamides therefore are most useful in infections where tissue inhibitors are absent—e.g. coliform infections of the urinary tract, meningococcal meningitis, and bacillary dysentery. The most useful sulphonamides are *sulphadiazine* and *sulphadimidine*; both give good blood levels and few toxic effects. They are specially valuable in urinary and cerebrospinal infections respectively. *Succinylsulphathiazole* or *phthalylsulphathiazole* are used mainly in an attempt to sterilise the bowel before surgery. In urinary infections, therapy should continue for weeks or months. The most important toxic effect is sensitisation appearing as a skin rash with or without fever. Deposition of crystals in the urinary tract has been reduced by using more soluble preparations and by insisting on an adequate urinary output. Sulphonamides should not be used for local application.

Nalidixic acid ('Negram') and **Nitrofurantoin** ('Furadantin') are given orally in the treatment of *Esch. coli* and some *Proteus* infections of the urine.

Isonicotinic acid and **Para-amino-salicylic acid** (p. 23).

ANTIBIOTICS

Some antibiotics, notably penicillin, streptomycin, and kanamycin are *bactericidal*, and kill susceptible bacteria. Others, e.g. tetracycline, chloramphenicol, novobiocin are *bacteriostatic* and merely inhibit the growth of bacteria. When leucocytes can enter the lesion they complete the destruction of the microbes.

Penicillin, unlike sulphonamides, retains its activity in pus and blood. It acts best on multiplying organisms and hence may be antagonised by simultaneously-administered bacteriostatic agents. Penicillin is especially valuable in infections by *Str. pyogenes*, pneumococci, non-penicillinase-producing staphylococci, *B. anthracis*, and clostridia of gas-gangrene. It is the treatment of choice for actinomycosis, syphilis, and gonorrhoea. Penicillin-sensitive bacteria rarely acquire resistance, though they may be replaced by resistant strains. However, some gonococci have now become relatively penicillin-resistant.

Many modifications of the penicillin molecule are now available. **Benzyl penicillin** (penicillin G), administered by injection, is still the most potent, but is destroyed by staphylococcal penicillinase and hence is ineffective against many staphylococci. **Procaine penicillin** is a slow release modification of benzyl penicillin. **Penicillin V** is acid-stable and given orally for relatively mild infections. The semi-synthetic penicillins, **methicillin**, and **cloxacillin** are unique in being insensitive to penicillinase and hence of great value in treating penicillin-resistant staphylococcal infections. This is their only valid use, since they are less potent than benzyl penicillin against other organisms. **Ampicillin** is a broad-spectrum penicillin, active against *Esch. coli* and some other gram-negative bacilli.

The main disadvantage of the penicillins is their tendency to provoke allergic reactions such as skin rashes, vomiting, and occasionally anaphylaxis (p. 11).

Streptomycin, given by injection, is active against tubercle bacilli, staphylococci, and gram-negative bacilli. If given for urinary infection, the urine should be made alkaline. Streptomycin has two disadvantages (i) organisms readily become resistant (ii) prolonged administration or excess dosage damage the eighth cranial nerve. The risk of toxicity is increased after the age of forty-five and by renal insufficiency. Streptomycin must not be given if the blood urea is raised.

The tetracyclines are broad-spectrum antibiotics, with almost identical actions. They are generally active against staphylococci and streptococci, gram-negative cocci, coliform bacilli, *H. influenzae*, actinomyces, and lymphogranuloma venereum. Many staphylococci, streptococci, and coliforms have now acquired resistance to tetracyclines. Administration of these drugs disturbs the intestinal flora and may cause digestive upset. An occasional, but serious complication is enterocolitis due to superinfection of the bowel by tetracycline-resistant staphylococci (p. 5).

If tetracyclines are given for more than a few days, vitamin B should also be given.

Chloramphenicol has a similar range of action as the tetracyclines. It is the drug of choice in enteric fever but, owing to the risk of blood dyscrasia, should rarely be given otherwise.

Polymyxin B and Colistin ('colomycin') are very similar drugs, sometimes valuable in infections by *Ps. pyocyanea*.

Erythromycin is a valuable non-toxic antibiotic mainly active against gram-positive bacteria. Staphylococci readily become resistant to it.

Neomycin and Bacitracin are useful for local application to superficial infections, but are too toxic for injection. Neomycin given orally is not absorbed, but is an effective intestinal antiseptic, though it may lead to antibiotic enterocolitis (p. 5).

USE AND SELECTION OF ANTIBACTERIAL DRUGS

It is important that these drugs are used with discretion and only when the sensitivity of the organism is known. Indiscriminate use, as in the 'antibiotic umbrella', may lead to the destruction of the normal flora and the persistence of resistant organisms which may then assume a pathogenic role. Even when the treatment of a serious or acute infection has to be started without delay, the bacteriologist's advice should subsequently be sought. New drugs should be used with caution, so as to reduce the selection of resistant strains. In general, if there is no improvement in three to four days, the treatment should be stopped or changed. If the infection responds, it is usually unnecessary to continue for more than three to four days after the temperature becomes normal, but long and intensive treatment is needed in special circumstances, e.g. osteomyelitis, actinomycosis, and tuberculosis.

ADMINISTRATION OF ANTIBIOTICS

Care must be taken with intramuscular injections or damage will be done to deep structures. The best site is the upper and outer quadrant of the buttock (fig. 4), or the antero-lateral aspect of the thigh.

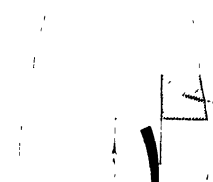


FIG. 4.—'Safe' area for intramuscular injection to avoid the sciatic nerve.

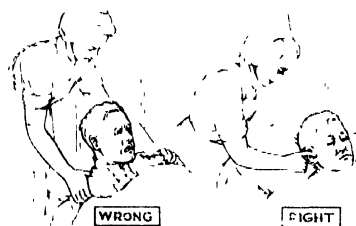


FIG. 5.—Wakening a sleeping person. (As used by North American Indians for centuries.)

If it is necessary to arouse a patient for therapy or other causes, gradually increasing pressure on the tragus should be applied, until the patient awakens, often with a smile! Shaking the patient awakens him with a 'jump', and the resulting irritability is not conducive to an immediate return to sleep (fig. 5).

HOSPITAL INFECTION

It is important to remember that resistant pathogenic organisms can be lurking in hospital wards, corridors, and even theatres. Superficial and deep wounds may become infected by organisms from the patient (self-infection) or by cross-infection from other patients. The most important hospital organisms are *Staph. aureus*; Gram-negative bacilli and the bacilli of gas-gangrene and tetanus (p. 7). Hæmolytic streptococci are much less common causes than in the pre-antibiotic era.

Tissue damage, hæmatomas, and foreign bodies in operation wounds increase the risk of sepsis. Sepsis in undrained, sutured wounds is usually the result of infection during operation. Drained and open wounds may be infected either at operation or later in the wards. Some theatre infections occur by self-infection; others by cross-infection from a member of the staff. A symptomless carrier may be responsible; but persons with septic

lesions are especially dangerous sources and should not enter the operation room.

Staphylococci

Ward infection does not always cause sepsis; staphylococci may merely colonise an already discharging wound. Wound discharges containing staphylococci are sources of cross-infection. Other important sources are staphylococcal infections of urine, skin, lung, bowel, and the noses of carriers among patients and staff. Superficial lesions such as varicose ulcers, eczema, and bed sores may also become sources, if colonised by staphylococci. Ward staphylococci may be conveyed to the theatre and there cause infection. Staphylococcal infection is a dangerous complication of cardiac surgery, and is sometimes caused by *Staph. albus* which ordinarily is non-pathogenic. Staphylococci may cause other post-operative complications—e.g. staphylococcal bronchopneumonia, urinary infection, parotitis, furunculosis, and enterocolitis.

Staphylococcal enterocolitis is fortunately uncommon. It is a severe (staphylococcal cholera) 'super-infection' of the gut by drug-resistant staphylococci usually following the administration of a broad spectrum antibiotic for bowel surgery. The sudden onset of profuse cholera-like diarrhoea with dehydration and prostration is the usual feature, and it may be rapidly fatal. A smear of faeces will probably show clusters of staphylococci and culture will yield enormous numbers. (Normal faeces often contain a few staphylococci.) Treatment is urgent and consists of replacement of fluid and electrolytes. The antibiotic should be changed to one effective against the causative staphylococcus (e.g. cloxacillin or chloramphenicol if appropriate). On account of this dangerous complication many surgeons use only succinyl-sulphathiazole for preparation of the bowel before colon surgery. This drug is not likely to give rise to trouble of this nature.

Gram-negative Bacilli.—Hospital infections by *Esch. coli*, *Proteus* and *Ps. pyocyanea*¹ have been increasing recently because some other organisms have been diminished by antibiotics. Some Gram-negative bacilli are favoured by certain surgical procedures. Thus tracheostomy wounds are peculiarly liable to infection by *Ps. pyocyanea* (though sometimes without much harm) and hence this opportunistic pathogen has become a problem in intensive-care wards, just as it has long been in urological wards and burns units. Gram-negative bacilli commonly cause sepsis in abdominal wounds. They infect the urinary tract after catheterisation and operation; the organisms may be introduced by inadequately disinfected cystoscopes, inept catheterisation, and open draining indwelling catheters.

Tetanus and Gas-gangrene (p. 44).—The spores of these organisms are often present in soil, dust, unbleached wool, and in the bowel. They are very resistant to heat and chemicals. These infections are the principal dangers from faulty heat sterilisers. Gas-gangrene may also be caused by the patient's own intestinal clostridia after lower-limb amputation, especially when resistance is reduced by diabetes and arteriosclerosis.

PREVENTION OF HOSPITAL INFECTION

Prevention mainly consists of reducing the *sources* of pathogens and restricting their spread. The breeding of resistant strains should be minimised by avoiding unnecessary

¹ *Pyocyaneus* = Blue pus.

antibiotics. Wholesale prophylactic antibiotic treatment defeats its object, but penicillin cover is sometimes justified, e.g. when vascular disease or diabetes predispose to gas-gangrene, and in the protection of tetanus patients from pneumonia.

Much attention to detail is required to carry these precepts into practice. No single precaution will succeed on its own. Sources are reduced by isolating patients with drug-resistant staphylococcal infections and by careful dressing of wounds.

Newborn infants may be dusted or bathed with hexachlorophene preparations to restrict staphylococcal colonisation of umbilicus and skin, and so diminish the incidence of pustules in infants and breast abscesses in mothers.

The spread of pathogens is diminished by general hygienic measures, including avoidance of overcrowding and good ventilation. Hands should be washed with soap and dried with disposable paper towels (*wet hands are dangerous*). When patients leave hospital, or more often if necessary, bedding may be disinfected by a suitable laundering process. Bath tubs should be disinfected with hypochlorite after use.

Infected discharges must be disposed of safely. In urological units, urinals should be disinfected after use; drainage vessels for indwelling catheters should contain disinfectant or incorporate a non-return valve.

Special care must be taken to exclude bacteria from susceptible tissues. Closed sterile bladder drainage prevents bacteria from ascending indwelling catheters (Chap. 46). Post-operative wounds should be left untouched as far as possible. When it is necessary to remove stitches or a tube, a meticulous non-touch technique must be followed. Before removal of stitches, the skin and the external portion of the stitch must be disinfected with an adequate skin antiseptic such as 70 per cent. alcohol.

The correct *heat-sterilisation* of instruments and dressings is sufficiently complicated to deserve skilled supervision in central sterile supply departments and operating theatres. Unbleached cotton-wool, sometimes used to pad splints, may contain tetanus spores and should never be used unless satisfactorily sterilised. Instruments which are damaged at high temperatures present special problems. Non-boilable cystoscopes may usually be 'pasteurised' in water-baths at 176° F. (80° C.) to kill non-sporing organisms; the manufacturers' advice on this matter should be sought.

At operation, the risk of self-infection is minimised by efficient disinfection of the patient's skin with 1 per cent. iodine or 0.5 per cent. chlorhexidine in 70 per cent. alcohol; the latter is less likely to cause sensitisation. Oral sulphonamide may be given before operations on the bowel. The risk of infection from the surgeon's skin is reduced by hand-washing with liquid hexachlorophene soap, rejection of pricked gloves, and the use of gowns with siliconised waterproof sleeves. Members of staff with septic lesions should not enter the theatre. Air contamination is minimised by restricting unnecessary movement of staff, entry of unnecessary visitors, and by ensuring that outdoor clothes are replaced by freshly-laundered theatre garments and over-shoes. Masks must be changed at the end of the session or more often, and should *never* be handled while worn. Airborne bacteria may be further reduced in new or refitted theatres by air-conditioning systems giving frequent changes of suitably humidified, filtered air at a positive pressure.

Finally, since even the best precautions cannot exclude all bacteria from wounds, it is most important not to reduce the resistance of tissues by rough handling, unnecessary drainage, and inadequate hæmostasis.

ACUTE SPECIFIC¹ INFECTIONS

ERYSIPELAS

Erysipelas is a spreading inflammation of the skin and subcutaneous tissues, due to infection by *Str. pyogenes*. The general health of the patient is usually below par, and debilitating conditions and the extremes of life are predisposing causes.

Symptoms.—The skin in the vicinity of a scratch or abrasion becomes irritable and feels stiff. After a few hours symptoms of toxæmia supervene, which rapidly increase in severity.

Signs.—If the infection commences in a wound, a rose-pink rash extends over the adjacent skin. The edge of the rash is raised, a feature which is often more easily appreciated with the finger than the eye. The colour of the rash and its obvious edge

¹ A specific disease is one which is produced by a single micro-organism.

distinguish erysipelas from cellulitis. As the rash extends, vesicles appear, which burst and discharge serum. Considerable œdema occurs when lax tissues are involved, particularly the orbit and the scrotum. The rash gradually fades, and for some weeks a brown discoloration of the skin remains, due to pigment set free as a result of destruction of red corpuscles.

Complications.—*Gangrene.*—Sloughing of skin and subcutaneous tissues occasionally occurs, particularly of lax tissues in patients of poor resistance.

Lymphatic Obstruction.—A severe attack of erysipelas is sometimes followed by fibrosis of the lymphatic vessels and nodes, so that lymphatic drainage is impaired. The eyelids are not uncommonly affected, greatly to the detriment of the patient's appearance and comfort (fig. 6).

Treatment.—Fortunately it is now a rare disease and is completely controlled by penicillin.

ERYSIPELOID¹

Erysipeloid is caused by the Gram-positive bacillus *Erysipelothrix rhusiopathiæ* which is introduced into the tissues by a punctured wound, usually as a result of a prick by a fish bone or scale, or a splintered meat bone. The organism is a common pathogen of animals and birds. The disease is occupational, and it is also seasonal, being most common in late summer and early autumn. The incubation period is from two to seven days, following which a purplish induration appears at the site of infection, usually on a finger. Induration and dusky discoloration gradually extend to the palm or back of the hand and adjacent fingers. Discomfort and stiffness may be sufficiently severe to disable the patient, but general symptoms of infection are slight, and regional lymph nodes are not affected. Temporary improvement is commonly followed by relapses, but after a period of from four to six weeks the condition gradually subsides.

Treatment consists in keeping the affected hand at rest, and intramuscular penicillin. Under this regimen the condition subsides in a few days. The disease is more common than is generally realised, and in some cases useless incisions have been made when the condition had been erroneously diagnosed as cellulitis.

TETANUS

About 200 to 300 cases of tetanus occur annually in England and Wales, with a probable mortality of about 40 per cent. This number is unlikely to diminish, as the majority follow a trivial or forgotten abrasion. Eradication can only follow universal active immunisation. Tetanus is relatively more common in other parts of the world. In rural India it is estimated to be the fourth commonest cause of death. Half the deaths in some Bombay hospitals are due to this cause.

The causal organism, *Cl. tetani*, is a straight slender rod, which forms a characteristic terminal spore giving the 'drumstick' appearance (fig. 7). It is Gram-positive and is an obligatory anaerobe; the growth requirements are dependent on anaerobic conditions produced by other organisms introduced into the wound at the same time. The organism can produce a toxin second only in potency to that of *Cl. botulinum*. Although the organism is common, especially in manure and soil, the complex conditions for growth and toxigenesis can only occasionally co-exist and thus the disease is uncommon, but seems to occur when least expected (J. Macrae).

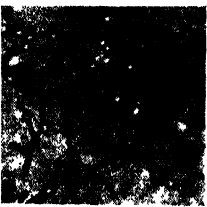


FIG. 7.—*Cl. tetani* showing typical terminal spores.

Tetanus is a wound infection; any wound will serve, but deep, contused, soil-contaminated injuries infected with pyogenic organisms and containing foreign bodies are most likely sources of tetanus toxæmia. The disease is wholly produced by the exotoxin. The organism itself does not damage tissues.

¹ Described by Marrant Baker, of St. Bartholomew's Hospital, in 1873, as *Erythema serpens*, and known in some localities as fish-handler's disease.

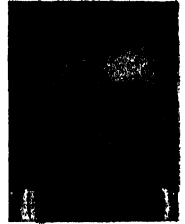


FIG. 6.—Lymphatic œdema of face and eyelids. The patient was unable to open his eyes.

The toxin, a very powerful poison, does, however, damage the tissues, notably the central nervous system. There is a dual action on nervous tissue: (1) it interferes with the acetyl-choline/cholinesterase balance at the peripheral motor end plates so that a continuous excess of acetyl-choline remains, producing clinically a sustained state of tonic muscular spasm, and (2) it causes extreme hyper-excitability of motor-neurones in the anterior horn cells which can result in explosive and widespread reflex spasm of muscle in response to even minor sensory stimuli. The toxin producing such effects is fixed in the tissues and cannot then be neutralised by antitoxin. Only free toxin can be neutralised.

Clinical Features.—Generally a short incubation period implies a serious attack, but an incubation period measured as from the time of wounding until the first symptom is often fallacious since we cannot know when the organism started to grow or to produce toxin. Indeed, it is known that wounds containing tetanus organisms have healed and been forgotten for years and then some local change produced the correct combination of factors allowing the organism to multiply and produce toxin. This should be remembered when re-opening an old accident or war wound.

A better prognostic index is the 'period of onset'. This is the time between the first symptom and the first reflex spasm. If this period has been less than forty-eight hours, death is very likely. As the period of onset lengthens so does the prognosis improve.¹

The first symptoms are difficulty in swallowing and stiffness of the jaw, succeeded by pain in the neck, back, and abdomen. Then tonic muscle spasm develops, often sparing the limbs relatively. The sardonic smile of tetanus is evidence of this stage of the disease (fig. 8), and there is considerable difficulty in swallowing and reduction in breathing capacity. Thereafter, reflex convulsions can occur affecting all the muscles, causing great pain, opisthotonus and sometimes muscle rupture (psoas, rectus abdominis, pectoral) (fig. 9). These spasms are spontaneous or may be induced by even trivial stimuli of noise, injury, or movement. If severe, they stop respiration and the patient becomes cyanosed. A cyanotic tetanic convulsion threatens life. Tonic muscular spasm remains between the reflex attacks, thus distinguishing tetanus from strychnine poisoning.

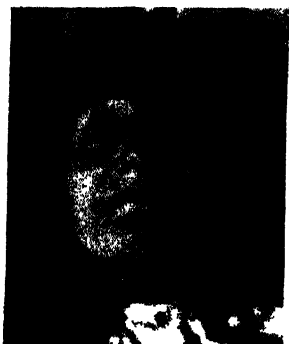


FIG. 8.—Risus sardonicus.

The temperature is usually a little elevated and the pulse is rapid. Without energetic treatment, death occurs in a few days, usually from pneumonia, or from respiratory failure during a cyanotic attack.

Infants and patients older than forty-five years fare badly with tetanus. When the infection enters the raw stump of the umbilical cord (tetanus neonatorum), the disease tends to be rapidly fatal.

Hippocrates, circa 460–377 B.C., is believed to have been the first to recognise this fact.

At an early stage, tetanus might be mistaken for tonsillitis, back strain, an acute abdominal condition, or hysteria (the latter, especially if dysphagia is the presenting symptom).

Treatment.—Prophylactic.—*Active immunity* is conferred by injections of adsorbed toxoid. One ml. is administered and repeated at intervals of six weeks and six months, and followed by a 'booster' dose every five years. If such an active immunity is present, an extra dose should be given at the time of injury. Tetanus toxoid practically eliminated tetanus during the 1939–1945 war.

Passive Immunity.—Every patient with a potentially infected wound who has no active immunity, must have an intramuscular injection of 1,500 international units of antitetanic serum (A.T.S.), given with due precautions (p. 10). Thereafter he must have active immunisation with toxoid. Many people develop immunity to horse serum. Thus, if a patient has already had a dose of horse serum (e.g. antitetanic serum) for one accident, he is likely to destroy the serum given at the time of the next one. In such a case, large (50,000 units) doses should be given and repeated every other day in the hope of staying the disease and allowing time for toxoid to produce a more permanent immunity. It is worth remembering that a prophylactic dose of equine antitetanic serum is probably only useful once in the life of a patient.



FIG. 9.—A torn rectus abdominis muscle from a case of tetanus, which followed a trivial wound of a toe. (R.C.S. Museum.)

The inherent disadvantages of equine antiserum (A.T.S.), can be avoided if human antitetanus globulin (A.T.G.) is available. This is an homologous antitoxin and its protective value is one hundred times that of the heterologous equine type. A dose of 250 units of A.T.G. will give adequate cover during the period needed to establish active immunity.

A.T.G. can be obtained in Great Britain, but supplies are small and its use has so far been limited, but well merits extension.

Of recent years much has been written about the value of penicillin and other antibiotics in preventing the onset of tetanus. Although the organism is sensitive to penicillin, it must be remembered that the conditions of growth in an infected wound make access of the antibiotic difficult; the spore is unaffected and is capable of surviving until the antibiotic disappears. Several cases of tetanus have already been seen who had adequate dosage of penicillin from the date of injury. Thus, it is sensible to prescribe penicillin, but unwise to depend upon it as a prophylactic agent.

It is essential that every patient who has had antitetanic serum should be immunised at once with toxoid.

Treatment of Established Case.—All cases should be isolated in quietness and comfort. It is not necessary to attempt excision of the wound. If pus is present it should be drained but, in general, ordinary wound toilet will suffice. 100,000 I.U. of antitetanic serum is given at once, half intravenously and half intramuscularly, to limit the further fixation of toxin. Unless the patient has had previous horse serum, the A.T.S. need not be repeated. If available, human antitetanus globulin (A.T.G.) is preferable to A.T.S.—only one dose is ever needed. In treatment, as in prophylaxis, tetanus antibody material, whether heterologous or homologous, is the only substance

capable of neutralising the toxin which causes this serious disease. Further treatment resolves itself into three stages, dependent on the development of the disease.

Stage 1.—*A mild case* is one where there is tonic rigidity but neither swallowing nor respiration is noticeably affected. Initial sedation should be by intramuscular promazine (Sparine) (100 to 150 mg.) and intramuscular amylobarbitone (200 mg.). These drugs will probably need to be continued six-hourly.

Stage 2.—*A seriously ill case* is one where there is swallowing difficulty. There may be some reflex spasm but no major cyanotic episodes. A naso-gastric tube is passed and sedation continued as above. A liquid balanced diet containing 2,000 calories and 50 G. of protein is given by the tube, unless the blood urea rises, when protein must be temporarily discontinued. If the patient has difficulty in breathing, he will require a tracheostomy in order to maintain a clear airway. The operation can often be done under local anæsthesia and a cuffed tracheal tube must be inserted. Nursing must now include meticulous care of tracheostomy tube—posture, suction, and humidification (p. 636). Sedation must be maintained as before.

Stage 3.—*Dangerously ill.*—Should the patient have a major cyanotic convulsion, the third stage of treatment is necessary. Curare, in doses of 25 to 40 mg. is given intravenously initially and afterwards intramuscularly to maintain complete relaxation. Simultaneously, intermittent positive pressure respiration must be provided until such time as no further muscle spasms occur (usually twelve to thirty days). Sedation as given in Stages I and II is stopped. This last stage of treatment makes such demands on medical and nursing services that it can only be used safely in a special, experienced respiratory unit. Such a case requires over 350 individual acts of nursing service each day, and a special nurse continually, while the monitoring requirements of artificial respiration need special equipment and skill.

The care of the '*dangerously ill*' case is often worsened by the development of complications made possible by the unnatural preservation of life brought about by artificial respiration. Such patients may develop severe negative nitrogen balance, myocarditis, vascular failure associated with extremes of blood pressure and pulse-rates, and severe damage to motor neurones in the cord and brain leading to major paralysis. These are all due to toxin. Thus, even if treatment prevents death by the classical events of spasm or pneumonia, patients may still die from a lethal dose of toxin. Although the major therapeutic effort is exerted in the control of spasm and respiration, the general care of the patient must not be forgotten. Penicillin and streptomycin are needed to prevent infection. The eyes must be protected while curare is used, or conjunctivitis rapidly develops. The curarised patient is unresponsive, but is conscious and sensitive: this must never be forgotten. The treatment described, if available, can reduce the tetanus death rate to about 15 per cent., but application of such treatment is difficult and obviously not possible except in especially equipped hospitals.

Gas-gangrene.—This acute specific disease is dealt with on p. 44.

SERUM REACTIONS

Patients who have had previous serum injections or who are prone to asthma or allergic diseases may have a local or general reaction when serum or other antigen is injected. Anaphylaxis is the term given to describe this reaction and the commonest causes in practice are the injection of antitetanic serum, of contrast media for intravenous pyelogram, cholangiogram or arteriogram, or a blood transfusion. The reaction varies in site and intensity.

Prevention.—Utmost care must be taken before any intravenous or subcutaneous injection is given. The older test by intradermal injection of small amounts has now been abandoned because skin sensitivity bears no constant relation to general sensitivity and death has occurred following an intramuscular injection of serum after a negative intradermal test. *When using serum, the best procedure is to dilute it ten times in normal saline and to inject it subcutaneously in small amounts.* If anaphylaxis develops, it will be mild and respond easily to treatment. Where trouble is anticipated intravenous Piriton 10 mg. given ten minutes before will probably prevent it. With antitetanic serum 35 per cent. of patients get some sort of reaction. The usual routine is to give 0.2 ml. of the serum, to wait half an hour, then give the

remaining 0.8 ml. and to wait for a further half-hour. Adrenaline must be immediately available.

Acute Anaphylaxis.—The symptoms may appear within a few minutes. They are most alarming and may rapidly lead to death. In general the treatment is to give adrenalin (1:1,000) 1 ml. subcutaneously or intravenous Piriton 10 mg., and to apply a tourniquet. There are four main types of acute reaction.

(1) *Respiratory distress* with urticaria, bronchospasm, and œdema of the glottis. Treatment is to introduce a large French's needle through the crico-thyroid membrane in order to give endotracheal oxygen. Calcium gluconate 20 ml. of 10 per cent. intravenously will help. If pulmonary œdema occurs, hydrocortisone succinate 100 mg. intravenously must be given.

(2) *Generalised Convulsions.*—This requires 50 mg. 2½ per cent thiopentone intravenously and repeat in five minutes if necessary.

(3) *Peripheral Vascular Failure.*—This requires phenylephrine 1 mg. intravenously and intravenous hydrocortisone. If this fails, a noradrenaline drip should be started.

(4) *Sudden Cardiac Arrest.*—This requires immediate cardiac massage: the heart must be restarted within three minutes (Chapter 30). The help of an anaesthetist is invaluable and all cases will require oxygen.

Local anaphylaxis is occasionally seen if further injections are given at the same site after the interval of a week. An acute inflammatory reaction occurs, which in rare cases progresses to sloughing of tissues.

Serum sickness is the commonest serum reaction and may occur in any patient. Urticaria, œdema, pains or effusions in joints, and elevated temperature are usual features, which frequently occur seven to ten days after the injection. Cold applications applied locally, and injection of adrenalin, minimise the discomfort. Oral anti-histamines such as Piriton 4 mg. four times a day must be given till the condition responds.

ANTHRAX

B. anthracis is a large Gram-positive rod which occurs in chains (fig. 10). It is aerobic and forms spores which are very resistant to heat and antiseptics. The disease occurs in many domestic animals and is likely to appear in men who handle cattle, carcasses, wool, hides, hair, or imported bone-meal and ivory.

The **cutaneous type** is the commonest human variety; the incubation period is from three to four days. The lesion usually commences on an exposed portion of the body, such as the hands, forearms, or face (fig. 12). An itching papule occurs, around which a patch of induration soon becomes evident. The papule suppurates and is replaced by a black slough, and a ring of vesicles appears



Fig. 10—*B. anthracis*, large rectangular bacilli surrounded by capsule.



FIG. 11.—Anthrax pustule.
(A. E. Hodgson, Liverpool.)

on the surrounding indurated area. This stage comprises the typical 'malignant pustule' (fig. 11). The induration extends subcutaneously, so that a brawny, congested patch develops around the site of infection. The regional lymph nodes are invariably involved. Toxæmia is always in evidence, and an elevated temperature and raised pulse-rate are important features in the diagnosis of an early case of anthrax. This is supported by finding the bacilli in a smear

of vesicle fluid, and is confirmed by culture and animal inoculation.

Treatment.—Penicillin is the treatment of choice.

Prevention.—Very careful precautions are taken to sterilise potentially infected animal products and wool from countries where the disease is endemic, such as Asia Minor and Pakistan.

Differential Diagnosis (Cutaneous Anthrax).—The two conditions easily mistaken for it are a virulent furuncle and accidental vaccinia. Fig. 12 shows the way in which accidental vaccinia can occur. The recently vaccinated child with the vaccinia pustule on its arm in full activity, while being carried by the mother or nurse, places the vaccinated area against her cheek.



FIG. 12. — The commonest cause of accidental vaccinia.

Pulmonary Type (syn. Woollorter's Disease).—Caused by the inhalation of spores, and characterised by a virulent bronchitis and bronchopneumonia, with toxæmia, dyspnoea, and blood-stained sputum. The organisms are found in the sputum. Immediate and intensive antibiotic therapy may save the patient.

Alimentary Type.—Follows the ingestion of spores, provided they escape destruction by the acid in the stomach. Severe enteritis follows, which resembles cholera. The patient collapses with severe abdominal pain and blood-stained watery diarrhoea. This type occurs in Africa but is unknown in Great Britain.

MONILIASIS

Candida (monilia) albicans is a pathogenic yeast-like organism frequently present in small numbers in the healthy mouth and bowel. It may cause primary infection in the newborn, or superinfection when flora are disturbed by antibiotic treatment. In **Thrush** (monilial stomatitis) white patches are seen in the mouth; it may occur in infants, in post-operative patients, and with ill-fitting dental plates. **Monilial vaginitis** is common in pregnancy and diabetes. *Candida* may infect moist skin under breasts, and nail folds.

Administration of broad-spectrum antibiotics often results in superinfection by *candida* in the respiratory tract and bowel, and may be responsible for digestive upsets. Systemic moniliasis with invasion of lung and blood-stream is very rare.

Candida infections are treated by local applications of gentian violet or of the antibiotic nystatin (myco-statin).



FIG. 13.—*Monilia* in sputum.

ACUTE ABSCESS

Bacteria which cause pus formation in any part of the body reach the infected area by one of three routes :

- (i) Direct infection from without, e.g. penetrating wounds.
- (ii) Local extension from some adjacent focus of infection, such as an alveolar abscess from an infected tooth root.
- (iii) Blood-stream or lymphatic vessels.

In the case of hæmatogenous infection, some predisposing factor may operate, e.g. a bruised muscle causes an extravasation of blood which forms a suitable nidus for pyogenic organisms. Acute osteomyelitis may occur after a minor injury to a limb.

The bacteria, having gained access to the tissues, multiply and produce toxins, and so cause acute inflammation. The vitality of the tissues is lowered, and the area is surrounded by a peripheral and painful zone of

acute inflammation, which is infiltrated with leucocytes and bacteria. Polymorphs contain a proteolytic enzyme which causes liquefaction of the tissues into pus, which is composed of dead leucocytes and bacteria. The tension in the abscess rises owing to the exudation of plasma and it may spread along the paths of least resistance to the surface of the body, or to a hollow viscus where the pus is eventually discharged.

Occasionally the resistance of the body is sufficient to destroy the bacteria before pus finds its way to the surface, in which case the fluid is absorbed, and either fibrosis follows or a cavity remains containing inspissated pus. For example, this condition may occur in the breast when a misplaced attempt has been made to cure an abscess by antibiotics. The resulting lump is called an 'antibioma'. In some cases, as in staphylococcal abscesses of bone (Brodie's abscess, p. 232), infection remains latent, but gives rise to exacerbations of inflammation consequent on local injury or impaired general health.

Symptoms.—The patient complains of malaise, the degree depending to some extent upon the size of the abscess, the virulence of the organism, and the tension within the cavity. Throbbing pain is characteristic of supuration, the pain becoming more acute if the affected part is dependent, e.g. an infected finger.

Signs.—(a) GENERAL.—The signs of inflammation are present to a varying extent. In severe cases rigors may occur.

(b) LOCAL.—The five classical local signs of inflammation are:

1. *Heat*—the inflamed area feels warmer than the surrounding tissues.
2. *Redness* of the skin over the inflamed area. Both this sign and the previous one are due to hyperæmia.
3. *Tenderness*, due to the pressure of exudate on the surrounding nerves. If the exudate is under tension, e.g. a furuncle of the external auditory meatus, pain is severe, whereas lax tissues, such as the scrotum, may swell enormously with but little discomfort.

4. *Swelling*, as a result of hyperæmia and inflammatory exudates.

5. *Loss of function*—an inflamed tissue does not perform its physiological function.

The readiness with which these signs present depends on the extent of the inflammation and its proximity to the surface. The swelling is at first brawny and œdematous; later softening and fluctuation are manifest (fig. 14). In some cases increasing œdema is characteristic of deep pus, as in acute mastitis. If untreated, an abscess tends to point; the skin or membrane covering it gives way and the contents are discharged, usually with marked amelioration of symptoms.



FIG. 14.—Abscess of neck. The arrows indicate fluctuation in two planes.

The *treatment* of pyæmia consists in endeavouring to prevent further emboli from reaching the blood-stream. Thus, in the case of pyæmia due to suppurative arthritis of the knee joint (nowadays a rare occurrence), amputation may be indicated, or if the condition is caused by thrombosis of the lateral sinus, then ligation and division of the internal jugular vein may interrupt the stream of emboli. Otherwise, abscesses are dealt with as they occur, and general treatment is instituted as for septicæmia.

WOUNDS

A wound is a loss of continuity of skin or mucous membrane as a result of injury. Wounds are either incised, lacerated, penetrating, contused or poisoned (see 'Bites', p. 17).

Incised wounds are due to an injury by a sharp instrument, and are characterised by hæmorrhage, with the possibility, according to their situation and depth, of injury to underlying structures (figs. 16 and 123).

Lacerated wounds, especially war wounds, are particularly prone to infection. In cases of recent wounds, e.g. due to industrial or road accidents, an emergency operation is performed in order to 'debride' the wound as completely as possible, and every hour's delay adds to the risk of infection becoming established.

Debridement.—The surrounding skin is cleaned and a few mm. (not more) of the skin edges of the wound are excised and all damaged tissue and foreign bodies are removed. All dead muscle must be excised meticulously with scissors so that only red, bleeding, contractile, and healthy muscle is left. Nerves should be covered with muscle if possible and carefully preserved. Hæmostasis is secured by pressure, hot packs and catgut ligatures where necessary. If loss of skin is not excessive, closure may be attempted.

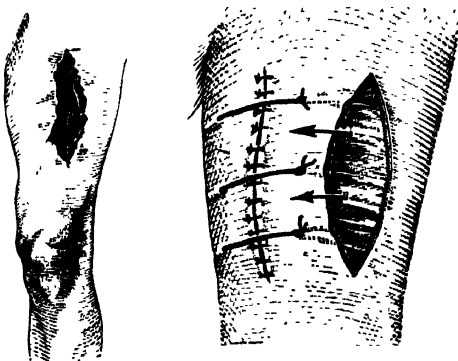


FIG. 15.—Excision of an extensive wound. Closure is facilitated by a relaxation incision. (The bare area is covered with a split-skin graft.)

Early skin cover is very important and a skin graft over the raw muscle usually takes very well. Sometimes a relaxation incision is wise (fig. 15). Experience from two world wars has taught us that such potentially infected wounds may be closed in three ways:

(1) **Primary Suture.**—For a clean wound inflicted not more than about six hours previously where there has been thorough debridement.

(2) *Delayed Primary Suture*.—Where even after debridement there is some doubt about infection, or where the wound is twelve to twenty-four hours old. Here the wound is lightly packed with paraffin or dry gauze. Two or three days later the dressings are removed in the theatre and the skin edges drawn together by a careful plastic technique, completing the skin cover by a skin graft where necessary.

(3) *Secondary Suture*.—This is only for late untreated wounds at about the sixth to tenth day. Owing to inflammatory reaction, excision must not be attempted, but a small amount of skin suture may be carried out in certain areas or at the edges of wounds if it is certain that pus will not pocket in the area. On the whole, such wounds are best treated by wet dressings of saline, saturated sodium sulphate or weak hypochlorite solution until the granulations are healthy and skin grafting is possible. In all cases systemic penicillin or other appropriate antibiotic is given. Complete rest of the part is important, and a plaster cast may help very considerably especially in the case of the limbs. It will need to be removed for the purpose of delayed primary suture and then possibly replaced for a few weeks till healing is satisfactory.

Penetrating wounds are notoriously deceptive. A sharp object can penetrate many inches with a mere slit in the skin as the only immediately obvious sign. Impaired movement or loss of sensation indicates injury to tendons or nerves, and hæmorrhage may be obvious, e.g. 'butcher's thigh' (p. 123). Penetrating wounds of the abdomen may be symptomless until internal hæmorrhage (fig. 16) or peritonitis indicates damage to blood-vessels, solid

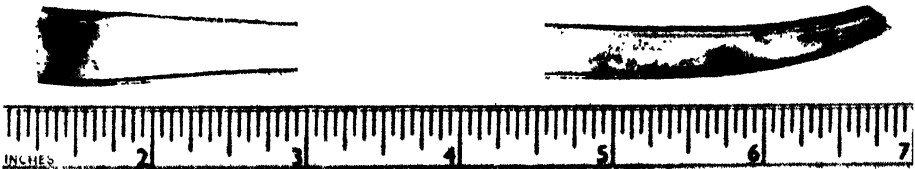


FIG. 16.—Following an air raid a man, aged fifty, walked into an adjacent hospital because of minor facial wounds. He collapsed and died a few minutes later, quite unaware that this slender piece of glass, which was just protruding through the skin, had penetrated three layers of clothing and his abdominal wall. It had severed the middle colic artery.

(Mildmay Mission Hospital, London.)

viscera or bowel; therefore the wound must be explored without delay to discover whether there is intraperitoneal injury.

Contused wounds, which commonly follow road accidents, are dealt with by removing any dirt or foreign bodies, and the application of non-adhesive or paraffin gauze dressings ('Gravel', p. 19).

In all cases in which tetanus *might* develop, appropriate prophylactic measures *must* be applied (p. 9).

BITES

Insect bites in this country are usually inflicted by wasps or bees, and anthrax has followed the bite of a horsefly. Anaphylaxis can follow a bee or wasp sting in sensitised people (p. 11). Vasomotor collapse, coma, and death may occur within twenty minutes, especially if the venom is injected directly into a vein, e.g. in the dorsum of the

hand. Wasps are fond of alcoholic drinks, and picnickers especially should keep an eye on their beer, lest, after drinking, they suddenly develop œdema of the glottis. Most bees, as distinct from wasps, suffer avulsion of their sting and poison gland, which is left protruding from the wound. Pressure on the gland squeezes poison into the tissues, so the gland and sting should be removed by scraping. Bee venom is acid, and should be neutralised by the application of ammonia, soda, or methylene-blue. On the contrary, the venom of the wasp is alkaline, and requires an acid, such as vinegar or lemon juice, for its neutralisation. Antihistamine drugs are given orally, and local application is of value in allaying local irritation.

Bites of animals such as the horse, cat, and dog require the usual treatment of wounds. Where there is the slightest suspicion that the animal is suffering from rabies, the bite should be freely excised. If possible, the responsible animal should be kept under observation, or, if it has been killed, Negri bodies should be sought in the brain. Prophylactic treatment can be obtained only at a special institute.

Snake Bites.—In England the only poisonous reptile is the adder, or viper (*Vipera berus*), but a fatal result is unlikely. During the decade 1944–1953 only three fatal cases occurred in England and Wales, two of whom were debilitated children.

Giddiness, vomiting, and cardiovascular collapse occur in proportion to the amount of venom injected, and the frailty of the patient. First-aid measures consist of the application of a tourniquet, and within the first few minutes sucking the wound may remove some of the venom. Drastic measures, formerly recommended, such as excision of the wound and the application of permanganate of potash merely cause unnecessary trauma and necrosis of tissue. The only anti-venom serum obtainable is prepared by immunising horses with the venom of the South African viper. Its efficacy against *Vipera berus* is unproved and its use is unjustified, as the danger of anaphylaxis is greater than the effects of the venom. In all cases antihistamine drugs are indicated, both for therapeutic effect, and as an antidote to serum sickness if antivenom was necessary.

Human bites are discussed on p. 164.

FOREIGN BODIES IN THE TISSUES

In every case of suspected foreign body which is opaque to X-rays, a radiograph must be taken in at least two planes. Other aids to X-ray localisation are the insertion of straight needles or Kirschner wires aimed at the foreign body, or Michel's clips applied to the skin over the presumed site. With comparatively superficial foreign bodies, personal observation and palpation in the X-ray room is of great assistance. When removal is attempted, a good light, ample time, and exsanguination of the limb are all valuable. Exsanguination assists by rendering the field bloodless, and in recent cases a reddish or brown track, due to extravasated blood, indicates the path of the foreign body. An electronic metal locator is often of value.

Hypodermic needles not infrequently break at the neck, where corrosion easily occurs. Stainless needles are advisable, and should be tested frequently. The most difficult needle to recover is one situated in the internal pterygoid muscle, which has broken when a dental surgeon has attempted a mandibular block. The glistening tendinous intersections in the muscle continually raise the surgeon's hopes, and he is fortunate if the needle is recovered within an hour, but patience is usually rewarded.

Domestic needles commonly become impacted between the small bones of the hand or foot. The patient may be entirely unaware of their entry. Unless a small fragment is lodged deeply, removal is advisable, as infection may otherwise develop, and startling cases are on record in which a needle has entered the venous circulation and become embedded in the heart muscle, or travelled to some distant part of the body.

Sewing-machine needles occasionally transfix the terminal phalanx and nail, and then break. After injecting a local anæsthetic the finger should be forcibly pressed on to a hard surface, so that the fragment retraces its path. The end then projects through the nail and is removed with forceps.

Indelible-pencil fragments occasionally become lodged in the subcutaneous tissue of the hand, particularly in children. The treatment is immediate excision of the fragment and adjacent tissue. If allowed to remain, a pigmented discharge will persist for months, and the exuberant granulations require constant attention.

Fish hooks, and similar articles which possess barbs, are removed by pushing the hook in such a direction that it emerges through the skin at the nearest point. The barb is then nipped off, and the hook withdrawn along the path of entry. Local anæsthesia is desirable.

Gravel is not uncommonly driven into the subcutaneous tissues of the face, hands, or knees. Ugly scars are the penalty for incomplete removal—a particularly distressing sequel if occurring on the face—‘tattoo marks’. In all but minor cases an anæsthetic and meticulous removal are indicated. Brisk rubbing with a nail-brush is sometimes helpful. Any small remaining fragments are encouraged to extrude themselves by the application of hot compresses of hypertonic saline, or 10 per cent. sodium sulphate.

Glass splinters from windows usually contain sufficient lead to render them opaque to X-rays. Fine clear glass from glass ampoules or syringes are often not radio-opaque. Every lacerated or punctured wound caused by glass must be examined radiographically, as it is surprising how often fragments of glass are otherwise missed. In the majority of cases removal is indicated.

Surgical operations in which metallic sutures are used occasionally lead to trouble. Thus, **wire sutures** in the abdominal wall sometimes break within a few days of their insertion, and the fragments may then cause a pricking pain in the wound. It is remarkable that even stout wire undergoes ‘metal fatigue’ in time and breaks into small pieces. The fragments in some situations cause serious trouble (fig. 17). **Pieces of clothing** may be found in war wounds, especially from trench mortars or shell fragments. Wool often contains sporing bacteria. Nylon and cotton are more easily sterilised.



FIG. 17.—Ten years after suture of a fractured patella. The stout silver wire has snapped, and two loose fragments have eroded into the joint.

All clothing, however, is contaminated by the wearer's own bacteria including, sometimes, gas gangrene clostridia from his bowel. Debridement is therefore essential in all wounds in order to extract every piece of cloth.

Swabs and packs are occasionally overlooked, especially during an abdominal operation, an oversight which is a not uncommon cause of litigation.

The foreign body causes a local peritoneal irritation, and usually within a few weeks a tender swelling is palpable. In due course suppuration occurs and weeks or months later the swab escapes with a discharge of pus either on the surface or into the alimentary canal. Radio-opaque threads (such as those in bank notes) should be incorporated in all swabs or packs, so that if the swab count is wrong at the end of an operation, it can be discovered by portable X-rays (fig. 18). Alternatively, a negative X-ray permits the surgeon to close the wound with an easy mind.

SINUSES AND FISTULÆ

A *sinus*¹ is a blind track lined with granulations leading from an epithelial surface into the surrounding tissues. A *fistula*² is an abnormal communication between the lumen of one viscus and the lumen of another, or the body surface. Sinuses and fistulæ may be congenital or acquired. Forms which have a congenital origin include pre-auricular sinuses (p. 454), branchial fistulæ (p. 519), and tracheo-oesophageal fistulæ (Chapter 31). The acquired forms often follow inadequate drainage of an abscess. Thus a perianal abscess may burst on the surface and lead to a sinus (erroneously termed a blind external 'fistula'). In other cases the abscess opens both into the anal canal and on to the surface, resulting in a true fistula-in-ano (Chapter 41).

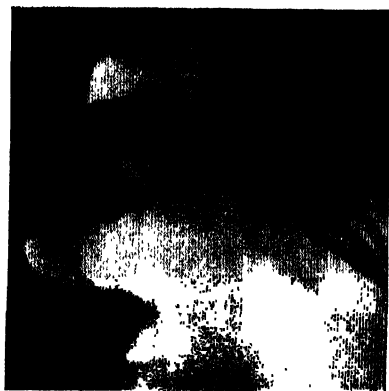


FIG. 18.—A 'lost' swab located by portable X-ray on the operating table during cholecystectomy.

Persistence of a Sinus or Fistula.—The reason for this will be found amongst the following:

- (1) A foreign body or necrotic tissue is present (e.g. a suture, sequestrum, or a fæcolith).
- (2) Inefficient or non-dependent drainage.
- (3) Irritating discharges, such as urine or fæces, maintain continuous inflammation. They are often associated with unrelieved obstruction of the lumen of a viscus or tube distal to the fistula.
- (4) Absence of rest, such as occurs in fistula-in-ano due to the normal contractions of the sphincter.
- (5) The walls have become lined with epithelium.
- (6) Dense fibrosis prevents contraction and healing.
- (7) Type of infection, e.g. tuberculosis or actinomycosis.
- (8) The presence of malignant disease.

Treatment.—The remedy depends upon the removal or specific treatment of the cause. For example, a cavity following an acute pyogenic abscess may need adequate drainage, if necessary by counter-openings. Packing with

¹ Sinus (Latin) = a hollow; a bay or gulf.

² Fistula (Latin) = a pipe or tube.

gauze moistened with a suitable antiseptic or granulation-promoting substance (ulcers, p. 37), will encourage healing from the bottom of the cavity. Antiseptics should occasionally be changed, as organisms appear to become immune to long-continued use of the same preparation. Rest is provided as efficiently as possible, and scraping is sometimes necessary to destroy any lining epithelium.

The treatment of sinuses and fistulæ affecting different sites and organs will be found on the appropriate pages.

CHAPTER 2

CHRONIC INFECTIONS. VENEREAL DISEASES.
PARASITES.

ACTINOMYCOSIS

This disease is caused by *Actinomyces israelii*, an anaerobic Gram-positive branching filamentous organism (fig. 19) which sometimes lives as a harmless parasite in the tonsillar crypts and dental cavities of the otherwise normal mouth. It is popularly supposed that it occurs in corn and grasses, but the pathogenic

organism does not do so. If the organism invades tissue, it causes a subacute pyogenic inflammation with considerable induration and sinus formation. Trauma and the presence of carious teeth are important predisposing factors in the development of lesions in the mouth.

Diagnosis depends on finding the organism in pus or in tissue section. Pus should be collected in a sterile tube (a swab is usually insufficient) and inspected in a good light for the presence of pin-head sized 'sulphur granules'. On microscopy the granules are seen to



FIG. 19—*Actinomyces israelii* in tissue section.

consist of Gram-positive mycelia. The peripheral filaments radiate¹ from the central part of the granule and may be surrounded by Gram-negative tissue clubs. The identity of the organisms may be confirmed by culture but the presence of secondary organisms often makes this difficult.

Actinomycotic lesions are characterised by the formation of a firm, indurated mass, the edges of which are indefinite, and infection spreads by direct invasion of adjacent tissues. Lymph nodes are not affected, but if a vein is invaded, pyæmic infection is likely to follow.

There are four main clinical forms of actinomycosis:

(i) *Facio-cervical* is the commonest form. The lower jaw is more frequently affected, often adjacent to a carious tooth. The gum becomes so indurated that it simulates a bony swelling. As extension occurs, nodules appear, which soften and burst. The overlying skin of the face and neck is indurated and bluish in colour, softening occurs in patches, and eventually abscesses burst through the skin (fig. 683). The characteristic features of the condition are chronicity, dense induration and sinuses surrounded by bluish skin (p. 531).

(ii) *Thorax*.—The lungs and pleura are infected either by aspiration of the fungus or, occasionally, by direct spread downwards from the pharynx or neck, or upwards through the diaphragm.

The disease extends through the lungs to the pleura and chest wall, which,

¹ Actinomyces = ray fungus.

in the late stages, is riddled with sinuses (fig. 20). An empyema is not uncommon, and the infection sometimes spreads through the diaphragm to the liver or subphrenic spaces. Clinically, the condition resembles tuberculosis, and in the early stages is only distinguished by the discovery of the organism in the sputum and confirmation by culture.

(iii) *Right iliac fossa* (Chapter 38).

(iv) *Liver* (Chapter 34).

Treatment.—*Actinomyces* is usually sensitive to penicillin, tetracycline and some other antibiotics, but the sensitivity should be checked in the laboratory. A prolonged, intensive course of penicillin (8 mega units reducing to 4 daily) is usually the best treatment.

TUBERCULOSIS

This disease derives its name from the characteristic lesion called a tubercle (a collection of inflammatory cells surrounding the organism, and later progressing to caseation) which is the response of the body to invasion by tubercle bacilli. These are mycobacteria and were first described by Koch in 1882. When stained they are difficult to decolorise with acid (hence the term 'acid-fast bacilli'). *Myco. tuberculosis* (fig. 21) may be human or bovine in type and is spread by airborne infection by the human type from open tuberculous cases, and by milk from infected cows in the case of the bovine bacillus. There are three main routes of primary infection. (1) By far the most common is by direct spread to the lungs where a primary focus occurs and thence by lymphatics to the hilar lymph nodes. With this primary infection, there is nearly always some hæmatogenous spread to other organs where the bacilli may lie dormant for many years. (2) Via the tonsils to the lymph nodes of the neck. (3) Via the lower ileum, probably in Peyer's patches, to the lymph nodes of the ileocæcal angle where they later show as calcified areas on X-ray.



Fig. 21.—*Mycobacterium tuberculosis* in human sputum.



FIG. 20.—Actinomycosis of the lung involving the chest wall.

The **treatment** of this disease has been one of the dramatic advances of recent years. Three drugs are used, which are given together for the first three months to avoid development of drug resistance:

1. Streptomycin 1 gm. intramuscularly per day.
2. Para-amino-salicylic acid (P.A.S.) 12–15 gm. a day.
3. Iso-nicotinic acid (Isoniazid) 300 mg. a day.

All three are bacteriostatic, and the first and last are bactericidal as well. Isoniazid has a small molecule, penetrates macrophages, and destroys bacilli in the middle of a tubercle. After three months' treatment the sensitivity of the organism will have been determined and it is usually only necessary to con-

tinue with the last two drugs. The whole course of treatment is two years. In nearly 1 per cent. of new cases the organism is resistant and alternative drugs must be considered.

Tuberculosis of the various organs is described in the appropriate chapters of this book.

LEPROSY (HANSEN'S DISEASE¹)

There are probably from ten to fifteen million leprosy sufferers in the world today.

Leprosy is a mildly contagious disease widely spread through the tropical and sub-tropical areas of the world. It is caused by the *Mycobacterium lepræ*, (fig. 22), an acid-fast bacillus morphologically like the mycobacterium of tuberculosis. The mode of transmission of leprosy is not known but the disease is usually contracted in childhood; the source of infection is thought to be from the skin or nasal secretions of a leprosy patient who is in intimate contact with the child. The fact that leprosy no longer spreads in northern European countries even from known infectious cases whose movements are not restricted, and the fact that doctors and nurses in leprosy hospitals almost never contract leprosy, even in the tropics, both suggest that leprosy requires for its transmission some factor associated with the poverty or lack of hygiene that is common in the areas where it is endemic. A vast change in the outlook for this disease has occurred during the last decade. The condition was formerly regarded as hopeless, but in spite of the fact that it is now curable only a small proportion of the cases of this widely spread disease are under treatment. It is probably true to say that leprosy causes more paralysis, deformity, and misery than any other disease, but only in a few centres is an attempt being made to treat the disease and correct the secondary disabilities.

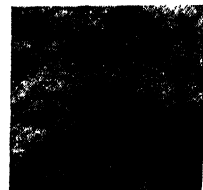


FIG 22.—Leprosy bacilli in tissue section.

The basic lesion in leprosy is a granulomatous infiltration of the superficial tissues of the body. Almost all the damage occurs within about 1 centimetre of the surface of the skin. The histological appearance varies with the resistance of the host, and this produces two main types of leprosy.

Lepromatous Leprosy

At one extreme, where there is little or no resistance, the bacilli multiply with little cellular response, until the subcutaneous tissues may be loaded with masses of bacilli, many of them distending macrophages into great acid-fast 'globi'. The cellular infiltrate is mainly of round cells and epithelioid cells.

Tuberculoid Leprosy

At the other extreme, where there is a strong tissue response, the bacilli are not numerous and are seldom seen except by special concentration methods. The histology is of a more inflammatory type with polymorphonuclear leucocytes as well as round cells and giant cells.

Between these two extremes lie the majority of cases which are therefore classified into various types nearer to one or other end of the spectrum. Characteristically *tuberculoid* leprosy causes sharply localised lesions often affecting only one part of the body, while *lepromatous* leprosy is symmetrical and extensive. Since the damage in leprosy is mainly due to the response of the host cells, *tuberculoid* leprosy causes early, severe but localised deformity, while *lepromatous* leprosy causes deformity late, and more mildly and widely spread. The most severely deformed patients are those affected by some of the intermediate forms (border-line, dimorphous, etc.), where the disease may be both widespread through the body and also rather violent in its reactions.

A unique feature of the disease is not only its predilection for the surface of the body, but also for the cool parts of the surface. Warm areas like the axilla and gluteal cleft are spared, while the parts of the upper respiratory tract, like the lining of the nose, are severely involved. The testis is affected, while the ovary, and other deeply placed

¹ Owing to the stigma attached to the word 'leper', Dr. R. G. Cochrane strongly suggests that the best name for leprosy is 'Hansen's Disease'. Dr. G. Armauer Hansen described *M. lepræ* in 1872.

Gerhard Henrik Armauer Hansen, 1841-1912. Norwegian Physician.
Robert Greenhill Cochrane, Contemporary. Lately Adviser in Leprosy, Ministry of Health, London. Medical Superintendent, Leprosy Hospital, Vadathorasalur, South Arcot, Madras, India.

glands and organs, are spared. Since leprosy does not affect the vital organs of the body, it rarely causes death, and patients do not even feel ill for most of the time they have the disease. From time to time, however, especially in *lepromatous* leprosy and during treatment, there may be periods of 'reaction' during which there may be fever, and nerve and joint pains. All lesions may appear inflamed and paralysis, eye lesions, and other permanent damage may appear.

One of the most characteristic features of leprosy is its effect on nerves (fig. 23). Histologically, the cellular infiltrate may be seen localised around nerve fibres in and under the skin and, on clinical examination, superficial nerves such as the ulnar and posterior auricular may be observed to be swollen and tender. The anaesthesia that results from nerve involvement is an important point in diagnosis and is also a cause of secondary damage and deformity. Much of the loss and disfigurement of hands and feet which has always been associated with leprosy is now known to be due not to leprosy itself, but to the damage and misuse which follows loss of pain sensation.

Medical Treatment. — Diaminodiphenylsulphone (DDS) is the latest of the modern treatments of leprosy. The usual dose is 100 mg. daily, but each case should be started gradually on very small doses and built up slowly. At any sign of *reaction* the drug is stopped. Diphenylthiourea 1906 Ciba has similar action to DDS and seems rather less liable to produce reactions. Most patients need to continue the drug for three to five years to become bacteriologically negative; lepromatous cases should be advised to continue a maintenance dose for life to avoid the possibility of relapse.

The deformities of leprosy are divided into *primary*—those which are caused directly by leprosy and its reactions, and *secondary*—those which result from anaesthesia and consequent misuse. The stigma of leprosy is the stigma of deformity, and a wide-open field awaits the plastic surgeon in this disease.

The Face:

Primary deformity: the skin of the face becomes thickened and sometimes nodular in lepromatous leprosy (fig. 24); the forehead, cheeks, nose, and ears are especially affected. The result in the acute phase is referred to as 'leonine facies'. This infiltration subsides under medical treatment, but may leave the skin wrinkled and without its normal support, producing, in a young person, a caricature of old age. The hair of the eyebrows falls out and the lateral cartilages and septum of the nose may be destroyed leaving collapse of the centre of the nose and lifting of the tip towards the bridge (fig. 25). The upper branches of the facial nerve may be paralysed, giving rise to lagophthalmos; the lower branches are sometimes partially paralysed.

The patient with the above deformities usually finds it quite impossible to return to normal social relationships even though his leprosy may be cured. Plastic surgery can completely transform such a face using a post-nasal inlay to the nose (fig. 25), and an 'island flap' for the eyebrows. A temporalis muscle segment reactivates the eyelids and gives a face-lift to restore more normal contours to the skin.

Eyes:

Some of the blindness of leprosy is simply due to exposure following paralysis of the eyelids. This is preventable by plastic surgery. Other causes of loss of vision are lepromatous infiltration of the anterior segment of the eye and acute allergic changes of the tissues associated with reaction. Acute plastic irido-cyclitis is one of the commonest manifestations of this allergic reaction. Any redness of the eye or loss of visual

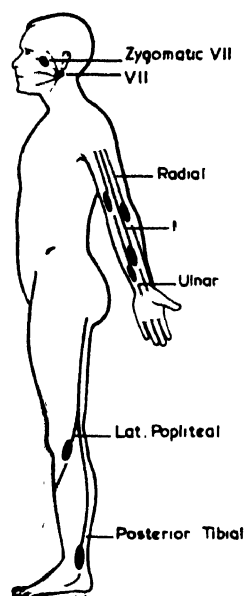


FIG. 23.—Sites of motor paralysis in leprosy

- (1) Ulnar nerve just above elbow joint or wrist joint.
- (2) Median nerve just above wrist joint.
- (3) Lateral popliteal at knee joint.
- (4) Post-tibial above ankle joint.
- (5) Facial nerve in the bony canal or the zygomatic branch.
- (6) Radial nerve at the elbow (rarely)

(After Paul Brand, F.R.C.S.)



FIG. 24.—Nodular lepromatous leprosy.

acuity in leprosy demands full examination and prompt treatment if the sight is to be saved.

Hands¹:

Primary Deformity.—In the upper limbs, leprosy causes paralysis frequently in the ulnar nerve at the elbow and in the median nerve at the wrist (fig. 23) but rarely in the motor part of the radial nerve (1%). Thus the patient commonly loses the use of all the small muscles of the hand but very few forearm muscles (fig. 26). The fingers are clawed and the thumb lies beside the palm, unable to oppose the fingers.



FIG. 26.—Clawed left hand in leprosy. Note that the thumb lies completely unopposed. (Dr. R. G. Cochrane, London.)

hands become scarred and progressively absorbed until only stumps remain. It takes patience and perseverance to teach every patient that his hands can be preserved only by constant alertness to foresee possible dangers, and constant gentleness to his own tissues which are not protected by pain. Once he is convinced that it is not leprosy that is destroying his fingers he may be willing to accept the discipline of caring for himself.



FIG. 25.—(Left) Extreme case of nasal collapse. (Right) After post-nasal epithelial inlay. (N. H. Anna, F.R.C.S.) (By permission from the *Annals of the Royal College of Surgeons of England*.)

Treatment:

The extensor carpi radialis brevis muscle is extended into the hand with free grafts which run along the lines of the lumbrical tendons to correct the clawing of the fingers. The flexor sublimis tendon to the ring finger is withdrawn in the forearm and re-routed to oppose the thumb along the line of the abductor brevis. In this way the fingers and thumb may be balanced and function almost normally. Before attempting operation it is important to make sure that the fingers are made mobile by massage and exercise.

Secondary Deformity.—Since the hand is often totally anæsthetic the patient frequently burns himself, or damages himself by the uninhibited strength which he uses through his fingertips. His

¹ The work on reconstruction of the hand in Hansen's disease was started by Professor Paul Brand at the instigation of Dr. R. G. Cochrane.

Feet:

Primary Deformity.—In the lower limbs the posterior tibial nerve is often involved at the ankle, giving rise to 'clawing' of the toes and anæsthesia of the sole of the foot. The lateral popliteal nerve may also be destroyed, giving rise to foot-drop. The medial popliteal nerve is never involved, so the tibialis posterior muscle can be safely used to correct foot-drop.

Secondary Deformity.—The anæsthesia of the sole of the foot is very serious because almost every patient with insensitive feet sooner or later develops trophic ulceration. If he then continues to walk on his ulcers, the condition progresses and the infection spreads until, after a few years, the foot is contracted and distorted and destroyed to the point where amputation must be advised.

It is important for the patient to understand the pathology of his ulcers and to realise that they are not due directly to leprosy. These ulcers heal readily with rest in a plaster cast and their recurrence can be prevented by the regular use of special footwear designed to spread the weight evenly over the whole foot.

VENEREAL DISEASES**SYPHILIS¹**

A detailed description of venereal diseases is outside the scope of this work, and those requiring it should refer to one of the many text-books on venereal diseases. We include here a general summary of these diseases, and affections of the various individual organs and structures are considered in their appropriate chapters.

The incubation period of acquired syphilis is from ten to ninety days. The diagnosis was revolutionised by the discovery in 1905, by Schaudinn, of the causative organism, the *Treponema pallidum* (*syn.* *Spirochæta pallida*) (fig. 27). The treponema can be discovered in serum obtained by scraping primary lesions. If enlarged lymph nodes are present, lymph can be aspirated with a fine needle, and examination will often reveal spirochætes.

By means of dark-ground illumination the treponema is seen as a spiral organism resembling a corkscrew in appearance. Some eight spirals are present, and the organism is about 8 in length. It must be distinguished from non-pathogenic spirochætes.

Specific Serum Tests

The *Wassermann* reaction, a complement-fixation test, which employs an antigen prepared artificially from normal tissue, is usually positive in untreated cases, about two weeks after the appearance of the primary sore. Treatment should not be delayed until the test is positive, but commenced immediately the treponema is demonstrated. Treatment instituted while the serum is negative yields excellent prospects of permanent cure. In untreated late primary and secondary syphilis the W.R. is almost always positive. In the tertiary stage a positive result is obtained in about 90 per cent. of cases. If the central nervous system is affected, the cerebrospinal fluid may give a positive reaction, even though the blood serum is negative. In addition to its diagnostic value, the W.R. is also a valuable control in estimating the efficacy and result of treatment. Flocculation tests (such as the *Kahn* test, etc.) depend on the occurrence of visible flocculation of an antigen suspension when mixed with syphilitic serum. They can be performed more rapidly than the W.R. and are valuable con-

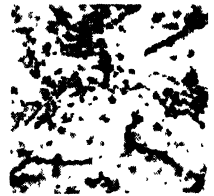


Fig. 27.—*Treponema pallidum* in the liver of a baby.

¹ Syphilis derives its name from a poem by a physician, Girolamo Fracastoro, published in Venice in 1530. The poem tells of the shepherd Syphilus, who was struck down by the disease as a punishment for neglecting the worship of Apollo.

On his return from Haiti to Barcelona in 1493, Columbus "brought back syphilis, parrots, and rare plants". The King and Queen of Spain received him with the highest honours.

Fritz Schaudinn, 1871–1906. A Prussian Zoologist.

August von Wassermann, 1866–1925. Director, Institute for Experimental Therapy, Berlin.

Reuben Leon Kahn, Contemporary. Bacteriologist, University of Michigan Medical School and Hospital, Ann Arbor, Michigan, U.S.A.

firmatory tests. Other conditions which render the serum positive to the W.R. are yaws, glandular fever, and leprosy. Weak reactions are sometimes obtained in miliary tuberculosis, malaria, typhus, vaccinia, relapsing fever, and advanced malignant disease.

Owing to these false positive reactions new tests have been developed and are very useful. The *Treponema Immobilisation Test* (T.P.I.) depends on the immobilisation by an antibody in patient's serum of a *Trep. pallidum* suspension from infected rabbit testes. It is sensitive and specific but technically difficult. The *Reiter protein complement-fixation test* (R.P.C.F.) introduced in 1949 employs as antigen a protein extracted from non-pathogenic treponema. It is nearly as sensitive and specific as the T.P.I. test and is technically simpler and more suitable for routine use. These treponemal tests greatly reduce diagnostic errors due to false positive Wassermann reactions.



FIG. 28.—Primary chancre of the tongue. On palpation its edges felt hard. The submaxillary lymph nodes were enlarged. A scraping revealed *Treponema pallidum*.

Primary sores on the lips usually result from kissing, and one case is recorded in which a gentleman with secondary ulceration of the mouth infected five young ladies at a dance, each of whom developed a chancre on the lip. Dental surgeons and obstetricians are particularly prone to inoculation on the fingers, but 'syphilitic onychiæ' are less common than formerly owing to the use of rubber gloves and more rigorous surgical cleanliness.

A primary sore is due to inoculation through a minute abrasion, and is first noticed as an indurated papule. Associated endarteritis leads to ulceration, and a typical Hunterian chancre develops. The ulcer presents a definite margin, with characteristic induration due to lymphocytic infiltration. In the case of vascular structures, such as the lip, considerable œdema of the underlying tissues is present. In about 20 per cent. of cases chancres are multiple (fig. 29), either as a result of infection of two or more abrasions, or owing to auto-inoculation of an apposing surface by the primary sore. The regional lymph nodes become enlarged

Clinical Features.—Acquired syphilitic manifestations are roughly divisible into three stages.

Primary Stage.—In 96 per cent. of cases the primary sore or chancre is on the genital organs.

In the male the chancre is usually obvious, but in the female a primary sore on the inner aspect of the vulva or on the cervix is often unnoticed by the patient, and the infection is likely to progress well into the second stage before its real nature is recognised. Extragenital chancres occur on the lips, usually the upper (fig. 529), the tongue (fig. 28), the tonsil, the anal margin, the nipple, the fingers and, rarely, in other situations.

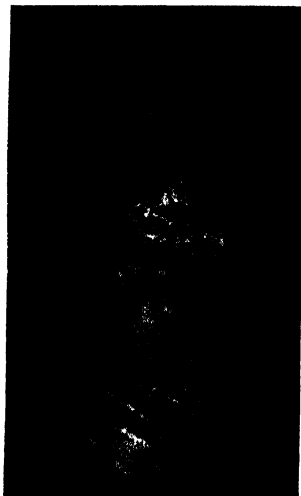


FIG. 29.—Multiple chancres with œdema. (Dr. Schneider, London.)

Hans Reiter, Contemporary. Formerly Professor of Hygiene, Friedrich Wilhelm University, Berlin, Germany.
John Hunter, 1728-1796. Surgeon to St. George's Hospital. To further his knowledge of venereal disease he inoculated himself with syphilis in 1767.

and firm ('shotty'), the skin is not reddened and there is no tendency to suppuration. In the case of a penile chancre, the dorsal lymphatics can often be felt to be 'wiry' when the subcutaneous tissue is rolled between the finger and thumb.

In some cases the initial papule disappears without ulceration, and the patient is genuinely unaware of its presence. If phimosis is present, secondary infection sometimes supervenes and the ulcer becomes acutely inflamed, with secondary acute lymphangitis and enlarged and tender lymph nodes. In virulent cases phagedena (p. 39) may develop.

A primary chancre must be distinguished from a traumatic ulcer (which follows irritation of an abrasion), a soft sore (p. 31), an early epithelioma, herpes, and, indeed, from any other lesion on the genitalia.

Secondary Stage.—Lesions occur from two to twelve months after infection, and are typically generalised and symmetrical. The W.R. is strongly positive in 98 per cent. of cases.

The general manifestations of the secondary stage include malaise, anæmia, a varying degree of pyrexia, and generalised enlargement of the lymph nodes. The epitrochlear, suboccipital, and posterior cervical lymph nodes are especially liable to be affected.

The main characteristics of a syphilitic eruption are the dull red or coppery colour, the absence of irritation, the symmetrical distribution, and the polymorphic nature, i.e. two or three types of rash are often present simultaneously. Papules appear at such mucocutaneous junctions as the anal margin, vulva, or angle of the mouth, or where cutaneous surfaces are constantly in apposition. As a result of their situation these papules become sodden, and frequently form large, foul, greyish masses, which are termed 'condylomata'. Later fleeting bone pains (osteoscopic) or periosteal nodes are often in evidence. Transitory and usually symmetrical effusions may occur in the larger joints. Iritis sometimes occurs, and gives rise to pain, lachrymation, circumcorneal congestion, and a sluggish and irregular pupil, and the colour may alter owing to œdema.

Tertiary Stage.—This commences some three years after infection, and may last throughout the patient's lifetime. The characteristic pathological changes are either diffuse gummatous infiltration, or local gumma formation, and almost any structure in the body is liable to be affected. In some organs both changes occur; thus a gummatous orchitis is sometimes associated with a local gumma, and a cirrhotic liver may harbour gummata.

A **gumma** is a mass of necrotic tissue surrounded by granulation tissue beyond which is a zone of fibrosis. Necrosis of tissue is partly due to toxins, and also to deficient blood supply resulting from endarteritis and surrounding fibrosis. If untreated, a gumma tends to enlarge and soften as the necrosis extends. If near the skin, hyperæmia and induration are evident. The centre of the indurated area then softens and eventually breaks down. The gumma discharges, and typically a wash-leather slough is seen at the bottom of the cavity, or on the floor of the ulcer, which is painless. The edges are characteristically sharply cut and circular in outline (fig. 38).

Gummatous ulcers, following subcutaneous gummata, are especially common on the leg, the backs of the thighs and forearms, and the face. A healed gumma leaves a 'tissue-paper' scar, which is silvery, supple and serpiginous, and sometimes surrounded by a pigmented area.

A gummatous ulcer of the leg has been mistaken for a varicose ulcer (fig. 30).

Treatment.—Public Health Regulations have greatly reduced the incidence of syphilis. Clinics are available in all large towns for treatment and for tracing contacts, and notices to this effect are posted in suitable public

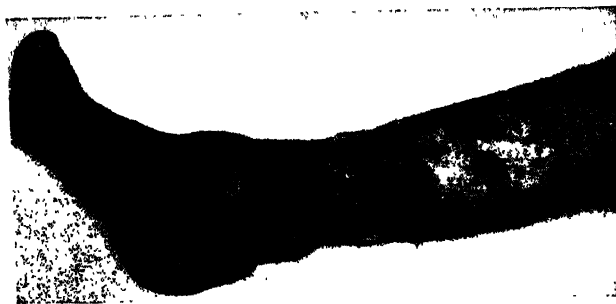


FIG. 30.—Gummatous ulcers, circular and serpiginous, with tissue-paper scars. (Case salvaged from a varicose vein clinic.)

places. In addition, lectures, diminished inebriety, propaganda work, and increased self-respect which is engendered by better education, all encourage sufferers to seek treatment. Of recent years, however, possibly associated with the 'new morality', syphilis is again

becoming much more common in this country. It is important to remember that homosexual practices may also transmit syphilis.

Treatment.—No antiseptic of any description should be applied to a suspected chancre until the serum has been examined bacteriologically. Pending the result a saline compress is applied.

Penicillin was first used in the treatment of syphilis in 1943 and has now supplanted all other remedies for the disease. It is necessary to maintain a continuous therapeutic level in the serum for seven to fourteen days. *Benzyl Penicillin* (*Penicillin G*) is the most effective type and causes the lesions to heal rapidly, crosses the placenta to reach the fœtus, and is effective in neurosyphilis. A single injection of *Procaine Penicillin*, 600,000 units intramuscularly, will maintain an effective blood level for twenty-four hours. Apart from penicillin sensitivity (p. 3), a reaction may occur in six to twelve hours which is characterised by fever, headache, bone and joint pains, and rigors. The cause of this reaction is unknown and fortunately it has no after-effects.

In some centres, injections of bismuth are still recommended to supplement the penicillin but, by and large, this practice is gradually being abandoned.

INHERITED (*syn.* CONGENITAL) SYPHILIS

This disease is arbitrarily divided into four grades of severity :

- (i) Abortion after the fourth month.
- (ii) Birth of a still-born and often macerated fœtus.
- (iii) The infant presents obvious syphilitic features, such as wasting, snuffles, skin eruptions.
- (iv) The child is apparently healthy, but subsequently develops syphilitic stigmata.

The following are the more important clinical lesions:

Mucous Membranes.—Inflammation of the mucoperiosteum of the nose causes a purulent discharge—known as 'snuffles'. Mucous patches, condylomata and eventually gummatous ulceration of mucous membranes and adjacent skin can occur. Radiating scars (rhagades) are sometimes left at the angles of the mouth.

Skin.—The commonest lesion is a bullous rash, known as 'syphilitic pemphigus', but a roseolar rash is not uncommon in the early months, especially on the buttocks.

Teeth.—The milk teeth erupt late and are ill-formed. The permanent incisors are peg-shaped, and often present a well-marked notch—Hutchinson's teeth (fig. 276). The central part of the crown of the first permanent molar is maldeveloped, especially in the lower jaw ('Moon's turreted molar').

Eye.—Iritis, sometimes accompanied by cyclitis, occurs in young children. The most characteristic lesion is interstitial keratitis, which usually appears between the ages of eight and sixteen years. The first indication is a 'ground-glass' appearance of

the cornea, associated with photophobia and lachrymation. Leashes of newly formed vessels appear later (salmon patches¹). One eye only is affected at first, but the second eye is subsequently involved. Prognosis should be guarded as opacities may remain in the cornea, or deeper-seated mischief may occur.

Ear.—Nerve deafness sometimes develops about puberty, and is often associated with interstitial keratitis.

Bones.—See p. 235.

Joints.—Clutton's joints are described on p. 282.

Other Organs.—A diffuse interstitial fibrosis occasionally involves the lungs. Syphilitic cirrhosis of the liver and splenic enlargement are not uncommon. Orchitis may be bilateral, and if occurring before puberty results in impotence.

Four typical lesions which shortly precede or occur at puberty are Clutton's joints, interstitial keratitis, otitis interna and orchitis epitomised as 'the halt, the blind, the deaf and the impotent'.

Treatment.—Prophylactic treatment is essential, and as a rule the mother is very tolerant to treatment during pregnancy. A healthy child is usually the reward of efficient ante-natal treatment. Treatment of an infected child should be instituted immediately after birth. A course of penicillin is prescribed in conjunction with cortisone should interstitial keratitis develop.

YAWS (FRAMBOESIA)

This is a contagious disease occurring endemically in tropical countries and caused by *Treponema pertenue* which is an organism indistinguishable from *Treponema pallidum*.² Infection usually occurs in childhood simply by contact and the organism enters the body by minor abrasions, commonly on the legs but sometimes on the arms or elsewhere. The Wassermann reaction (p. 27) is strongly positive.

As in the case of syphilis, three stages are recognised. The primary sore is usually on the leg, foot, or ankle. The secondary stage follows in four to twelve weeks, when cutaneous papillomata appear, especially on the limbs and ano-genital region. These papillomata ulcerate and appear as multiple pink raised areas resembling raspberries (hence the name Framboesia³). The tertiary stage is characterised by deep ulceration which when healed leaves tissue-paper scars resembling those of healed gummata. Yaws does not affect the cardiovascular and nervous system. The disease responds to penicillin but terramycin appears to be even more effective.

CHANCROID (*syn.* SOFT SORE)

This type of venereal disease is caused by the specific bacillus of Ducrey (*Hæmophilus ducreyi*). It is common in tropical countries, but in England is mainly confined to seaports. The incubation period is short, and in two or three days a vesicle appears, which becomes infected and breaks down to form an ulcer about a week after infection. The sores are commonly multiple and painful, and are associated with enlargement of the inguinal lymph nodes. Suppuration usually follows (bubo), and aspiration of pus through healthy skin is preferable to incision, as sinuses heal but slowly. Venereal sores are sometimes due to both *H. ducreyi* and spirochætes, so in all cases a search must be made for the latter organisms.

An uncomplicated soft sore persists and enlarges unless adequate treatment is prescribed. As the organism is penicillin resistant, sulphonamide and streptomycin are prescribed, and a five-days' course usually suffices.

GONORRHOEA

This venereal disease is caused by *Neisseria gonorrhœa*—a Gram-negative kidney-shaped coccus occurring in pairs. In pus from early infections the

¹ Not to be confused with the salmon-patch birthmark (p. 103).

² The real answer to 'What's Yaws?' is 'Syphilis'. ³ Framboise (Fr.) = raspberry.

Henry Clutton, 1850–1909. Surgeon, St. Thomas's Hospital.

Augusto Ducrey, 1860–1940. Professor of Dermatology, Pisa, Italy, isolated the bacillus in 1889.

Albert Neisser, 1855–1916, Professor of Skin and Venereal Diseases, Breslau University, recognised the gonococcus in 1879.

cocci are easily found on Gram staining; large numbers may be seen inside the pus cells. In chronic cases, especially in females, the gonococci become scanty and secondary invaders multiply, so that microscopic diagnosis becomes difficult or impossible. Bacteriological diagnosis should therefore be confirmed by culture. Unfortunately, the culture technique is difficult, but if it is not done the diagnosis will be missed in many cases, especially in women.

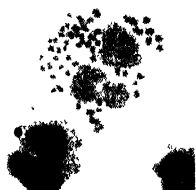


FIG. 31.—*Neisseria gonorrhoeae* (Gram's stain).

The complement-fixation test is of value for confirming the diagnosis in chronic and systemic infections, e.g. arthritis, but is usually negative in patients treated by antibiotics early in the infection.

IN THE MALE

Acute Stage.—The disease manifests itself two to ten days after sexual intercourse with an infected partner. The early symptoms are itching and redness of the external urethral meatus, the lips of which are sticky. A viscid discharge appears, which soon becomes thick and yellowish. Anterior urethritis develops within a day or two, with the characteristic symptom of scalding pain on micturition. Acute retention is a very rare complication. Malaise and slight elevation of temperature are present during the acute stage, and the inguinal lymph nodes are sometimes tender. After ten to fourteen days acute symptoms abate, but the discharge persists.

LOCAL COMPLICATIONS.—*Anterior Urethritis.*—Folliculitis following infection of glands of Littre is a common complication. Balanitis is sometimes troublesome, and chordee (Chap. 48) may result from inflammation of the corpus spongiosum or corpora cavernosa. Cowperitis occasionally occurs on one or both sides.

Posterior Urethritis.—This complication is liable to occur at any time, either from extension of the infection or ill-advised treatment, such as the passage of instruments. It is recognised by the frequency or urgency of micturition, with slight hæmaturia at the end of the act, aching in the perineum, painful erections and turbidity of a second specimen of urine after the first flow has washed out the anterior urethra.

Acute prostatitis (Chap. 47) causes rectal and perineal pain which is worse on defæcation. Retention of urine is likely to occur if suppuration ensues. The inflamed prostate is easily palpable per rectum. Acute vesiculitis causes frequent and painful emissions of purulent or blood-stained semen. Epididymitis usually occurs from the third to the fifth week, and is preceded by pain in the groin or lower abdomen due to inflammation of the vas deferens. Basal cystitis is common and causes frequency and pain at the end of micturition.

Chronic prostatitis (Chap. 47), with which is associated chronic vesiculitis, is a common cause of persistence of infection. Strictures (Chap. 48), formerly common, are becoming increasingly rare. They are due to sub-epithelial inflammation of the wall of the urethra, or chronic folliculitis.

'Gonorrhœal' warts occasionally occur on the glans or prepuce, and are due to an associated virus infection.

METASTATIC COMPLICATIONS.—Infection of joints and fibrositis may occur in neglected cases. Endocarditis occurs as a rare complication, and is associated with pyæmic abscesses. Iridocyclitis sometimes occurs in chronic cases, and necessitates repeated instillation of atropine. Most cases respond favourably to cortisone.

Chronic or Latent Stage.—The discharge is often very inconsistent and may occur only after such events as undue exercise or alcoholic excess. Typically, a 'morning dewdrop' or 'gleet' appears, which is thick and whitish in colour. Massage of the prostate and vesicles and examination of any expressed fluid will probably reveal latent infection. Partial emptying of the bladder followed by prostatic massage and completion of the act is a valuable test for posterior urethritis and prostatitis. Any threads which appear should be examined bacteriologically.

Urethroscopic examination may reveal folliculitis, erosions, abscesses and other abnormalities, and also allows the application of local treatment.

IN THE FEMALE

The early symptoms are much less pronounced than in the male. Infection usually commences in the urethra or cervix, and Bartholin's glands are infected in 2 per cent. of cases.

The symptoms of acute infection include a sensation of heat and discomfort of the vulva and pain on micturition, but in about 30 per cent. of cases symptoms are negligible. Should the cervix become infected, a blood-stained discharge is noticed and backache follows.

Complications.—Vaginitis is common in children who are accidentally infected, but adults usually escape. Cervicitis sometimes occurs spontaneously, or is encouraged by unwise instrumentation. Salpingitis, which may be accompanied by oöphoritis or peritonitis, is a dreaded complication which is apt to cause sterility. Proctitis often occurs, and is commoner than in males owing to the greater ease of infection.

Chronic or Latent Stage.—Chronic gonorrhœa occurs as urethritis, cervicitis, or Bartholinitis, and any discharge from these organs must be meticulously examined. The symptoms accruing from chronic infection are very slight and the patient may merely notice an occasional yellowish discharge.

Local Complications.—Chronic endometritis occasionally occurs, and results in menorrhagia, metrorrhagia and mild dysmenorrhœa, associated with backache. Salpingitis is often quiescent, but exacerbations are liable to follow sexual excess, debility or labour. Warts are not uncommon and are sometimes large and numerous.

Treatment.—Procaine penicillin is commonly given immediately the diagnosis is assured, and one injection of 600,000 units is usually curative. However, penicillin-resistant strains are increasing in number, and at one clinic, in spite of 600,000 units for five days, 20 per cent. of patients had positive smears or relapsed shortly after treatment. For patients who relapse or fail to be cured, an alternative drug should be used after the sensitivities of the strain of gonococcus have been ascertained. The patient is warned of the risks of conjunctivitis and transmission of infection, and he is kept under

observation for three months, the blood being tested monthly for syphilis, as penicillin may mask a syphilitic infection or prolong the incubation period.

OPHTHALMIA NEONATORUM

Infection by the gonococcus at birth is an important cause of blindness. The incubation period is twenty-four to forty-eight hours, and is followed by chemosis, lachrymation and purulent discharge. Corneal ulceration and sloughing may follow in neglected cases. If infection occurs, the eye must be irrigated with penicillin (10,000 units in 1 ml.) at frequent intervals, and intramuscular penicillin is administered.

NON-GONOCOCCAL INFECTIONS OF THE URINARY TRACT

Non-gonococcal urethritis, of various types, is described in Chapter 48.

CAT-SCRATCH FEVER

This is a distinct clinical entity with a world-wide distribution, and is probably due to a virus of the lymphogranuloma-psittacosis group. Localised inflammation occurs at the site of the lesion, associated with fever, malaise, and anorexia. This subsides in a few days, but from two to several weeks later the regional lymph nodes become enlarged. Suppuration often occurs, but the pus is sterile, and after evacuation the abscess subsides. Diagnosis is usually suggested by unilateral involvement of lymph nodes and the history of cat scratches. It is confirmed by a skin test with antigen prepared from human lymph-node pus. This distinguishes the condition from chronic pyogenic or tuberculous adenitis with which it is often confused. Broad-spectrum antibiotics have been tried; they do not influence the disease, though they may reduce the degree of fever.

Lympho-granuloma Inguinale is described in Chapter 41.

Granuloma Inguinale is described in Chapter 48.

Orf (Contagious Pustular Dermatitis of Sheep).—A virus disease of sheep transmitted to humans who handle infected animals and their skins or carcasses. It forms granulomata on the hands.

PARASITIC DISEASES

Filariasis is a disease widely spread through tropical and sub-tropical countries. It is due to a nematode worm, *Filaria sanguinis hominis* which is transmitted by a mosquito (*Culex fatigans*). Once in the human body, the female worm finds its way to the lymphatics and lymph nodes (especially the inguinal group). It attains sexual maturity in six to eighteen months and produces vast numbers of microfilariae, so that there may be 50 millions in the blood at one time. Obstruction of the lymph vessels ensues and this is manifested in (1) varicosities of the lymphatic vessels producing chylous ascites and hydroceles and, sometimes, chyluria, and (2) solid oedema (elephantiasis) often affecting the legs, scrotum, and arms, though it may occur anywhere. The best method of treatment known at present is Hetrazan (diethylcarbamazine-citrate), which appears to sterilise or kill the adult female worm. Prevention by anti-mosquito measures is, of course, vitally important. Elephantiasis may require surgery (p. 151).

Hydatid disease is discussed on p. 673 and Chapter 34.

Bilharziasis is discussed in Chapter 46.

Trichiniasis is discussed on p. 317.

Amoebiasis is discussed in Chapter 34.

DERMATOMYCOSES

Superficial infections of closely related mycelial fungi occur in the skin, hairs, and nails. *Tinea pedis* (athlete's foot) is contracted from small pieces of infected skin on the floors of communal showers or changing rooms. The skin of the toe web becomes macerated, peels, and cracks. It is prone to secondary infection with the streptococcus giving rise to lymphangitis and a red brawny oedema of the lower leg. The nails become brittle, yellow, and irregular. Treatment is by prolonged use of the antibiotic griseofulvin with Whitfield's Ointment (benzoic and salicylic acid) locally.

**ORIENTAL SORE (*syn.* DELHI BOIL
BAGHDAD SORE, ETC.)**

This disease is due to infection by a protozoal parasite, *Leishmania tropica*, and is a common condition in Eastern countries which is occasionally imported to Western zones. An indurated papule appears on an



FIG. 33.—Enlargement of the liver and spleen due to amyloid disease. Amputation was performed through the left hip joint for osteomyelitis of the upper end of the femur, but sinuses persisted. He succumbed six months later.

exposed surface, usually the face. If untreated this breaks down to form an indolent ulcer (fig. 32), which eventually leaves an ugly, pigmented scar. The condition readily responds to intravenous injections of antimony tartrate, but very small lesions can be treated by carbon dioxide snow.



FIG. 32.—Oriental sore, boy aged eight years. (Professor Riyaz-i-Qadeer, F.R.C.S., Lahore.)

Amyloid Disease

This form of dysproteinæmia is becoming increasingly rare but may be seen in cases of chronic empyema, bronchiectasis, chronic tuberculosis, and rheumatoid arthritis. The abnormal protein is deposited in the walls of the arterioles and later in the larger vessels. Microscopically, methyl violet stains the infiltrated tissues a rose pink, while normal structures are stained blue. It specially affects the kidneys, intestine, liver, and spleen. Polyuria occurs due to infiltration of the glomeruli of the kidneys. Diarrhoea ensues owing to infiltration of the mucosal villi. The spleen and liver become enlarged and palpable. It is most important to bear in mind that this

disease is a possibility in anyone who has prolonged suppuration from whatever cause. The diagnosis can be made on rectal biopsy—this method is simple, safe, and reliable. If the source of the infection can be eradicated, early amyloid disease will resolve.

CHAPTER 3

ULCERATION AND GANGRENE

ULCERATION

AN ulcer is a discontinuity of an epithelial surface. There is usually progressive destruction of surface tissue, cell by cell, as distinct from death of macroscopic portions (e.g. gangrene or necrosis).

Ulcers are classified as *non-specific*, *specific*, and *malignant*: the two latter groups are discussed in appropriate chapters.

Non-specific ulcers are due to infection of wounds, or physical or chemical agents. Local irritation, as in the case of a dental ulcer, or interference with the circulation, e.g. varicose veins, are predisposing causes.

Trophic Ulcers.¹—These are due to an impairment of nutrition, which normally depends upon an adequate blood supply and a properly functioning nerve supply. Ischæmia and anæsthesia will therefore cause these ulcers. Ulcers of the tips of the fingers occur in chronic vasospasm (Raynaud's disease), and in syringomyelia. In the leg, ischæmic ulcers occur around the ankle or on the dorsum of the foot. They are very painful and resist local treatment. Ulcers due to anæsthesia (from diabetic neuritis, spina bifida, tabes dorsalis, leprosy, or a peripheral nerve injury) are often called *perforating ulcers* (fig. 34). Starting in a corn or bunion, they penetrate the foot, and the suppuration may involve the bones and joints and spread along fascial planes upwards, even involving the calf.



FIG. 34.—Perforating ulcer in a diabetic.

The life-history of an ulcer consists of three phases; these are extension, transition, and repair. During the *stage of extension* the floor is covered with exudate and sloughs, while the base is indurated. The edge is sharply defined, and discharge is purulent and even blood-stained. The *transition stage* is occupied in preparation for healing. The floor becomes cleaner, the sloughs separate, induration of the base diminishes, and the discharge becomes more serous. Small reddish areas of granulation tissue appear on the floor, and these link up until the whole surface is covered (fig. 35). The *stage of repair* consists of the transformation of granulation to fibrous tissue, which gradually contracts to form a scar. The edge of the ulcer becomes more shelving, and epithelium gradually extends from it to cover the floor (at a

¹ Trophē (Greek) = nutrition.

rate of 1 mm. per day). This healing edge consists of three zones—the outer of epithelium, which appears white, the middle one bluish in colour (where granulation tissue is covered by a few layers of epithelium), and the inner reddish zone of granulation tissue covered by a single layer of epithelial cells.

Local (topical) treatment of non-specific ulcers.—In addition to the general treatment (where applicable) of an underlying cause (e.g. diabetes, arterial disease), a legion of lotions and non-adhesive applications are used to aid the separation of sloughs, hasten granulation, and stimulate epithelialisation of non-specific ulcers of the skin. Hypochlorite solution (e.g. Eusol¹) and 0.5 per cent. silver nitrate are popular in the early stages, and 1 per cent. zinc sulphate solution (e.g. Lotio rubra) later. Local insulin (2 to 3 ml. of 20 units per ml.) floated on to the ulcer and dried in a current of oxygen directed through a funnel is also popular. Other applications include paraffin gauze, ointments incorporating zinc oxide, 1 per cent. hydrocortisone cream, and silver foil. Household vinegar (1:6) is efficacious against pyocyaneus. Excessive granulations, commonly known as 'proud flesh', need to be discouraged by excision, curettage, or by the application of a caustic, such as silver nitrate. Chronic or indolent ulcers often respond to infra-red radiation, short-wave therapy, or ultra-violet light.

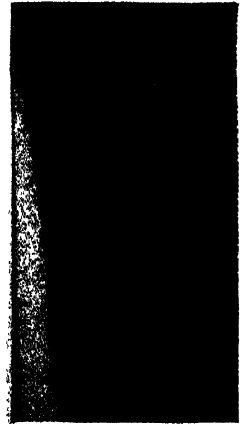


FIG. 35.—Healing venous ulcers of the leg.

Large ulcers in the healing phase should be covered by a free split skin graft as soon as the granulations are healthy (p. 114). Healthy granulations are flat, are below the level of the surrounding skin, are pink in colour, and do not discharge pus. In order to make excessive granulations healthy, they should be excised, curetted, or cauterised (see above), and firm wet dressings are applied four-hourly to keep them flat.

CLINICAL EXAMINATION OF AN ULCER

This should be conducted in a systematic manner. The following are, with brief examples, the points which should be noted:

Site, e.g. 95 per cent. of rodent ulcers occur on the upper part of the face.



Carcinoma typically affects the lower lip, while a primary chancre is usually on the upper.

Size, particularly in relation to the length of history, e.g. a carcinoma extends more rapidly than a rodent ulcer, but more slowly than an inflammatory ulcer.

Shape, e.g. a rodent ulcer remains circular until of a larger size than a carcinoma. A gummatous ulcer is typically circular (fig. 38), or serpiginous, due to the fusion of multiple circles. An ulcer with a square area or straight edge is suggestive of 'dermatitis artefacta' (fig. 36).

FIG. 36.—Dermatitis artefacta. This condition is due to self-mutilation, e.g. by the application of irritants, such as corrosives. The patient usually has a hysteric temperament, or litigation may be involved. The ulcer will heal if protected by a dressing which cannot be disturbed by the patient.

Edge (fig. 37).—A healing non-specific ulcer has a shelving edge. It is undermined and often bluish if tuberculous, vertically punched-out if syphilitic (fig. 38), rolled or rampart if a rodent ulcer, and raised and everted if an epithelioma.



FIG. 37.—1. Healing ulcer. 2. Tuberculous ulcer. 3. Rodent ulcer. 4. Epithelioma.

itic (fig. 38), rolled or rampart if a rodent ulcer, and raised and everted if an epithelioma.

Floor.—The floor is that which is seen by an observer, e.g. watery or apple-jelly granulations in a tuberculous ulcer, a wash-leather slough in a gummatous ulcer.



FIG. 38.—Gumma of the tongue. Note the punched-out appearance of the ulcer.

Base.—The base is what can be palpated. It may be indurated as in a carcinoma, or a primary chancre, or attached to deep structures, e.g. a varicose ulcer to the tibia.

Discharge.—A purulent discharge indicates active infection. A blue-green coloration suggests infection with *Pseudomonas pyocyaneus*. A watery discharge is typical of tuberculosis. It is blood-stained in the extension phase of a non-specific ulcer. *Bacteriological examination* may reveal colonisation by the coagulase positive staphylococcus. Spirochaetes are found in a primary chancre (p. 27).

Lymph nodes are not enlarged in the case of a rodent ulcer, unless due to secondary infection. In the case of carcinoma, they may be enlarged, hard, and even fixed. The inguinal nodes draining a syphilitic chancre of the penis are firm and 'shotty', but the submandibular nodes draining a chancre of the lip are greatly enlarged.

Pain.—Non-specific ulcers in the extension and transition stages are painful (except the anæsthetic trophic type). Tuberculous ulcers vary, though that of the tongue is very painful. Syphilitic ulcers are usually painless (an anal chancre is sometimes very painful).

General Examination.—Evidence of debility, cardiac failure, anæmia, or diabetes must be sought. A gummatous ulcer may be associated with other stigmata, e.g. chronic superficial glossitis.

Pathological examinations, e.g. biopsy, may confirm carcinoma. The W.R. or Mantoux test may be of value.

Marjolin's Ulcer. See p. 105.

GANGRENE

Gangrene implies death with putrefaction of macroscopic portions of tissue (fig. 39). It is commonly seen affecting the distal part of a limb, the appendix, a loop of small intestine, and sometimes organs such as the gall-bladder, the pancreas, or the testis. Note that the term 'necrosis' applies

mainly to the death of groups of cells, though it is extended to include bone, i.e. a 'sequestrum'. A 'slough' is a piece of dead soft tissue, e.g. skin, fascia, or tendon.

VARIETIES OF GANGRENE ACCORDING TO CAUSE

1. Symptomatic (or Secondary), due to arterial obstruction complicating existing disease, e.g. thrombosis of an atherosclerotic artery; embolus from the heart in auricular fibrillation or after coronary thrombosis; arteritis (with neuritis) in diabetes; thrombo-angiitis (Buerger's disease); arterial shut-down in Raynaud's disease, and ergotism; the effect of intra-arterial injections—thiopentone, nor-adrenaline, and cytotoxic substances. In respect of this list of causes, a classical mnemonic is the word 'RESTED', signifying: Raynaud, Ergot, Senile, Thrombosis, Embolism, Diabetes.

2. Infective: Boils and carbuncles, gas gangrene, gangrene of the scrotum, phagedena, cancrum oris et noma.

3. Traumatic:

(a) *Direct*, such as crushes, pressure sores, and the constriction groove on strangulated bowel.

(b) *Indirect*, due to injury of vessels at some distance from the site of gangrene, e.g. pressure on the popliteal artery by the lower end of a fractured femur (fig. 40), or the gangrenous contents of a hernial sac.

4. Physical, e.g. burns, scalds, frostbite, trench feet, chemicals, radium, X-rays, and electricity.

5. Venous Gangrene (p. 48).



FIG. 39.—Atherosclerotic gangrene.



FIG. 40.—Indirect traumatic gangrene. A spike of bone in a comminuted fracture of the femur had lacerated the popliteal artery.

through a variety of shades according to circumstances (purple, mottled, pallor, dusky grey) until finally taking on the characteristic dark brown, greenish black, or black appearance which is due to the disintegration of hæmoglobin and the formation of iron sulphide.

Clinical Types.—Dry gangrene and moist (wet) gangrene.

Dry gangrene occurs when the tissues are desiccated by gradual slowing of the blood-stream, and typically occurs as a result of atherosclerosis, e.g.

CLINICAL FEATURES OF GANGRENE

A gangrenous part lacks arterial pulsation, venous return, capillary response to pressure (colour return), sensation, warmth, and function. The colour of the part changes

senile gangrene. The affected part becomes dry and wrinkled, discoloured from disintegration of hæmoglobin, and greasy to the touch.

Moist gangrene occurs when venous as well as arterial obstruction occurs, or when the artery is suddenly occluded, as by a ligature or embolus. Infection and putrefaction always follow, and the affected part becomes swollen, discoloured, and the epidermis may be raised in blebs. Crepitus can sometimes be detected on palpation, due to infection by saprophytic gas-forming organisms.

Moist gangrene is manifest also in such conditions as acute appendicitis and strangulated bowel.

SEPARATION OF GANGRENE

Separation by Demarcation.—A ‘*zone of demarcation*’, between the truly viable and the dead or dying tissue, appears first. It is indicated on the surface by a band of hyperæmia and hyperæsthesia. Separation is achieved



FIG. 41.—Moist gangrene. Note the extension up the leg.

by the development of a layer of granulation tissue which forms between the dead and the living parts. These granulations extend into the dead tissue, until those which have penetrated farthest are unable to derive adequate nourishment. Ulceration follows, and thus a ‘*final line of demarcation (separation)*’ forms which separates the gangrenous mass from healthy tissue.

In dry gangrene, if the blood supply of the proximal tissues is adequate, the final line of demarcation appears in a matter of days and separation begins to take place neatly and with the minimum of infection (so-called separation by aseptic ulceration). Where bone is involved complete separation takes longer than when soft tissues alone are affected, and the stump tends to be conical as the

bone has a better blood supply than its coverings.

In moist gangrene there is more infection, and suppuration extends into the neighbouring living tissue, thus causing the final line of demarcation to be higher than in dry gangrene (separation by septic ulceration) (fig. 41). This is why dry gangrene must be kept as dry and aseptic as possible, and every effort should be made to convert moist gangrene into the dry type.

Vague Demarcation. Spread of Gangrene.—In many cases of gangrene from atherosclerosis and embolism, the line of final demarcation is very slow to form or does not develop. Unless the arterial supply to the living tissues can be improved (see Arterial Surgery, pp. 117–123), the gangrene will slowly spread to adjacent tissues or toes, or will suddenly appear as

'skip' areas farther up the limb. Signs of skipping should always be carefully looked for. Black patches suddenly appear, perhaps on the other side of the foot, on the heel, on the dorsum of the foot, in front of the ankle, and in the calf. *Infection*, being another cause of the spread of gangrene, may spread upwards beyond the line of separation along the lymphatic vessels or cellular tissue into healthy parts, and extensive inflammation then results. Except in diabetic gangrene without concomitant atherosclerotic obstruction, these forms of spread do not usually respond to efforts to save the limb, and an above-knee amputation is then necessary. To attempt local amputation in the phase of vague demarcation is to court failure, as gangrene reappears in the skin-flaps (die-back).

TREATMENT OF GANGRENE

General Principles.—Treatment depends largely upon the variety of gangrene, the site or organ affected, and the blood supply of adjacent structures. *A life-saving excision or amputation* is needed for such emergencies as a gangrenous appendix or loop of small intestine, gas gangrene, a badly crushed limb, or a rapidly spreading symptomatic gangrene. *A limb-saving attitude* is needed in most cases of symptomatic gangrene affecting hands and feet. The surgeon is concerned with how much can be preserved or salvaged.¹ With arterial disease, so much depends upon there being a good blood supply to the limb above the gangrene, or whether a poor blood supply can be improved by such measures as direct arterial surgery (embolectomy, grafting, or disobliteration), or interruption of the sympathetic nerve supply by a paravertebral block or sympathectomy. (These procedures are described in Chapter 9.) A good or improved blood supply indicates conservative excision and amputation. Poor blood supply, or failure to improve it, indicates amputation at a higher level, through absolutely healthy tissue.

General treatment includes that of cardiac failure, atrial fibrillation, and anæmia, in order to improve the tissue oxygenation. Oxygen inhalation (p. 80) is sometimes used, but hyperbaric oxygen in a compression chamber (where available) seems more promising (p. 80). A nutritious diet, essential in all forms of gangrene, and the control of diabetes when present, are additional items of care. Pain, especially night pain, may be difficult to relieve. Non-addictive drugs should be used, e.g. dihydrocodeine (D.F. 118).

Local Treatment.—(a) *Care of the affected part* includes keeping it absolutely dry. Surgical spirit, exposure, and the use of a fan may assist in the dessication. (The last two measures help to relieve pain.) The limb must not, under any circumstances, be heated.

(b) *Protection of local pressure areas*, e.g. the skin of the heel or the malleoli, otherwise fresh patches of gangrene are likely to occur in these places. A

¹ Sir William Fergusson (1808–1877), Surgeon, Royal Infirmary, Edinburgh, later Professor of Surgery, King's College Hospital, London, said that amputation is 'one of the meanest, and yet one of the greatest operations in surgery: mean, when resorted to where better may be done—great, as the only step to give comfort and prolong life'.

bed-cradle, padded or Sorbo rings, foam PVC blocks, or a 'sheepskin' (Acrilan)¹ are useful preventive aids.

(c) *Minor Surgical Toilet*.—Careful observation of a gangrenous part will show whether the lifting of a crust, or the removal of hard or desiccated skin, will assist in demarcation or the relief of pain.

Amputations of the Leg for Gangrene.—These may be conservative, radical (definitive) and semi-conservative (compromise), and as stated above, the indications depend upon the blood supply of the part of the limb immediately adjacent.

(a) *Conservative*.—There need be no undue haste to remove a dry, painless, gangrenous toe where the final line of demarcation is satisfactory. A diabetic or frostbitten part can in theory be left to separate. An excision through the line of demarcation is usually performed when separation is proceeding well or if there is localised infection. These wounds are left open to heal by granulation (fig. 42). If bone and tendons are to be sectioned at a higher level, this can be done through the cuff of skin and soft tissue. Special flaps are not fashioned in conservative amputations unless a portion of a foot or the forefoot is to be removed. A trans-metatarsal amputation (fig. 43) is

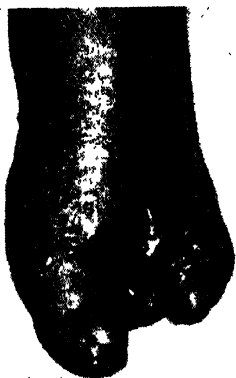


FIG. 42.—Conservative amputation for diabetic gangrene.

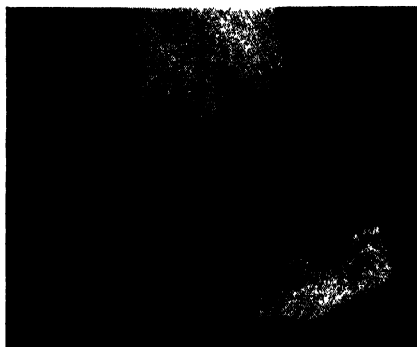


FIG. 43.—Transmetatarsal amputation for diabetic gangrene of the toes.

only possible when there is a viable long plantar flap extending almost to the base of the toes. This has to be wrapped round the severed forefoot and sutured to the dorsal skin. A long dorsal flap cannot withstand the subsequent trauma from a shoe.

(b) *Radical (Definitive)*.—Where conservative amputation is impossible because of failure of demarcation, spread of gangrene, failure of direct arterial salvage surgery or sympathectomy (p. 121), a mid-thigh amputation is indicated. Nowadays there is no statutory length of the stump. It is, in fact, essential to amputate high enough to ensure that the wound will heal soundly. This usually means a bony stump of some 6 to 8 inches (15 to 20 cm.) below the greater trochanter. Equal anterior and posterior flaps of skin and subcutaneous tissue and deep fascia are used. The muscles are divided at the level of bone section (quadriceps divided with the knee flexed, and hamstrings with the knee extended). A tourniquet is not used to control hæmorrhage, as in atherosclerotic and embolic gangrene there is surprisingly little bleeding owing to the obstructed main vessels. The sciatic nerve is cut cleanly across. It should not be ligated, though usually there is brisk bleeding from the artery to the nerve, which requires a ligature.

After sawing across the bone (the periosteum should not be stripped up) the leg is removed and full hæmostasis is secured. Muscles may be grouped together by suturing around (but not over) the stump before the skin flaps are carefully sutured, edge to

¹ A sheepskin or a large pelt made of washable fibre ('Acrilan' pad made by Northpads, London). The air pockets in wool make it specially resilient to pressure, so these pads may be used wherever areas need protection. The idea stems from cattle and sheep ranches where saddle-sores of horses are prevented by the interposition of a sheepskin.

edge. The stump is drained, either through the wound or via a separate incision, and mild suction is applied via a sealed drainage bottle.

(c) *Semi-conservative (compromise)* amputations about the knee are frequently performed when conservative amputations of the foot will clearly be of no avail (or have failed) and where blood supply in the region of the knee is satisfactory (an arteriogram may have shown this). These amputations are either below knee, through knee, or supracondylar (Stokes-Gritti). The blood supply *must* be adequate and it is well for the surgeon to advise the patient that in the event of there being little bleeding in the flaps and muscles, the radical above-knee amputation will be necessary.

After-care.—The stump is immobilised between sandbags for forty-eight hours, and there should be adequate sedation for pain. Thereafter the patient is encouraged by a programme of promotion to a full and active participation in the community. This programme entails exercises, sitting up in a chair, getting up on crutches, and finally the measurement for, fitting of, and rehabilitation with, an artificial limb (prosthesis). Early dressing and the removal of the drain require sedation, and an assistant to hold the limb gently but firmly between two hands. When the wound of an above- or below-knee amputation has healed, the patient holds his limb himself while he is taught how to bandage the stump in such a way as to mould it into the cone-shape necessary for fitting the prosthesis. Removal of the sutures on the fourteenth day depends upon satisfactory healing, and if there is any doubt on this score, they should be left in for three or even four weeks.

Complications.—*Early*, include the following: reactionary hæmorrhage, which requires return to the theatre for operative hæmostasis; a hæmatoma, which requires evacuation; infection, usually from a hæmatoma. Any abscess must be drained. Depending upon the sensitivity reactions of the organisms cultured, the appropriate antibiotics are given. Gas gangrene can occur in a mid-thigh stump—the organisms coming from contamination by the patient's fæces (p. 44).

Wound dehiscence and gangrene of the flaps are due to ischæmia, and a higher amputation may well be necessary. Fat embolism (p. 133) is another but uncommon sequel, occurring within the first two days.

Late.—Pain is usually the presenting symptom. It may be due to: unresolved infection, e.g. a sinus, osteitis, bone necrosis and sequestrum formation; a bone spur; a scar adherent to bone; an amputation neuroma from the outgrowth of nerve fibrils which become attached to skin, muscle or fibrous tissue; a phantom limb.

Phantom Pain.—Patients frequently remark that they can feel the amputated limb and sometimes that it is painful. The surgeon's attitude should be one of reassurance that these feelings will disappear. He should on no account foster the subject and talk about phantom-limb pain in front of the patient, as it is very refractory to treatment once it is established.

Other late complications include ulceration of the stump (pressure effects of the prosthesis or increased ischæmia). Rarely, an ulcer is artefacta (fig. 36). Some patients are troubled by cold and discoloured stumps, especially during the winter, and sympathectomy may be required.

ASPECTS OF CERTAIN VARIETIES OF GANGRENE

Atherosclerosis and embolism, the main underlying causes of symptomatic gangrene, are discussed in Chapter 8, together with Buerger's and Raynaud's disease.

Ergot is a common cause of gangrene among dwellers on the shores of the Mediterranean Sea and in the Russian steppes who eat rye bread infected with *Claviceps purpurea*. The extremities, and sometimes the nose and ears, are affected. It also occurs in migraine sufferers, who, for prophylactic reasons, unwittingly take ergot preparations over a long period. Overdosage can also occur following the ergot treatment of post-partum hæmorrhage.

Diabetic gangrene is due to three factors. These are trophic changes resulting from peripheral neuritis, atheroma of the arteries resulting in ischæmia, and excess of sugar in the tissues which lowers their resistance to infection (fig. 44). As in the senile type, gangrene is usually preceded by some slight trauma, and is either of the moist or dry variety.

The neuropathic factor impairs sensation, and thus favours the neglect of minor injuries and infections, so that inflammation and damage to tissues are ignored. Muscular involvement is frequently accompanied by loss of reflexes, and deformities. In some cases the feet are splayed and deformed (neuropathic joints). Thick callosities develop on the sole, and are the means

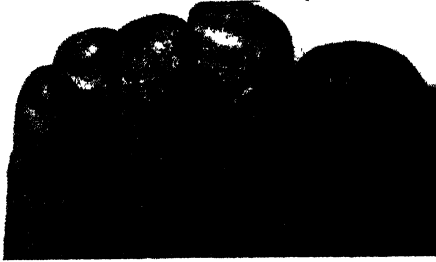


FIG. 44.—Early diabetic gangrene.

whereby infection gains entry, often following amateur chiropody. Infection involving fascia, tendon, and bone can spread rapidly upwards via subfascial planes.

Clinical examination and investigations include those on the urine and blood for diabetes. Palpable dorsalis pedis and posterior tibial pulses and the ab-

sence of rest pain and intermittent claudication imply that there is no associated major arterial disease (atherosclerosis). A bacteriological examination is made of any pus. An X-ray will reveal the extent of any osteomyelitis.

TREATMENT.—The diabetes must be brought under control by diet and insulin. The gangrene is treated on the lines already described on the previous pages, the accent being on conservatism if there is no major arterial obstruction. A rapid spread of infection requires drainage of the area by incision and the removal of any obviously dead tissue.

INFECTIVE GANGRENE

Gas gangrene (clostridial myositis), although uncommon, is a regular contributory cause of death in the case of accidents. It is now considered that the anaerobes are introduced into the wound from infected clothing more commonly than by soil, hence wounds of the thighs and buttocks are especially liable to infection owing to faecal contamination. Excessive hæmorrhage and the use of a tourniquet are predisposing causes, and, as in all cases of gangrene, the leg is more prone to be affected than the arm.

The causative organisms fall into two groups—those which break down starch and those which break down protein. *Cl. welchii* (*aerogenes capsulatus* or *perfringens*) is the most important member of the saccharolytic group; it is a Gram-positive anaerobe with a definite capsule. The proteolytic group include *Cl. sporogenes*, which splits protein into ammonia and sulphuretted hydrogen.

The diagnosis of true gas gangrene, i.e. gas in the tissues combined with gangrene, is essentially clinical. Pain, swelling, œdema, and toxæmia, usually develop within forty-eight hours. There is a rapid increase in the pulse-rate, a slight rise, or, in severe cases, a fall of temperature, and vomiting. The mental condition usually remains clear, and death occurs suddenly. (We have seen a patient die while engaged in completing a football forecast.) On examination of the wound, stitches are seen to be under tension, and, through the pouting edges, thin brownish fluid exudes, which possesses a sickly foul odour. The limb is tense and swollen, and palpation often reveals crepitus.

An X-ray will demonstrate the presence of gas in intramuscular planes, and is very useful in the diagnosis of early cases. Gas and foul fluid form in the cellular spaces; contractability of the muscles is soon lost, and their colour changes successively from the dull red of boiled ham to green and then black.

If septicæmia occurs, gas is produced in many organs, notably the liver, which at necropsy drips with frothy blood, and is well named a 'foaming liver'.

Treatment.—Prevention.—The essential prophylactic measure is early *debridement* of the wound, i.e. meticulous excision of dead and dying tissue. Partial suturing is sufficient, and secondary suture can be undertaken at a later date (p. 17). Prophylactic *penicillin* should also be administered.

Anti-gas gangrene serum ampoules contain 22,500 I.U. of polyvalent antitoxin (9,000 units *Cl. welchii*, 9,000 units *Cl. œdematiens*, 4,500 units *Cl. septicum*). A dose should be given intramuscularly in cases where muscles have been severely lacerated or foreign bodies have penetrated deeply into the tissues.

Treatment of Established Disease.—The patient's life depends upon:

(i) Preparation for immediate operation, with blood transfusion (or plasma if necessary).

(ii) Injection of 4 mega units of penicillin immediately, followed by $\frac{1}{2}$ mega unit four-hourly for 4–8 days.

(iii) Adequate excision of all affected groups of muscles, through *long* incisions in the limb (on both sides if necessary). If total clearance is not possible, amputation must be performed (e.g. disarticulation through the hip-joint for gas gangrene in an above-knee amputation stump).

(iv) Anti-gas gangrene serum (3 ampoules of polyvalent serum immediately, and repeated six-hourly I.V.).

(v) If available, oxygen drenching in a pressure chamber (hyperbaric oxygen, p. 80) reduces the toxin production of the organisms.



FIG. 45.—Comminuted fracture of humerus due to fragment of high-explosive, three days after injury in the battle of Dunkirk. Bubbles of gas are present under the triceps muscle.

Gas Infections.—Less severe forms of gas infection occasionally occur:

(i) *Local Type.*—Invasion is sometimes limited to a single muscle, which becomes necrotic while adjacent muscles escape.

(ii) A *gas abscess* frequently occurs if a contaminated foreign body is present in a wound (fig. 45). It subsides when the abscess is properly opened ('uncapped'), and the foreign body removed.

(iii) *Subcutaneous infection* is sometimes seen spreading for a considerable distance around a wound. Crepitus is easily palpable, and the skin becomes khaki-coloured as a result of hæmolysis. *Unless this condition is recognised, a needless amputation may be performed.* The only surgical treatment required is thorough drainage of the wound by multiple incisions into the affected subcutaneous tissues, down to, but not including, the deep fascia, otherwise infection may be carried into the underlying fascial planes or muscles.

Cancrum oris et noma is discussed on p. 490.

Carbuncles and boils are considered on p. 99.

TRAUMATIC GANGRENE

This variety of gangrene follows either local injury or occlusion of blood-vessels, and thus is either direct or indirect.

(i) **Direct traumatic gangrene** is due to local injury, and may arise as a result of crushes, or pressure, as in the case of splints or plasters, or bedsores.

Gangrene following a direct and severe injury, e.g. a street accident in which a heavy vehicle passes over a limb, is of the moist variety, and if the affected part is devitalised, removal without delay is indicated. As the tissues are presumably healthy, amputation is performed as close to the damaged part as will leave the most useful limb.

Bedsores (*syn.* decubitus ulcers) are either acute or chronic. The acute or *trophic* variety is associated with disease or injury of the spinal cord, and often progresses with alarming rapidity in spite of every care and attention.

The chronic or *postural bed sore* is predisposed to by five factors—pressure, injury, anaemia, malnutrition, and moisture (fig. 46). Prophylactic treatment is of the utmost importance. Thus pressure over bony prominencies is counteracted by a two-hourly change of posture and protection by foam P.V.C. blocks, or a 'sheepskin' (footnote, p. 42). A water-bed or a ripple-bed is sometimes desirable. Injury due to wrinkled drawsheets and in maceration of the skin by sweat, urine, or pus is combated by skilled nursing.



FIG. 46.—Postural bedsores over the sacrum.

A bed sore is to be expected if erythema, which does not change colour on pressure, appears. The part must be kept dry. An aerosol silicone spray may be used. Actual bedsores may either be treated by lotions, as described on page 37, or by exposure to keep them as dry as possible, with the appropriate antibiotic, given systemically. The hæmoglobin of the patient should be maintained at 100 per cent., by transfusions of packed cells if need be. If the patient

is young and otherwise healthy, free excision and sliding skin grafts are often successful (p. 114).



FIG. 47.—Indirect traumatic gangrene of a new-born infant's finger, accidentally caused by a thread of cotton. (Dr. Doyne Bell, London.)

(ii) **Indirect traumatic gangrene** is due to interference with blood-vessels, and some of the more important causes are as follows:

(a) Obstruction to artery and vein, as occurs in the loop of bowel contained in a strangulated hernia, or following pressure by a fractured bone on the main vessels of a limb (fig. 40) or digit (fig. 47).

(b) Thrombosis of a large artery, following injury.

(c) Ligation of the main artery of a limb, as after division by injury. The likelihood of gangrene then depends upon the sufficiency of the collateral circulation (p. 117).

Treatment directed to the cause, e.g. closed or open reduction of a fracture, will sometimes prevent the onset of gangrene. In cases of threatened gangrene the limb must be kept cool, so as to reduce metabolism to the minimum (p. 123). When gangrene is slow to develop, delay is sometimes advantageous in that a line of demarcation will indicate the level of vitality. If moist gangrene spreads rapidly, amputation is needed to safeguard healthy tissue and in some cases to save the patient's life.

PHYSICAL AND CHEMICAL CAUSES OF GANGRENE

Frostbite is due to exposure to cold, especially if accompanied by wind or high altitudes (e.g. explorers and airmen). It is also encountered in the elderly or the ragrant during cold spells. Pathologically, there is damage of the vessel walls, which is followed by transudation and oedema. The sufferer notices severe burning pain in the affected part, after which it assumes a waxy appearance and is painless. Blistering, and then gangrene, follows.

Treatment.—Frostbitten parts must be warmed *very gradually*. Any temperature higher than that of the body will be detrimental, as the affected part will merely rise in temperature equal to that of its surroundings. Many frostbitten limbs have been either stewed or roasted in ignorance, and gangrene thereby encouraged. The part should be wrapped in cottonwool and kept at rest. Friction, e.g. rubbing with snow, is contraindicated, as it damages the already devitalised tissues. Warm drinks and clothing are provided and powerful analgesics are required to relieve the pain which heralds the return of circulation. Paravertebral injection of the sympathetic chain (p. 122) may be helpful in relieving associated vasospasm (see footnote, p. 123). Amputations should be conservative (p. 42).

Trench foot is due to cold, damp, and muscular inactivity, and is predisposed to by tight clothing, such as garters, puttees, or ill-fitting boots. Prophylaxis is therefore of paramount importance. Numbness is followed by pain, which is excruciating when boots are removed. The skin is mottled like marble, and in severe cases blisters containing blood-stained serum develop, and moist gangrene follows. The pathology is similar to that of frostbite, and the treatment is the same.

Ainhum (fig. 48), a disease of unknown etiology, usually affects male Negroes. A fissure appears at the level of the interphalangeal joint of a toe, usually the fifth. The fissure becomes a fibrous band, which encircles the digit and causes necrosis. The treatment is amputation.

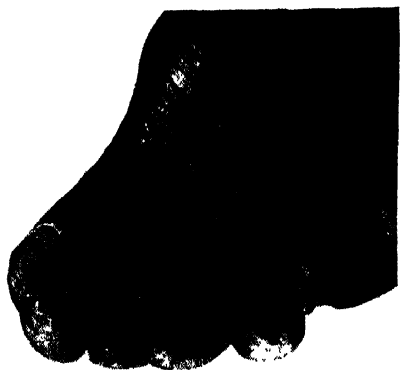


FIG. 48.—Ainhum (see text).

Intra-arterial injection of thiopentone is an occasional accident when a high division of the brachial artery results in one of its two terminal branches, usually the ulnar, passing superficially downwards in the antecubital fossa. Pulsation of the vessel, and the withdrawal of bright red blood should prevent this calamity.

Injection causes immediate and severe burning pain and blanching of the hand, usually accompanied by an interjection from the patient! The needle should be left in position, and 5 ml. of 2 per cent. procaine and/or 2 per cent. papaverine sulphate injected in order to obviate vascular spasm. Dilute heparin solution (p. 131) may also be given intra-arterially if the needle is in position.

If the needle is not in position, and the facilities are available, the artery may be exposed, opened, and any clots flushed out with dilute heparin solution. Then vein-patch repair (p. 121), and/or peri-arterial papaverine, or intra-arterial fibrinolytics (p. 132) may be employed. Brachial or stellate block must also be performed, and repeated if necessary. Even so, gangrene of one or more fingers may occur.

Chemical Gangrene.—Carbolic acid is the most dangerous, as anæsthesia masks the pain which occurs before the onset of gangrene. *Carbolic compresses should never be used*, for fingers have been lost by application of compresses even as dilute as 1:80. The gangrene is due to local arterial spasm. In addition there is danger of severe systemic effects from absorption of the carbolic. Local bicarbonate soaks should be applied. Later, excision of the slough and skin grafting are necessary.

VENOUS GANGRENE

A rare condition, due to extensive thrombosis in peripheral veins, there being normal arterial pulsations. It may occur in the toes and forefoot without any apparent cause, though it may be a manifestation of visceral neoplasm (Trousseau's sign, p. 142). It also occurs in peripheral thrombosis due to polycythæmia vera.

Treatment.—The limb is elevated, anticoagulant therapy commenced (p. 131), and conservative surgery performed when the final line of separation appears.

CHAPTER 4

TUMOURS AND CYSTS

THIS chapter concerns the general surgical pathology of tumours. Tumours arising in connection with special structures or organs are considered in their appropriate chapters.

A tumour is a new formation of cells of independent growth usually arranged atypically, which fulfils no useful function and has no typical termination. The term 'tumour' should be reserved for new-growths; its loose application to inflammatory swellings, such as Pott's puffy 'tumour' (p. 376), or to enlargement of an organ due to hypertrophy, should be abandoned.

Causation

Over 300 years ago it was stated that 'any kind of external irritation, whether from motion, heat, or acrimony, may cause cancer'. Natives of Kashmir are prone to develop carcinoma of the skin on the inner sides of the thighs and lower abdomen. This is due to their habit of keeping warm by squatting and hugging earthenware pots containing glowing charcoal (the pot being termed a *kangri*), with the result that the adjacent skin is irritated by heat and fumes.

It is common knowledge that women can swallow in comfort fluids at a considerably higher temperature than men can tolerate, which fact may explain the greater incidence of post-cricoid carcinoma in females.

'Chimney-sweeps' cancer (Chapter 49), and 'countryman's lip' (p. 457), are other examples of carcinoma due to chronic irritation.

Regeneration of tissue appears to encourage malignant changes in the newly formed cells, which are presumably in a state of instability. Primary carcinoma of the liver is sometimes seen in cases of cirrhosis, and apparently arises from the liver cells which are endeavouring to regenerate. Similarly, squamous-celled carcinoma occasionally occurs in a chronic ulcer (fig. 49, also Marjolin's ulcer, p. 105), and a fibrosarcoma arising in a scar is not uncommon. Viruses cause cell instability and are therefore implicated. A wart is the commonest example of a virus causing a tumour (see also Burkitt's tumour, p. 158). In some situations the site of fusion of embryonic elements encourages the development of carcinoma, e.g. carcinoma is prone to occur at the junction of the anterior two-thirds and the posterior third of the tongue, also carcinoma is not uncommon at the junction of the anal canal and the rectum.

Carcinoma is not hereditary. However experience shows that it appears to 'run in families'.

Tumours reproduce cells which are similar to those from which they arise, although if the tumour grows rapidly the resemblance becomes less obvious (*anaplasia*). It sometimes happens that the epithelium from which the tumour grows has already changed its characteristics. The gall-bladder is normally lined by columnar epithelium, but the presence of gall-stones may result in the epithelium undergoing squamous-celled *metaplasia*.



FIG. 49. — Squamous-celled carcinoma in a varicose ulcer (note everted edge).

CLASSIFICATION

The classification of tumours is fraught with difficulty owing to their varied and sometimes atypical appearances. Following the suggestion of Adami, tumours can be subdivided into two groups, teratomas and blastomas.

Teratomas are composed of cells of one individual within the tissues of a second individual (fig. 50). These tumours may arise from 'toti-

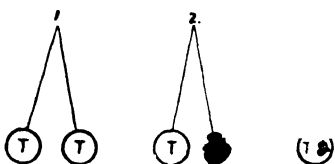


FIG. 50.—Twins and teratoma.

1. Normal twins.
2. One normal twin, the other an acardiac, anencephalic monster.
3. A teratoma—the twin brother's remnants are included in his normal brother.

potent' cells, or contain representative cells from all three embryonic layers: ectoderm, endoderm, and mesoderm. Teratomatous dermoids, for example, contain hair, teeth, muscle, gland tissue (see below).

Dermoid Cysts

Dermoid is a somewhat loose term given to cysts lined by squamous epithelium occurring in various parts of the body. Sebaceous cysts are lined by superficial squamous cells and should really be called 'epidermoids'.

(i) *Teratomatous dermoids* (see above) are found in the ovary, testis, retroperitoneum, superior mediastinum, and the presacral area. Malignant change occurs not infrequently in these tumours and it is wise to regard them all as liable to carcinomatous or sarcomatous change.

(ii) *Sequestration dermoids* (p. 62) are not teratomas and are formed by the inclusion of epithelium beneath the surface at places where lines of developing skin meet and join (external angular process, p. 481, root of nose, p. 482, branchial cysts, p. 518).

(iii) *Implantation dermoids* (p. 63) may follow puncture wounds, commonly of the fingers.

Blastomas develop from unipotent cells, and arise from any one of the three embryonic layers (e.g. neuroblastoma, p. 56).

INNOCENT OR MALIGNANT

By far the most important clinical differentiation is whether the tumour is innocent (benign) or malignant. This decision affects the whole future of the patient. Fortunately, it is usually possible to say with confidence which type of tumour is present but, occasionally, even the pathologist, with all the clinical and histological evidence to hand, will find it difficult.

An **innocent or benign tumour** is usually encapsulated, and does not disseminate nor recur after removal. Growth is uniform throughout the whole mass and symptoms are due to its size, position, and pressure (except in certain adenomas which secrete a *hormone* which may affect bodily functions). Frequently innocent tumours are multiple.

Malignant Tumours.—The characteristics of malignancy are: (a) Invasion of surrounding tissues, (b) Pleomorphism (variable shapes) of cells, (c) Rapid growth, (d) the tendency to metastasise to other parts of the body by the lymphatics and the blood-stream, (e) general weight loss and a rapid downhill path.

METHODS OF SPREAD OF MALIGNANT TUMOURS

(i) *Local Extension.*—Malignant tumours infiltrate adjacent tissues and spread by direct invasion. This feature is the most reliable evidence of

malignancy when the tumour is examined microscopically. Active growth occurs chiefly at the edge. Tissues are not always invaded in a uniform manner. Invasion takes place most readily along connective-tissue planes, whereas fascia or aponeurosis forms a temporary barrier. An avascular structure, such as articular cartilage, resists invasion to a remarkable extent.

(ii) *Blood-stream*.—This is the most common method of dissemination of a *sarcoma*, as the venous clefts, so typical of a sarcoma, readily permit malignant cells to enter the blood-stream. These malignant emboli are liable to be arrested in the lungs, where they form secondary deposits, sometimes accompanied by a blood-stained pleural effusion. It is probable that in some cases malignant cells grow along the pulmonary capillaries into the veins, and so reach the systemic circulation. Large veins are sometimes extensively invaded by sarcoma (fig. 51).



FIG. 51.—Intravenous spread of sarcoma.

Carcinoma disseminates less commonly by the blood-stream, although a carcinoma of the kidney may invade the renal vein, with the result that early secondary deposits may be found in the lungs. Cancer cells may be detected in the venous blood draining the region of a malignant tumour. Warren Cole has shown that during operation on a cancer of the breast, for instance, a shower of cancer cells pass into the axillary vein.

(iii) *Lymphatics*.—The spread of *carcinoma* along lymphatics occurs both by permeation and by embolism. In the former case the malignant cells grow along the lymphatic vessels from the primary growth; this may even occur in a retrograde direction. The presence of malignant cells stimulates perilymphatic fibrosis, but this does not stop the progress of metastases further along the lymphatic chain. Some structures, such as bones, are occasionally affected by lymphatic permeation.

In some instances, notably malignant melanoma, groups of cells may so overcome the surrounding fibrosis, as to give rise to intermediate deposits between the primary growth and the lymph nodes (fig. 52).

In the case of lymphatic embolism, cancer cells invade a lymphatic vessel and are carried by the lymph circulation to a regional node, so that nodes comparatively distant from the tumour may be involved in the early stages.

(iv) *Inoculation (Implantation)*.—Inoculation of carcinoma has been observed in situations where skin or mucous membrane is closely in contact with a primary growth. Examples of this 'kiss cancer' are carcinoma of the



FIG. 52.—Melanomatous deposits in sub-cutaneous lymphatics of the abdominal wall. (R.C.S. Museum).

lower lip affecting the upper, and carcinoma of the labium majus giving rise to a similar growth on the opposite side of the vulva.

Recurrence after operation is occasionally due to implantation of malignant cells in the wound. Examples of this mischance are the appearance of a malignant deposit in the bladder scar after suprapubic removal of a primary growth, and nodules of carcinoma in the scar of the incision after mastectomy.

(v) *Peritoneal*.—When the peritoneum is involved, cells from a carcinoma may spread like snowflakes all over its serous surface. This transcoelomic spread is specially notable when cells from a colloid carcinoma of the stomach gravitate on to an active ovary and give rise to malignant ovarian tumours (Krukenberg's tumour, Chapter 32) which may be the first manifestation of gastric malignancy.

GRADING AND STAGING OF MALIGNANT TUMOURS

Grading and staging are used to assess the degree of malignancy of the tumour as an indication of the prognosis, and may be used as a guide to determine the type and the extent of the treatment which is required.

Broders' Grading of Malignant Tumours.—Tumours are divided into four categories, according to the degree of differentiation shown by component cells. Squamous-celled carcinoma best illustrates these points, which include the degree of anaplasia, the absence or presence of prickle cells, and the number of mitotic figures.

Grade I. This is the least malignant, and not more than 25 per cent. of cells are lacking in well-marked differentiation. The remainder show keratinisation and obvious differentiation.

Grade II. From 25 per cent. to 50 per cent. are undifferentiated.

Grade III. From 50 per cent. to 75 per cent. are undifferentiated.

Grade IV. Over 75 per cent. of cells are undifferentiated, i.e. anaplastic and with mitotic figures, and are the most malignant.

This grading must not be taken too literally in estimating treatment or prognosis, e.g. different parts of the same tumour may show varying grades, and Grade I neoplasms may have disseminated widely, whereas a Grade IV tumour may still be localised. Anaplastic tumours are usually radio-sensitive, but commonly recur, whereas highly differentiated tumours tend to be radio-resistant.

Dukes' Staging.—This is a method of classifying the spread of carcinoma of the rectum and is described in Chapter 41.

TNM Classification of Carcinoma of the Breast.¹—This is a detailed clinical staging which is arrived at simply by the clinician ascertaining the following points during his examination of the patient: What is the extent of the primary Tumour within the breast? Are any lymph Nodes affected? Are there any Metastases? The information so obtained is scored as follows:

Tumour	Nodes	Metastasis
T ₁ - 2 cm. or less. No skin fixation.	N ₀ - No nodes.	M ₀ No metastasis.
T ₂ - More than 2 cm., but less than 5 cm. Skin tethered or dimpled. No pectoral fixation.	N ₁ - Axillary nodes movable (a) not significant, (b) significant.	M ₁ Metastases are present including involvement of skin beyond breast, and contralateral nodes.
T ₃ - More than 5 cm., but less than 10 cm. Skin infiltrated or ulcerated. Pectoral fixation.	N ₂ - Axillary nodes fixed.	
T ₄ - More than 10 cm. Skin involved but not beyond breast. Chest-wall fixation.	N ₃ - Supraclavicular nodes. Œdema of arm.	

¹ Adopted by the International Union against Cancer. TNM Classification is now being used in cases of bladder tumour.

Thus, for example, one patient may have a carcinoma which is $T_1N_0M_0$, while in another the extent of the disease may be $T_3N_2M_1$.

This scoring can be applied quite easily to the commonly used method of *clinical stage grouping* of carcinoma of the breast (described on p. 607), by the use of the following diagram in cases where there are no metastases (M_0).

BENIGN TUMOURS

There are many types of benign tumours and in this section only the main groups will be considered.

Papilloma

A papilloma consists of a central axis of connective tissue, blood-vessels, and lymphatics; the surface is covered by epithelium, either squamous, transitional, cuboidal or columnar, according to the site of the tumour.

The surface of a papilloma may be merely roughened, or composed of innumerable delicate villous processes, as in the case of those occurring in the kidney, bladder and rectum. In these situations papillomas resemble malignant tumours, as secondary growths arise by implantation, and, sooner or later, the tumour becomes frankly malignant (Chapter 46).

Other common sites for papillomas are the skin, the colon, the tongue and lip, the vocal cords (fig. 839), and the walls of cysts, particularly those in connection with the breast and ovary.

FIBROMA

A true fibroma (containing only fibrous connective tissue) is rare. Most fibromas are combined with other mesodermal tissues such as muscle (fibromyoma), fat (fibrolipoma), and nerve sheaths (neurofibroma), etc. Multiple tumours are not uncommon, as, for example, in neurofibromatosis (von Recklinghausen's disease, p. 56).

Fibromas are either *hard* or *soft*, depending on the proportion of fibrous to the other cellular tissue. The hard variety not infrequently occur on nerve sheaths. Soft fibromas are common in the subcutaneous tissue of the face in middle-aged and elderly people, and appear as soft brown swellings.¹

Desmoid.—This unusual type of fibroma occurs in the abdominal wall (Chap. 42).
Keloid (p. 116).

LIPOMA

A lipoma is a slowly growing tumour composed of fat cells of adult type. Lipomas may be encapsulated or diffuse.

Diffuse lipoma occasionally occurs in the subcutaneous tissue of the neck, from which it spreads on to the preauricular region of the face. The tumour is not obviously encapsulated, and gives rise to no trouble beyond being unsightly. It is excised if the patient wishes to improve his appearance.

	T ₁	T ₂	T ₃	T ₄
N ₀	I		III	
N ₁	II			
N ₂	III		III	
N ₃				

FIG. 53.—TNM scoring related to clinical stage grouping when there are no metastases.

¹ Oliver Cromwell, 'Protector' of England from 1653 until his death in 1658, was disfigured by one of these tumours which he called a 'wart', he insisted his painters should draw him 'wart and all'.

Encapsulated lipomas are among the commonest of tumours. The characteristic features are the presence of a definite edge and lobulation. If the proportion of fibrous tissue is not excessive, a sense of fluctuation may be obtained. These tumours have a widespread distribution, as they can occur in any part of the body where fat is found. As would be expected, a lipoma deeply situated is liable to be mistaken for other swellings, as difficulty arises in recognising the typical signs. Most lipomas are painless, but some give rise to an aching sensation which may radiate.

Multiple lipomas are not uncommon. The tumours remain small or moderate in size, and are sometimes painful, in which case the condition is probably one of *neurolipomatosis*. *Dercum's disease* (*adiposa dolorosa*), characterised by tender deposits of fat, especially on the trunk, is an associated condition.

Should the lipoma contain an excessive amount of fibrous tissue it is termed a *fibrolipoma*. In other cases considerable vascularity is present, often with telangiectasis of the overlying skin, in which case the tumour is a *nævolipoma*. A retroperitoneal

lipoma, or one in the thigh (fig. 54), occasionally undergoes sarcomatous changes. Myxomatous degeneration and calcification sometimes occur in lipomas of long duration.

Clinically, circumscribed lipomas are classified according to their situation:

(i) *Subcutaneous* are most commonly found on the shoulders (fig. 55) or back, although no part of the body is immune. It must be remembered that a lipoma is occasionally present over the site of a spina bifida. A careful examination should distinguish such conditions as a tuberculous abscess or a sebaceous cyst, which may superficially resemble a lipoma. Subcutaneous lipomas occasionally become pedunculated, or the influence of gravity may cause the tumour gradually to change its position.

(ii) *Subfascial*.—Lipomas occurring under the palmar or plantar fascia are liable to be mistaken for tuberculous tenosynovitis, as the tough, overlying fascia masks the definite edge and lobulation of the tumour. However, the swelling is circumscribed, and wasting of muscles is negligible. Difficulty is encountered in complete removal as pressure encourages the tumour to ramify. Subfascial lipomas also occur in the areolar layer under the epicranial aponeurosis, and if of long duration they erode the underlying bone, so that a depression is palpable on pushing the tumour to one side (fig. 56).

(iii) *Subsynovial* arise from the fatty padding around joints, especially the knee. They are apt to be mistaken for Baker's cysts (p. 318) but are easily distinguished as, in distinction to a cyst or bursa, their consistency is constant whether the joint is in extension or flexion.

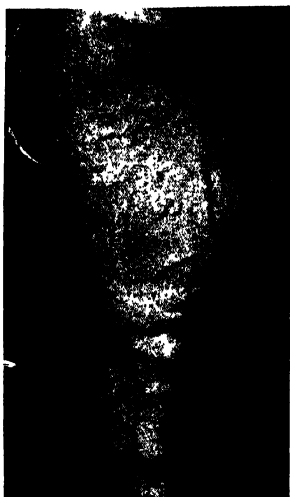


FIG. 54. — Liposarcoma of thigh which had been present for over twenty years. The scar indicates a previous attempt at removal.



FIG. 55. — A lobulated, subcutaneous lipoma.

(iv) *Intra-articular*.—The term 'lipoma arborescens' is somewhat misleading, as the condition is not neoplastic, but rather a fatty and fibrous infiltration of synovial tags.

(v) *Intermuscular*.—These occur particularly in the thigh or around the shoulder. Owing to transmitted pressure the tumour becomes firmer when the adjacent muscles are contracted. Weakness or aching results, owing to mechanical interference with muscular action. The condition is often difficult to distinguish from a fibrosarcoma, and exploration is necessary.

(vi) *Parosteal* occasionally occur under the periosteum of a bone, and are difficult to diagnose with confidence if deeply situated.

(vii) *Subserous* are not common, but are sometimes found beneath the pleura, where they constitute one variety of innocent thoracic tumour. A retroperitoneal lipoma may grow to enormous dimensions, and simulate a hydronephrosis or pancreatic cyst. A lipomatous mass is frequently found at the fundus of the sac of a femoral hernia, but this is a condensation of extra-peritoneal fat rather than a neoplasm.

(viii) *Submucous* occur under the mucous membrane of the respiratory or alimentary tracts. Very rarely a submucous lipoma in the larynx causes respiratory obstruction. A submucous lipoma occasionally occurs in the tongue. One situated in the intestine is likely to cause an intussusception, which is the first indication of its presence (fig. 57).

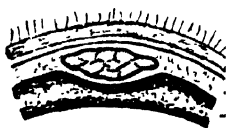
(ix) *Extradural*.—A lipoma is a rare variety of spinal tumour. Owing to the absence of fat within the skull intracranial lipomata do not occur.

(x) *Intraglandular*.—Lipomas occasionally arise from the fat within the lobules of the breast, and they have been found in the pancreas, and under the renal capsule.

As will be noted, lipomas are very widely distributed in the body. The 'universal tumour' is an appropriate term, which should be remembered when a swelling causes diagnostic difficulty.

Treatment.—If a lipoma is causing trouble on account of its site, size, appearance, or the presence of pain, removal is indicated.

During operation, any finger-like projections of the tumour into the surrounding tissue must also be removed. Although the tumour is relatively avascular, care is needed to obtain complete hæmostasis in the resulting cavity—drainage often is necessary—otherwise hæmatoma is common, which may be followed by infection and delay in wound healing.



SCALP
EPICRANIAL APONEUROSIS
AREOLAR TISSUE
SKULL

FIG. 56.—Erosion of the skull due to a subaponeurotic lipoma.



FIG. 57.—Intussusception caused by a submucous lipoma of the cæcum. (R.C.S. Museum).

ADENOMA

Adenomas arise in connection with secretory glands, and resemble to a greater or lesser extent the structure from which they arise. They are encapsulated tumours, and sometimes profoundly influence metabolism, as in the case of the thyroid, parathyroid and pancreas. Occasionally an adenoma contains a large proportion of fibrous tissue, e.g. the hard fibro-adenoma in the

breast, while in other situations, notably the pancreas and thyroid gland, cystic degeneration is common. Those arising from secretory glands of mucous membrane are liable to pedunculation, as in the case of rectal 'polypus'. Adenomas in certain situations tend to undergo malignant changes, e.g. those occurring in the thyroid gland.

NEUROMA

True neuromas are rare tumours, and occur in connection with the sympathetic system. They comprise the following types :

(a) *Ganglioneuroma*, which consists of ganglion cells and nerve fibres. It arises in connection with the sympathetic cord, and therefore is found in the retroperitoneal tissue, or in the neck or thorax. It usually occurs after the first decade, and is entirely innocent, causing symptoms merely by its size and position.

(b) *Neuroblastoma*, which is less differentiated than the ganglioneuroma, the cells being of an embryonic type (p. 50). The tumour somewhat resembles a round-celled sarcoma, and disseminates by the blood-stream. It occurs in infants and young children. It may occasionally undergo spontaneous remission.

(c) *Myelinic neuroma*, is very rare, being composed only of nerve fibres, for the ganglion cells are absent. They arise in connection with the spinal cord or pia mater.

False neuromas arise from the connective tissue of the nerve sheath and are really neurofibromas. The following varieties are described.

Local.—A single neurofibroma is usually found in the subcutaneous tissue, although occasionally a 'trunk neuroma' grows from a peripheral nerve or a cranial nerve (e.g., the acoustic tumour). The 'painful subcutaneous nodule' forms a smooth firm swelling, which may be moved in a lateral direction, but is otherwise fixed by the nerve from which it arises. Paræsthesia or pain is likely to occur from pressure of the tumour on the nerve fibres which are spread over its surface. Cystic degeneration or sarcomatous changes occasionally occur.

Generalised Neurofibromatosis (*syn. von Recklinghausen's Disease of Nerves*).—Cranial, spinal and peripheral nerves may all be diffusely or nodularly thickened (fig. 58). The overgrowth occurs in connection with the endoneurium. Associated pigmentation of the skin is common, and sarcomatous changes may occur.

Plexiform Neurofibromatosis.—This rare condition usually occurs in connection with branches of the fifth cranial nerve, although it may occur in the extremities (fig. 59). The affected nerves become enormously thickened as a result of myxo-fibromatous degeneration of the endoneurium. If occurring in the scalp, the underlying skull may be eroded, and in other situations the involved skin sometimes hangs down in pendulous folds. Plexiform neurofibromatosis is sometimes associated with the generalised type of neurofibromatosis. Sarcoma rarely develops.



FIG. 58.—Neurofibromatosis.

Elephantiasis Neuromatosa is a rare and congenital condition. The skin is coarse, dry, and thickened, resembling an elephant's hide, and the subcutaneous tissues become greatly thickened. If a leg is affected, the patient finds walking increasingly difficult.

AMPUTATION 'NEUROMAS'. — Fusiform swellings occur at the ends of divided nerves after amputation of a limb. These swellings consist of fibrous tissue and coiled nerve fibres (p. 43).

Hæmangiomas are described in Chapter 7 on p. 103.

GLOMANGIOMA (syn. GLOMUS TUMOUR)

These tumours arise from a cutaneous glomus. The glomera are composed of a tortuous arteriole which communicates directly with a venule, the vessels being surrounded with a network of small nerves. These specialised organs regulate the temperature of the skin, and are found in the limbs, especially the nail-beds. The tumour resembles a nævus, and is compressible. The associated pain is out of all proportion to the size of the tumour, which may be only a few millimetres in diameter. The pain is burning in nature and radiates peripherally, and is often more noticeable when the limb is exposed to sudden changes of temperature.

On section the tumour consists of a mixture of blood spaces, nerve tissue and muscle fibres derived from the wall of the arteriole (angio-myoneuroma). Large cuboidal cells are frequently seen (glomai cells). Glomal tumours grow very slowly, and do not become malignant. They should be excised.



FIG. 59.—Plexiform neurofibromatosis affecting the right arm.

HAMARTOMA

The term hamartoma is roughly translated from the Greek as a 'fault' or 'misfire' tumour, and its original meaning was 'missing the mark in spear throwing'. It is a developmental malformation consisting of a tumour-like overgrowth of tissue or tissues proper to the part. The possible range therefore is very wide, and the lesions are often multiple.

Common lesions that are hamartomas are benign pigmented moles, and the majority of angiomas and neurofibromas.

On rare occasions a malignant change occurs in a hamartoma, but for practical purposes the lesion is benign (Peters).

MALIGNANT TUMOURS

CARCINOMA

Carcinoma, which arises from tissues of ectodermal and endodermal origin, is the commonest form of malignant new-growth, and it appears to be increasing in frequency. This increase may be more apparent than real, and is explained by more accurate methods of diagnosis and the greater number of people who now survive to riper years. In some situations, such as the alimentary canal and bronchi, an actual increase has occurred during recent years. There is now no reasonable doubt that cigarette smoking is a potent factor in the causation of bronchial carcinoma which has increased tenfold in the past twenty years. Fortunately, owing to increasing avoidance of predisposing causes, other types of carcinoma, notably that of the skin, lip, and tongue, are less frequent than formerly.

Carcinomas are conveniently classified according to the type of cell from which they arise, i.e. glandular, squamous, or basal-celled.

(i) **GLANDULAR** is widely distributed, and commonly occurs in the alimentary tract, breast and uterus, and less frequently in the kidney, prostate, gall-bladder, and thyroid. Glandular carcinomas not only arise from secreting columnar epithelium, but also from ducts when the cells are cubical. The three types of glandular carcinoma are as follows:

(a) *Carcinoma simplex*, in which the cells are arranged in circumscribed groups, no glandular structure being recognisable. This type commonly occurs in the breast, and the majority of cells are spheroidal in shape.

(b) *Adenocarcinoma*, so called from the tendency of the cells to form acini, which resemble those of the gland from which they are derived. The alveoli are ductless, and the walls are composed of layers of cells which invade the surrounding tissues. This type is common in the stomach and colon. The cells of the primary growth, and even of the metastases, sometimes retain secretory powers.

(c) *Colloid*, which develops in tumours arising from cells which secrete mucin, and is a degenerative process. The mucin permeates the stroma of the growth, which appears as a gelatinous or semi-translucent mass. This type is typically seen in growths of the colon and stomach.

Glandular carcinoma is also subdivided into various types, e.g. encephaloid, scirrhus and atrophic scirrhus. These distinctions depend clinically on their rate of growth, and pathologically on the relative proportions of fibrous tissue and gland elements. Examples occur in the breast (p. 603).

(ii) **SQUAMOUS**—which arise either from surfaces covered by squamous epithelium, or as a result of *metaplasia*. Thus, prolonged irritation of the renal pelvis by stones causes the normal transitional epithelium to revert to a less differentiated type, so that squamous-celled carcinoma develops.

Squamous-celled carcinoma is particularly liable to occur as a result of chronic irritation (p. 49). The regional lymph nodes are likely to be invaded, but blood-borne metastases are rare. The lymph nodes occasionally undergo mucoid degeneration, to which secondary infection from the primary growth may be superadded, so that, if the skin gives way over the affected node, a glairy, semi-purulent fluid is discharged.

Macroscopically, squamous-celled carcinomas are either papilliferous or ulcerative. On section solid masses of polyhedral cells are seen, which invade the deeper structures. 'Cell-nests' are usually apparent in slowly growing cases, and are due to deeper cells becoming flattened and undergoing keratinisation. 'Prickle' cells are characteristic, and resemble the prickle cells present in the epidermis.

(iii) **BASAL-CELLED** (*syn.* Rodent Ulcer, p. 105).

SARCOMA¹

Sarcomas occur in connection with structures of mesoblastic origin. They differ from carcinomas, not only in their derivation, but also in their

¹ Sarkos, Gr. = flesh.

age incidence, as sarcomas are most common during the first and second decades. Moreover, sarcomas often grow with greater rapidity, and dissemination occurs mainly by the blood-stream. Microscopically, the cells of a carcinoma are arranged in masses or columns, whereas sarcoma cells are separated from each other. If the tumour grows slowly, the sarcomatous cells reproduce tissue similar to that from which the tumour originated, e.g. osteosarcoma or chondrosarcoma. In some cases a sarcoma develops in pre-existing benign tumours, such as a fibroma or a uterine fibroid, and also in bones which are affected by osteitis deformans (p. 246). Cases are on record in which a sarcoma, usually of bone, apparently resulted from an injury, which may lead to a medico-legal problem.

The macroscopic appearance of a sarcoma varies considerably. As the word implies, most tumours appear as a fleshy mass, but their consistency depends on the relative proportion of fibrous and vascular tissue. An avascular fibrosarcoma appears as a hard, almost white tumour, whereas a sarcoma of the breast is frequently soft, hæmorrhagic and often cystic, owing to mucoid degeneration. Hæmorrhage commonly occurs in a sarcoma, owing to the very thin walls of the veins, which in some places are represented merely by venous spaces. The absorption of extravasated blood is said to account for the irregularities of temperature which are so characteristic of a rapidly growing sarcoma.

Sarcomas of separate organs are considered in their appropriate chapters.

Fibrosarcoma.—This tumour occurs in scars, muscle sheaths (fig. 60) and as a fibrous epulis (p. 470). A fibrosarcoma of a muscle sheath presents



FIG. 60.—Fibrosarcoma arising from the sheath of the right pectoralis major. Infra-red photography reveals dilated veins over the tumour.

as an elastic or firm and slowly growing swelling. It either commences as a true fibroma, and gradually becomes sarcomatous, or is actually of low-grade malignancy from the beginning. Dilated veins over the tumour suggest malignancy, and if not obvious they may be demonstrated by infra-red photography (fig. 60). Malignant changes are hastened by incomplete efforts at removal. *The moral is that wide excision with surrounding healthy tissues should be practised in all cases.* This may mean amputation in the case of a limb.

These tumours are composed of spindle cells of varying lengths. In many cases it is difficult to distinguish with certainty, even microscopically, be-

tween a fibroma and a fibrosarcoma of low-grade malignancy. Fibrosarcomas not uncommonly arise in scar tissue, sometimes many years after the scar developed.¹

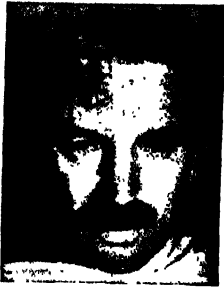


FIG. 61.—Fungating fibrosarcoma of the scalp. (T. A. Bouchier-Hayes, F.R.C.S.I., Dublin.)

If untreated, or if wide local excision is unsuccessful, a fibrosarcoma eventually fungates through the skin (fig. 61). Metastases are widely scattered, and, unfortunately, radiotherapy has but little effect on either the primary growth or on the secondary deposits.

Reticulosarcoma (*syn.* lymphosarcoma) arises in lymph nodes, tonsils, Peyer's patches or lymph nodules in the intestines. Lymph nodes of the neck or mediastinum are most commonly affected (p. 155).

Synovioma.—This rather uncommon tumour may arise in any synovial joint or tendon sheath, especially those of the hand. It appears as a soft, painless swelling, and sarcomatous changes may develop. Diagnosis can only be established by extirpation and biopsy of the tumour.

Nævus and Melanoma are described in Chapter 7 (p. 107).

ENDOTHELIOMA

The endothelial linings of blood-vessels, lymphatic spaces and serous membranes occasionally give rise to neoplasms. They are not usually malignant and dissemination occurs in a minority of tumours. They occasionally arise from the pleura and rarely from the pericardium or peritoneum. Although the original cells are flattened, they become spheroidal or cuboidal when neoplastic changes occur. The 'endothelioma' (meningioma) of the dura mater is thought by some to arise from the arachnoid membrane, which is not an endothelial structure (p. 381).

Peritheliomas are tumours arising in the endothelial lining of small blood-vessels or lymphatics. Carotid body tumours are probably of this nature (p. 532).

BENIGN—→MALIGNANT

Certain innocent neoplasms are prone to undergo malignant changes, and it is important, both for treatment and prognosis, to realise when this occurs. Some or all of the following changes may be recognised:

- (i) *Increase in size*—comparatively rapid enlargement is always suspicious, e.g. a soft fibro-adenoma of the breast which is becoming sarcomatous.
- (ii) *Increased vascularity*—dilated cutaneous veins, expansile pulsation of the tumour, or ulceration and bleeding in the case of a superficial growth.
- (iii) *Fixity*—due to invasion of surrounding structures, e.g. carcinomatous changes in a prostatic adenoma may cause 'tethering' of the rectal mucosa.
- (iv) *Involvement of adjacent structures*—carcinomatous changes in an adenoma of the thyroid should be suspected if the recurrent laryngeal nerve is implicated. Facial palsy suggests a malignant parotid tumour.
- (v) *Dissemination*—discovery of secondary deposits is occasionally the clue to malignancy. Thus the true nature of a doubtful lump in the breast is rendered evident if involved lymph nodes or osseous metastases are detected.

¹ Sir James Paget, 1814-1899, surgeon, St. Bartholomew's Hospital, London, described this type as a 'recurrent fibroid'.

Johann Peyer, 1653-1712. *Anatomist of Schaffhausen, Switzerland.*

Similarly, the discovery of a secondary deposit in a bone may lead to detection of a hypernephroma.

COCAINE MEDICATION (*mist. EUPHORIA*)

In the absence of pain chlorpromazine is often sufficient to render a patient tranquil.

Morphine (or a related alkaloid) is invaluable in cases of intractable pain which fail to respond to other analgesics. However, when weariness and depression eventually overtake a patient who is dying from any incurable disease cocaine is an eminently humane drug, which should be used more frequently. The euphoria which it engenders renders the patient comparatively cheerful, and relieves his mental and physical distress. Not only is the patient benefited, but the burden and sorrow of those who watch by the bedside is correspondingly lightened.

The formula used at The Royal Marsden (formerly Cancer) Hospital, London, is morph. hydrochlor gr. $\frac{1}{4}$ (15 mg.), cocaine hydrochlor gr. $\frac{1}{4}$ (10 mg.), gin 60 m. (4 ml.), honey 60 gr. (4 G.), aq. chlor ad $\frac{1}{2}$ oz. (15 ml.). This formula can be varied, e.g. if pain is not severe the morphia can be omitted or substituted by heroin, which is less likely to cause nausea and flatulence. Honey disguises the bitterness of cocaine. The mixture is similar to the 'Brompton cocktail'; more gin may be added in suitable cases, and towards the end the amount of cocaine may well be increased.

CYSTS

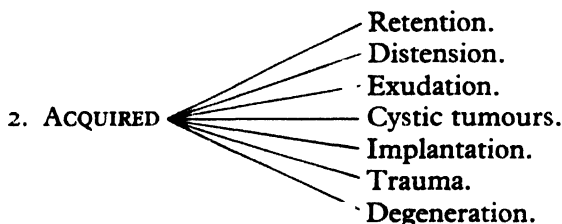
The word 'cyst' is derived from the Greek word meaning 'bladder'. The pathological term 'cyst' means a swelling consisting of a collection of fluid in a sac which is lined by epithelium or endothelium.

True cysts, are nearly always lined by epithelium. If infection supervenes, the lining may be composed of granulation tissue. Lymphatic cysts are lined by endothelium. The fluid is usually serous or mucoid in character. The colour varies from brown (due to staining by altered blood) to almost colourless. However, in epidermoid, dermoid, and branchial cysts the contents are like porridge or toothpaste, as a result of the accretion of desquamated cells. Cholesterol crystals are often found in the various types of fluid.

False Cysts.—Certain collections of fluid are not regarded as true cysts. They are usually exudation and degeneration cysts, and the lining, if present, is endothelial rather than epithelial. A pseudo-cyst of the pancreas is an encysted collection of fluid in the lesser sac. In tuberculous peritonitis fluid may be walled off in cystic form by adherent coils of intestine. Fluid may collect in the centre of a tumour (cystic degeneration), due to hæmorrhage or colliquative necrosis. This can also happen in the brain as a result of ischæmia, and an 'apoplectic cyst' is formed.

Types of Cyst.—Cysts are usually classified as follows:

- | | | |
|---------------|---|---|
| 1. CONGENITAL | { | Sequestration dermoids.
Tubulo-embryonic (tubulo-dermoid).
- Cysts of embryonic remnants. |
|---------------|---|---|



3. PARASITIC

Hydatid. Trichiniasis. Cysticercosis.

Congenital Cysts.—*The sequestration dermoid* is due to the fact that dermal cells may be buried along the lines of closure of embryonic clefts and sinuses by skin fusion. The cyst is therefore lined by epidermis and contains the paste-like desquamated material mentioned above. The usual sites are (a) the midline of the body—especially in the neck, (b) just above the outer canthus (the external angular dermoid) (p. 481), (c) in the anterior triangle of the neck (branchial cyst, p. 518).

Tubulo-embryonic (tubulo-dermoid) cysts occur in the track of an ectodermal tube used in development, e.g. a thyroglossal cyst from the thyroglossal duct; a post-anal dermoid from the post-anal gut. In the brain, ependymal cysts arise from the sequestration of cells of the infolding neurectoderm.

*Cysts of Embryonic Remnants.*¹—These arise from embryonic tubules and ducts which normally disappear or are only present as remnants. There are many examples in the genital and urinary systems. Congenital cystic kidneys are said to be the result of cyst formation of persistent mesonephric glomeruli and tubules (Chapter 45). In the male there are those which arise from remnants of the paramesonephric duct (Müllerian)—the hydatid of Morgagni, or from the mesonephric body and duct (Wolfian) (Chapter 45). Cysts of the urachus, and the vitello-intestinal duct are other examples of cysts of embryonic remnants.

Acquired Cysts.—*Retention cysts* are due to the accumulated secretion of a gland following obstruction of a duct. Examples are seen in the pancreas, the parotid, the breast, the epididymis, and Bartholin's gland. A sebaceous cyst starts with the obstruction of a sebaceous gland, but this is followed by the downgrowth and the accumulation of desquamated epidermal cells, thus making it an epidermoid cyst.

Distension cysts occur in the thyroid from dilatation of the acini, or in the ovary from a follicle. Lymphatic cysts and cystic hygromas are distension cysts. *Exudation cysts* occur when fluid exudes into an anatomical space already lined by endothelium, e.g. hydrocele, a bursa, or when a collection of exudate becomes encysted. These are false cysts (p. 61).

Cystic Tumours.—Examples are cystic teratomas (dermoid cyst of the ovary (p. 50)) and cystadenomas (pseudomucinous, and serous cystadenoma of the ovary).

Implantation dermoids arise from squamous epithelium which has been driven beneath the skin by a penetrating wound. They are classically found

¹ These cysts are not to be confused with teratomatous cysts (e.g. Dermoid, p. 50) which are cystic tumours.

Johannes Peter Müller, 1801–1858. Professor of Anatomy and Physiology, Berlin

Giovanni Batista Morgagni, 1682–1771. Professor of Anatomy, Padua, Italy.

Kaspar Friedrich Wolff, 1733–1794. Professor of Anatomy and Physiology, St. Petersburg (now Leningrad).

Caspar Secundus Bartholin, 1656–1738. Professor of Medicine, Anatomy, and Physics, Copenhagen, Denmark.

in the fingers of women who sew assiduously (fig. 62). The contents are therefore desquamated cell-debris which may undergo mucoid degeneration.

Trauma.—A hæmatoma may resolve into a cyst. This sometimes happens to hæmatomas of muscle masses in the loin and antero-lateral aspects of the thigh. They are located between muscle, fascial, or subcutaneous planes and contain straw- or brown-coloured fluid containing cholesterol crystals. They become lined by endothelium. Aspiration is only of temporary value, and a cure depends upon complete excision of the lining.

Degeneration Cysts.—These have already been discussed under false cysts.

Parasitic Cysts.—These are encysted forms in the life-cycle of various worms.

Hydatid cyst of the *Tænia echinococcus*.—This is described later according to the organ involved, e.g. liver, Chapter 34; lung, p. 673.

Trichiniasis.—Cysts of the *Trichina spiralis*, affecting muscle, p. 317.

Cysticercosis.—Cysts of *Tænia solium*. A disease of the pig, man being rarely affected. The cysts occur in any organ. They calcify and may cause clinical effects according to their situation, especially in the brain. Only those cysts which are actually causing symptoms should be excised.



FIG. 62.—Implantation dermoid.

Clinical Features of a Cyst.—The swelling usually has a smooth spherical appearance, though oval or loculated forms may be encountered. Fluctuation depends upon the pressure of fluid within; a tense cyst feels like a solid tumour, though careful palpation between two fingers may elicit a characteristic elasticity. In addition, a solid tumour is most hard at the centre; a cyst is least hard at the centre. If fluctuation is present, a cyst may be confused with a cold abscess or a lipoma. A cold abscess usually has a peculiar rim of thickening surrounding the soft centre. A lipoma may well test clinical acumen. *Transillumination*, while brilliantly clear in cysts containing serous fluid, does not really distinguish between a lipoma and a dermoid or branchial cyst. There is even an old axiom that 'when in doubt, hedge on fat'. According to circumstances, a test aspiration or excision reveals the true nature of the swelling.

Cysts may be painful, especially when infection or hæmorrhage causes a sudden increase in the intracystic tension. Quite often cysts appear to change in size for no apparent reason.

Effects of a Cyst.—These are according to site and size. As with benign tumours, a cyst may compress ducts and blood-vessels. For example, the common bile duct may be obstructed by a choledochal cyst, a renal cyst, or a hydatid cyst. The pelvic veins may be obstructed by an ovarian cyst, the patient presenting for treatment of her varicose veins. The sheer size of an ovarian cyst may so increase intra-abdominal tension as to bring the patient to hospital with symptoms of a hiatus hernia.

Cachexia Ovarica.—Enormous cysts are rarely seen nowadays; however



FIG. 63.—Cachexia
ovarica.

fig. 63 shows a patient who successfully underwent an operation for a giant ovarian cyst containing 15 litres of fluid. The site, size, and weight of the cyst combine to cause cachexia, lordosis, œdema of the legs, and an anxious appearance.

Complications.—Infection.—The cyst becomes tense and painful, and adherent to surrounding tissues. An abscess may form and discharge on the surface and result in an ulcer or a sinus (*viz.* Cock's peculiar tumour, p. 101). Healing will not occur until the whole lining of the cyst or the embryonic track is excised.

Hæmorrhage.—Sudden hæmorrhage, as may occur in a thyroid cyst, causes a painful increase in size. In this particular case breathing may be difficult because of pressure on the trachea.

Torsion may occur in cysts which are attached to neighbouring structures by a vascular pedicle. Ovarian dermoids are often brought to notice in this way as acute abdominal emergencies. The cyst (or cysts—they may be bilateral), turns to a purple or black colour as the venous, and then the arterial supply is cut off.

Calcification follows hæmorrhage, or infection, and is often the result of reaction to a parasite, e.g. hydatid cyst.

CHAPTER 5

HÆMORRHAGE. BLOOD TRANSFUSION. SHOCK

HÆMORRHAGE

TYPES OF HÆMORRHAGE

1. Arterial. Venous. Capillary.

Arterial hæmorrhage is bright red. Coming from the high-pressure side of the circulation, the blood spurts as a jet which rises and falls in time with the pulse. In protracted bleeding, particularly when quantities of intravenous fluids other than blood are given, it can become watery in appearance.

Venous hæmorrhage is a darker red and escapes as a steady jet from the low-pressure side of the circulation. The colour darkens still further from excessive oxygen desaturation when blood loss is severe, or in respiratory depression or obstruction. During an operation the appearance of blue-black venous blood requires the immediate attention of the anæsthetist.

Very rarely, if the above causes are clearly ruled out, cases of sulph- or met-hæmoglobinæmia will be uncovered (confirmed by spectroscopy).

Venous bleeding can be under increased pressure, as in asphyxia. Portal vein pressures (Chap. 30) are high enough to cause rapid blood loss, especially in portal hypertension. Pulmonary artery hæmorrhage is dark red (venous blood) at around 30 mm. Hg, and bleeding from the pulmonary veins is bright red.

Capillary hæmorrhage is a bright red, often rapid ooze. If continuing for many hours blood loss can become serious (as in hæmophilia, p. 76).

2. Primary. Reactionary. Secondary.

Primary hæmorrhage is that which occurs at the time of injury or operation.

Reactionary hæmorrhage may follow primary hæmorrhage within 24 hours (usually 4–6 hours) and is mainly due to rolling ('slipping') of a ligature or dislodgement of a clot. The precipitating circumstances are (a) the rise of blood pressure and the refilling of the venous system on recovery from shock, (b) coughing and vomiting which raise the venous pressure (e.g., reactionary venous hæmorrhage can occur within a few hours of thyroidectomy if coughing and vomiting cause acute congestion of the deep veins of the neck), (c) sudden or violent movements.

Penetrating wounds involving main veins in the thigh or groin are potentially fatal, as exsanguination may follow the removal of a first-aid dressing which has apparently controlled the bleeding (Butcher's thigh, p. 123). Such a wound should never be treated in a perfunctory manner; it requires careful examination and closure in an operating theatre. Venous hæmorrhage, whether primary or reactionary, can tax the skill of even an experienced surgeon.

Secondary hæmorrhage occurs after 7–14 days, and is due to infection and sloughing of part of the wall of an artery. Predisposing factors are pressure of a drainage tube, a fragment of bone, a ligature in an infected area, or cancer.

It is also a complication of arterial surgery and amputations. It is heralded by 'warning' hæmorrhages, which are bright red stains on the dressing, followed by a sudden severe hæmorrhage which may be fatal. A warning hæmatemesis may occur in the case of a peptic ulcer and is a danger signal which it is imprudent to ignore. In advanced cancer the erosion of a main vessel (e.g. carotid, uterine) by a locally ulcerating growth is a swift and merciful termination to the patient's suffering.

3. Internal (concealed) and External (revealed) Hæmorrhage.

Internal bleeding may be *concealed* as in ruptured spleen or liver, fractured femur, ruptured ectopic gestation, or in cerebral hæmorrhage. Concealed hæmorrhage may become *revealed* as in hæmatemesis or melæna from a bleeding peptic ulcer, as in hæmaturia from a ruptured kidney, or via the vagina in accidental uterine hæmorrhage of pregnancy.

SIGNS OF HÆMORRHAGE. PHYSIOLOGICAL AND PATHOLOGICAL EFFECTS (OLIGÆMIC SHOCK)

Acute Blood Loss.—Signs.—External bleeding is obvious, but a serious internal hæmorrhage must be recognised without delay by the general signs of blood loss. These are: *Increasing pallor and pulse rate, restlessness* and, in protracted bleeding, deep sighing respirations (*air hunger*). These are accompanied by a cold and clammy skin, empty veins, and later, by thirst, tinnitus and blindness.

The pulse volume will be low (thready pulse) as the blood-pressure falls, though it is important to remember that this is *not* a sign to be relied upon. Be very wary of a normal or raised blood-pressure in the face of other signs of hæmorrhage in an apparently healthy person, for collapse and death can occur suddenly. This phenomenon was noticed in the first world war (1914–18) by front-line research.

Effects.—In short, blood loss threatens the oxygen supply to, and therefore the life of, tissue cells. Pending the arrest of hæmorrhage and the replacement of blood, the function of vital structures such as the heart and brain stem is largely preserved by the increasing pulse rate and peripheral vasoconstriction (even though vasoconstriction may have deleterious effects on the liver and kidneys). Unchecked or untreated blood loss results in failure of the heart and vasomotor centre to maintain a sufficient perfusion of oxygen for their own purposes and death follows (the death cycle—see below).

Hæmorrhage means a loss of circulating blood volume from the whole of the vascular system. 60–70 per cent. of the blood volume is accommodated in the low pressure venules and veins and in the splanchnic vessels, and a loss of up to 10 per cent. (500–600 ml.) is adequately compensated for by venoconstriction and therefore the contraction of this large venous reservoir. (The spleen contributes only about 50 ml.) Further blood loss results in a diminished venous return and therefore a fall in cardiac output. The blood pressure, and therefore the perfusion of oxygen, is threatened, so a fall is prevented by increased heart rate and vasoconstriction. This is effected via sympathetic nervous activity, adrenaline, noradrenalin and the adrenal corticoids—which probably sensitise the vasoconstrictor nerve endings to noradrenalin. Clinically this compensation is shown as pallor, cold clammy skin, empty veins and increased pulse rate. Restlessness comes with the effects of anoxia in the mid-brain (mesencephalic reticulum) while changes in the rate and depth of respiration are due

partly to effects on the brain stem (chemoreflex), and partly in response to an increase in the airway dead space and metabolic acidosis.

The brain and the myocardium, which are the structures vital to the preservation of life in this emergency, are spared from vasoconstriction. The liver and kidneys, however, may suffer irreparable damage from the tissue anoxia caused by the intense and prolonged vasoconstriction in severe blood loss, with liver failure or acute renal failure claiming victims, even though the hæmorrhage is successfully treated (e.g. in ruptured abdominal aneurysm).

Vasoconstriction fails to maintain the blood pressure when blood loss increases beyond 20–30 per cent. Hypotension becomes severe with a 50 per cent. loss, and the perfusion of myocardium and the brain stem is affected. Thus the heart fails and vasoconstriction of the venous reservoir fails—each affecting the other in a vicious circle which ends in death.

Failure of vasoconstriction will occur sooner if any conditions causing vasodilation are present (alcohol, anaesthesia, infection, heating the patient up by too many blankets or a heat cradle).

Infants are intolerant of blood loss because a small amount may represent a considerable percentage of the total blood volume, and also because the peripheral circulatory resistance is normally high and so cannot be so effectively increased. In the elderly, arteriosclerosis affecting brain, heart and the peripheral circulation will hinder an adequate response.

Natural Blood Volume and Red Cell Recovery.—The recovery of blood volume commences within a few hours of hæmorrhage by the withdrawal of fluid from the tissues into the circulation. There is hæmodilution. Plasma proteins are replaced by the liver. Red cell recovery takes some five to six weeks. The iron content will be less than normal if stores are depleted or absorption is impaired (e.g. after gastrectomy, Chapter 32).

Chronic Hæmorrhage.—Examples of causes in surgical practice are bleeding hæmorrhoids, fibroids, carcinoma of the caecum, peptic ulcer. There is no diminution of the blood volume as there is time for plasma replacement, but red cell replacement lags behind (microcytic hypochromic anæmia) resulting in a state of anæmic hypoxia, requiring an increased cardiac output. These patients develop high-output cardiac failure; they must not be transfused with normal blood, but require packed cells instead. Acute hæmorrhage is poorly compensated in such cases, as oxygen carriage is already depleted.

CLINICAL AND LABORATORY MEASUREMENT OF HÆMORRHAGE

1. Bedside observation of the appearance of the patient is essential (see signs of hæmorrhage, p. 66). The severity of blood loss and shock can be gauged by feeling the temperature of the nose. A cold nose usually means that shock is severe.

2. Record keeping of the pulse rate and the blood pressure are also essential (in spite of the fact that the blood pressure can be misleading). Readings are taken at $\frac{1}{2}$ or $\frac{1}{2}$ hourly intervals during an emergency and thereafter 4-hourly. The patient is not roused from sleep for a blood pressure reading if the pulse is not raised.

3. Hæmoglobin Level.—This is estimated as grams per 100 ml., normal values being 12–16 gm./100 ml. There is no immediate change in hæmorrhage, but within the space of a few hours the level is lowered by the hæmodilution caused by the movement of tissue fluid into the vascular compartment

in order to restore the blood volume. The degree of dilution can be gauged by the hæmatocrit reading (PCV).

4. The hæmatocrit (fig. 64) is a stout glass tube 110 mm. long with a 2.5-3.00 mm. bore and it is graduated 0-100. It is filled up to the 100 mark with a small quantity (about 0.8 ml.) of oxalated or heparinised freshly drawn blood, and centrifuged at 3,000 revs/min. for 30 minutes. The packed cell volume (PCV) is then read from the millimetre reading gradations. The normal range for venous blood is: men, 40-56 per cent.; women, 35-48 per cent.; children, 32-44 per cent. The PCV can also be measured on capillary blood, using very small amounts, in the microhæmatocrit centrifuge. This is particularly useful in shocked patients where it is often difficult to obtain a sample from a vein.



FIG. 64.— A hæmatocrit.

As explained above, some hours after the start of hæmorrhage, the PCV will be lowered by hæmodilution, and the reading obtained will enable a correction to be made of the hæmoglobin level, so declaring the real degree of anæmia. In dehydration states and the plasma loss in burns, the PCV will be increased (hæmoconcentration), because the plasma volume is diminished without a concomitant loss of red cells and hæmoglobin.

5. Measuring Blood Loss

(a) *Blood Clot*.—Blood clot the size of a clenched fist is roughly equal to 500 ml.

(b) *Swelling in Closed Fractures*.—Moderate swelling in closed fracture of the tibia equals 500-1500 ml. blood loss. Moderate swelling in a fractured shaft of femur equals 500-2000 ml. blood loss (Ruscoe Clarke).

(c) *Swab Weighing*.—In the operating theatre, blood loss can be measured by weighing the swabs before and after use and adding the total so obtained (1 gm = 1 ml.) to the volume of blood collected in the suction or drainage bottles. In extensive wounds and operations, the blood loss estimated in this way is much less than the total amount of blood, plasma and water actually lost from the body. For operations such as radical mastectomy or partial gastrectomy it is necessary to multiply the swab weighing total by $1\frac{1}{2}$. For larger wounds, as in abdomino-thoracic or abdomino-perineal operations, multiply by 2 (fig. 65). *Washing swabs* and estimating the Hb content of the washings is another method.

OPERATION	SWAB WEIGHING	SWAB WASHING	BLOOD VOLUME
RADICAL MASTECTOMY	392	323	600
PARTIAL GASTRECTOMY	210	210	680
RIGHT PNEUMONECTOMY (INTRAPERICARDIAL)	485	212	919
DESOMPHAGETOMY (THORACIC ABDOMINAL)	553	540	1800

FIG. 65

(d) *Blood Volume Determinations*.—Either the volume of circulating plasma, or the volume occupied by the red cells, must be measured first. The hæmatocrit reading will give the ratio of plasma to red cells, and thus the total blood volume can be arrived at. *Methods*.—The measurement of the circulating plasma or red cell volume involves the injection of a known quantity of a substance which will be evenly diluted as it mixes with, and circulates in, the blood. Radioactive iodinated serum albumin

(RISA) is used to give plasma volume, and red cells 'tagged' with radiochromium or radioiron are used for red cell volume measurement. After allowing time for the substance to mix, a sample of blood is withdrawn. A measured volume of this sample is examined in the scintillation counter to give the dilution of the amount originally injected, and from this the whole of the diluting volume (plasma or red cells) can be arrived at. The normal blood volume is 70–85 ml. (average 77 ml.) of whole blood per kilogram (2.2 lb.) body weight, or 35–50 ml. plasma/kg. Thus it varies between a few hundred ml. in a neonate up to 5–6 litres in an adult (more in certain pathological conditions, e.g. high output cardiac failure from chronic anæmia or arterio-venous fistula). The Volemetron apparatus (fig. 66) is commonly used for this estimation.

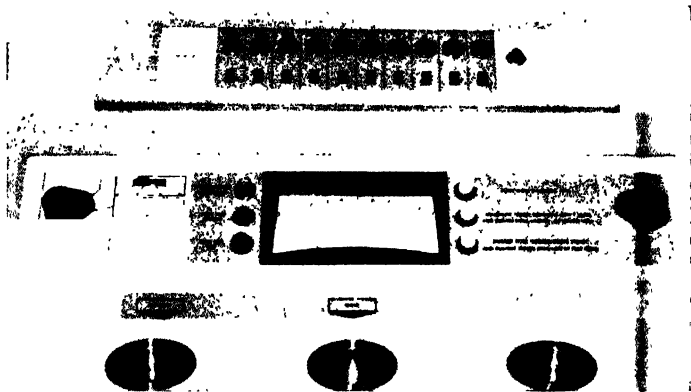


FIG. 66.—Volemetron. The centre dial reads the blood volume in litres; ranges 0–10 L. for adults and 0–5 L. for children. The black knob on the left side is for computing the adult or children's blood volume. The black knob on the right is for selecting the stages used for the procedure. In the foreground are three wells where the radioactive dose- and blood-samples are placed for measurement. At the back is seen the memory display panel used with 125' or 51''.

(e) *Measurement of Central Venous Pressure (C.V.P.)* (fig. 67)

An immediate definitive diagnosis of a collapsed patient is often difficult. Hæmorrhage, pulmonary embolism, cardiac infarction, mesenteric thrombosis, acute pancreatitis, also pleural, peritoneal, intestinal, and bacteriæmic shock may present similarly. Delay in giving blood or other intravenous fluids (too little as well as too late) to those in need of it, denies them the chance of survival, while on the other hand, a patient's condition may be worsened by infusion if the cardiac function is impaired. Measurement of the C.V.P. is of practical value because it indicates the state of the venous reservoir, the venous return, and cardiac efficiency, and therefore whether a transfusion should be given.

Method.—It can be measured (a) internally by an intravenous catheter (p. 90) connected to a simple saline manometer, the range being 12 cm. H₂O (high) to 3–6 cm. H₂O (low) (Freeman), or (b) externally by a gauge (McGowan and Walters) which measures the difference in level between the manubrium and the blood column in the external jugular vein. Therefore if the C.V.P. is low the venous return should be supplemented by intravenous infusion, but not if the pressure is high. By careful monitoring a rapid transfusion can be given in a low pressure state. Any sustained rise of pressure to the maxima demands immediate cessation of the infusion.

THE TREATMENT OF HÆMORRHAGE

This can be summarised :—

- I. Stop the blood loss by—Pressure and Packing. Position and Rest. Operation. Ligation, Repair, Excision.

James Freeman, Contemporary. Senior Anaesthetic Registrar, General Infirmary, Leeds.
Graham Kemp McGowan, Contemporary. Director of the Department of Clinical Pathology, United Bristol Hospitals.
Glyndwr Walters, Contemporary. Consulting Pathologist, Wolverhampton Hospital.

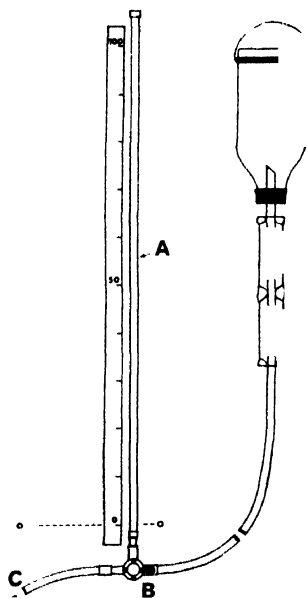


FIG. 67.—Saline manometer for measuring central venous pressure. A = The sterile glass or plastic tube manometer against a centimetre scale; a spirit level is used level the zero to the manubrium sterni. B = The three-way stop-cock allows (i) saline to run via C into the vein from the reservoir, (ii) to fill the manometer, and (iii) to exclude the reservoir and to allow the fluid in A to fall to the level of the C.V.P.

2. Restore blood volume by—Blood Transfusion (p. 72): Saline; Dextran (p. 76); Plasma (p. 77).

Methods of Stopping Hæmorrhage

Pressure and Packing.—*The first-aid* treatment of hæmorrhage from a wound is a pressure dressing, this being made from anything handy which is soft and clean (linen), but even a rolled up forage cap stuffed into a wound has been effective in battle (Drummond). The dressing or pack should be bound on tightly.

Other examples of pressure being used to control hæmorrhage include: the use of forefinger and thumb or a clothes-peg for epistaxis (the majority of nose-bleeds come from the anterior nares); the use of a double balloon in the œsophagus and the stomach (Sengstaken tube, Chapter 34) to control the bleeding from œsophageal varices.

Packing by means of 6-yard (6.5 m.) rolls of wide gauze is an important standby in operative surgery. If several rolls are used the ends must be tied together to ensure complete removal later. (A young schoolboy fell over the handlebars of his bicycle. His liver was split in half, down to the hilum. Many rolls of gauze packing stopped the hæmorrhage. They were removed a week later and the boy returned to school and completed advanced level education.)

N.B.—If on removal of pressure or packing, bleeding appears to have ceased completely, do not assume that all is well, especially when dealing with deep wounds involving large veins. Continued close observation is required.

Tourniquets are to be avoided in *first aid*. They can only be used around the thigh or upper arm; they are difficult to apply tightly enough to interrupt arterial blood flow, and only cause venous congestion and increased blood loss. There have been many examples of tourniquets of some kind being used for ruptured varicose veins causing exsanguination by only occluding venous return. (For example, a policeman was brought into a casualty department with seven tourniquets in place. The venous bleeding ceased as soon as they were removed!) Tourniquets tight enough to obstruct arterial flow may cause contusion of the artery wall and thrombosis, especially if atheroma is present. Nerve conduction can also be seriously impaired, while to leave a tourniquet on for over an hour is to invite muscle death and the risk of renal failure (crush syndrome, p. 80). To sum up, it can be said that tourniquets are dangerous because they may be applied too loosely, too tightly, or for too long.

In the operating theatre tourniquets are used (a) to control hæmorrhage temporarily while exploration and repair are being carried out; (b) for some amputations (not for atherosclerotic gangrene); (c) in order to obtain a bloodless field for orthopædic and soft tissue operations (cartilage, tendon, nerves, ganglia of tendon sheaths). The sphygmomanometer cuff is very satisfactory as a pneumatic tourniquet; the cuff is

inflated after elevation of the limb for five minutes has materially reduced the vascularity. Alternatively, a bloodless operation field can be obtained by an Esmarch bandage applied spirally to the elevated limb commencing distally, and then bound on tightly. The distal spirals are unwound to expose the limb for operation.

Removal of a Tourniquet.—The fact that a tourniquet is in place and the time of application must be written large on the theatre blackboard and deleted only when it is removed. It is a wise precaution to attach the tourniquet to the operating table, so that it cannot be overlooked with the patient is lifted on to the trolley.

In the ward as well as the theatre, Esmarch bandages may be applied temporarily to the limbs in order to reduce the vascular compartment in shock, so aiding the heart to maintain blood flow to vital structures.

Venous Tourniquet.—A light rubber or a pneumatic tourniquet is applied to the upper arm prior to intravenous injections and sampling. It is also used in the diagnosis of varicose veins (p. 146).

Position and Rest.—*Elevation of limbs* (e.g. in ruptured varicose veins) employs gravity to reduce bleeding. Elevation also causes vasoconstriction (Lister). *A bed elevator* is often used to raise the foot of the bed as an aid to vasoconstriction in reducing the size of the vascular compartment, so facilitating blood flow to the brain and the restoration of a satisfactory blood pressure. Gravity is also used in certain operations, as in thyroidectomy when the patient is tilted feet downwards (anti-Trendelenburg position) or as in stripping of varicose veins when a head-down tilt is used (Trendelenburg).

Donovan's manoeuvre may be used to remove an extension of a hypernephroma up the inferior vena cava. After removal of the kidney, the patient is tilted steeply head downwards. The vena cava is occluded below the renal veins and then opened, so that forceps or sucker can remove any growth that is present. Venous blood from the other kidney is not troublesome. Air embolus is prevented by careful attention to the positive pressure phase of controlled respiration by the anaesthetist.

Absolute rest is vital. This means nursing the patient in a comfortable recumbent position, and he should be relieved of unnecessary exertion. He does not wash or feed himself, and assistance is given for turning in bed, which must be at least two-hourly to prevent pressure sores.

Sedation also brings rest. Morphine (10–20 mg.) relieves pain, calms restlessness, and aids coronary and cerebral blood flow. Though it is contra-indicated where there is respiratory depression (as in head injuries), its use is invaluable where respiratory movements and coughing are inhibited because of chest pain. Morphine is best given intramuscularly or intravenously in hæmorrhage and shock. A subcutaneous dose is not easily absorbed because of vasoconstriction, and if repeated doses are given in this way an excessive amount is liable to be absorbed when the circulation improves. Morphine does not in itself produce sleep, and the patient, though relieved of pain, may remain wakeful and anxious. Amylobarbitone (120 mg.) should be used if there is no respiratory depression, and drugs like chloral hydrate (1–2 G.) and paraldehyde (5–10 ml., i.m.) are excellent.

Examples of Operative Techniques in Hæmorrhage.—Artery forceps (e.g. Spencer Wells) or 'hæmostats' are mechanical means of controlling bleeding by pressure. The clamped vessel can be ligated with catgut, cotton, thread or silk, or coagulated with diathermy. Small subcutaneous vessels are sometimes twisted off. When an incision is made through the scalp for craniotomy, the profuse bleeding is not easily arrested by direct forcipressure, so the cranial aponeurosis is picked up by a series of

*Johann Freidrich August von Esmarch, 1823–1908. Professor of Surgery, Kiel, Germany.
Lord Lister, 1827–1912. Professor of Surgery, Glasgow and Edinburgh Universities and King's College Hospital London.*

*Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Leipzig.
Hugh Donovan, 1897–1959. Surgeon. Queen Elizabeth Hospital, Birmingham
Thomas Spencer Wells, 1818–1897. Surgeon. Samaritan Hospital, London.*

forceps which are everted together, thus exerting pressure. Silver clips (Cushing) may be applied to cerebral vessels.

Suturing may be employed. The vessel can be underrun or transfixd by needle and suture and then ligated, while if the continuity of a main vessel is to be restored, 4/0 silk or polypropylene is used (p. 121) on a 20-mm. atraumatic needle.

Pressure by packing, using rolls of wide gauze has been previously mentioned, but light pressure with a 'peanut' of gauze held by forceps aids the sealing of an arterial suture line after reconstruction following trauma, embolectomy, or in artery grafting. About five minutes is required for the platelets to seal the join.

Patches of vein or Dacron mesh may be used to repair a vascular defect. A patch of muscle, lightly hammered, provides thrombokinase to stop a troublesome ooze.

Other topical applications for oozing include gauze or sponge which is absorbed by the body. 'Oxycel' or gelatin sponge provides a network upon which fibrin and platelets can be deposited. This is the modern counterpart of the use of cobwebs by our forefathers, or sphagnum moss by our neolithic ancestors.

Gauze soaked in adrenalin soln. (1:1,000) or 'Stypven' (Russell viper venom) are other topical applications. Bone wax (Horsley) is used for oozing bone.

Excision of a bleeding viscus may be necessary, e.g. a ruptured spleen must be excised. However, a ruptured kidney is treated conservatively if possible (Chap. 45).

BLOOD TRANSFUSION

The indications in surgical practice are briefly as follows:

1. Acute hæmorrhage, depending upon the physical signs (p. 66).
2. At operation, in certain major cases and emergencies where blood and fluid loss is considerable e.g. abdomino-thoracic and abdomino-perineal and arterial operations. The very young and the elderly have a special intolerance to blood loss (p. 67).
3. Preoperatively and postoperatively in anæmic patients (below 9 gm./100 ml.). Care is needed in chronically anæmic patients, in whom there is already an increased cardiac output, for a transfusion can overload the heart. In such cases packed red cells are needed instead of whole blood.
4. Burns and dehydration shock, but only as an adjunct to saline, plasma, or dextran therapy.
5. Severe infections, e.g. septicæmia, gas gangrene, B. coli shock from strangulated bowel, especially if the hæmoglobin falls below 9 gm./100 ml. Transfusion raises the patient's resistance, and anæmia is controlled.
6. To increase coagulability, especially as a preoperative measure in obstructive jaundice, in hæmophilia and purpuric diseases.
7. To obtain temporary improvement in certain blood diseases, such as aplastic anæmia, leukæmia or Hodgkin's disease (p. 156). As an adjunct to the use of cytotoxic drugs in the treatment of cancer.
8. Certain cases of erythroblastosis foetalis (Rh factor, p. 73).
9. Carbon monoxide poisoning. Venesection removes the inert carboxyhæmoglobin and transfusion provides a fresh supply of oxyhæmoglobin.

Methods of Transfusion

Collection of Blood.—The donor lies on a table, and a rubber tube is tied round the arm above the elbow, or a sphygmomanometer cuff is applied, the pressure being raised to 70 or 80 mm. Hg. After introducing a few ml. of local anæsthetic, a 15 Standard Wire Gauge (or equivalent) needle, carrying about 25 cm. of tubing, is inserted into a suitable vein, and the desired quan-

Harvey Cushing, 1869–1939. Professor of Surgery, Johns Hopkins Hospital, and Harvard University.

Patrick Russell, 1728–1806. Physician of Aleppo.

Sir Victor Horsley, 1857–1916. Surgeon, University College Hospital, London. Died of heatstroke or paratyphoid fever in Mesopotamia. One of the editors (McN. L.) attended his funeral.

tity of blood is allowed to flow into a 600-ml. bottle containing 120 ml. of anti-coagulant (ACD) solution.¹ Screw-cap bottles fitted with rubber corks are provided by the Blood Transfusion Service. Two glass tubes pass through the cork, one to act as an air vent and the other to admit the tube which withdraws blood from the donor. The risk of contamination is thus minimised, and the blood is stored in the bottle pending use.

Blood Banks and Stored Blood.—The great demand for blood has led to the establishment of blood banks in suitable centres. The first change which occurs in stored blood is destruction of the white cells, probably owing to proteolytic ferments which they contain, and after a week nearly all granulocytes are replaced by amorphous masses. At the same time, leakage of hæmoglobin occurs from the erythrocytes, but although the red cells shrink they are not destroyed, and the vital property of ability to combine with oxygen is not seriously impaired. Stored blood can be transfused with safety during the first month of storage, and transfusions within the second month have not been followed by serious reactions.

Packed Cells.—These are in the concentrated blood which remains after supernatant plasma is removed (fig. 68). The plasma is stored for future use.

Blood Grouping. Rh Typing. Incompatibility. Cross-matching.

Over twenty different antigens have been recognised in human red cells, but for practical purposes these can be divided into two classes:

1. *Antigens to which Antibodies occur Naturally.*—There are only two, named A and B. Their presence or absence determines the four major human blood groups, the A B O grouping, viz:

Group O: the red cells do not contain either A or B.
the serum contains anti-A and anti-B.

Group A: the red cells contain A but not B.
the serum contains anti-B but not anti-A.

Group B: the red cells contain B but not A.
the serum contains anti-A but not anti-B.

Group AB: the red cells contain both A and B.
the serum contains neither anti-A nor anti-B.



FIG. 68.—Stored blood, showing supernatant plasma. The 'pilot' bottle provides blood for cross-matching.

2. *Antigens without Natural Antibodies: Rh (D) typing.*—Antibodies to red cell antigens other than A and B are created only when cells with the antigens are injected into patients without them. Fortunately only the Rh (D) antigen (the Rhesus factor), present in 85 per cent. of humans, readily gives rise to antibodies. When the red cells contain Rh the patient is Rh-positive; when they do not, the patient is Rh-negative.

Incompatibility.—For practical purposes, such as blood transfusion, it is necessary to know which of the antigens A, B and Rh are present in the red cells, and to make sure, by cross-matching, that the donor's and the recipient's bloods are compatible. Incompatibility causes first agglutination and then

¹ Acid Citrate Dextrose (ACD) solution, containing 3 G. anhydrous dextrose, 2 G. disodium citrate monohydrous and water to 120 ml. The solution is put into the bottle before sterilisation and may become discoloured (harmlessly) by the formation of caramel.

hæmolysis of the donor cells. Severe hæmolysis causes acute renal tubular necrosis (Crush syndrome, p. 80). and the patient is in danger of dying of renal failure. Thus every blood transfusion must be preceded by:

1. Blood Grouping.—(A B O and Rh type) of patient and donor to ensure that the patient's serum does not contain any antibodies to the donor's red cells.

2. Cross-matching (donor's cells and patient's serum) to confirm the above and to exclude any acquired antibodies to the donor's red cells.

Methods.—A B O grouping and Rh typing requires full laboratory facilities using special tube techniques. The cross-matching technique takes two hours, and it is advisable to ensure by a tube test that the patient's serum does not agglutinate the donor's red cells suspended in albumin as well as saline. The Coombs' test (see below) is also included.

In dire emergency, if saline, plasma, or dextran are not sufficient and blood must be given without delay, the surgeon, accepting this responsibility, will either act on a provisional report given in 30 minutes, or will use Group O (Rh negative) blood, which can in theory be given to anyone because the cells do not contain an agglutinin.

Also in emergencies and where there are no laboratory facilities, grouping and cross-matching may revert to the old slide methods:

Slide Method of Grouping and Cross-matching.—Stock sera from Group A and Group B are placed side by side on a slide. Blood obtained by a prick with a sterile cutting needle is diluted 1:20 in saline in order to avoid rouleaux; a drop of this suspension is added to each, and after five minutes is examined microscopically. If clumping of corpuscles, looking like particles of red cayenne pepper, is seen in the serum of Group A, then the corpuscles are those of an individual of Group B. If clumping occurs only in the serum of Group B, then the corpuscles are from Group A. If clumping is present in both, then the corpuscles are from Group AB, and if in neither, they belong to Group O.

Cross-matching.—Mix on a slide one drop of patient's serum and one drop of a 1:20 saline suspension of donor's red cells and rock for five minutes. Clumping = incompatibility.

Rh Incompatibility.—Antibodies to Rh are produced when Rh-positive red cells are transfused into an Rh-negative patient. An analogous condition may happen when an Rh-negative woman carries an Rh-positive foetus; its red cells may cross the placenta into her circulation so that she forms anti-Rh. These, like all maternal antibodies, pass into the foetal circulation, the foetal red cells are destroyed by them, the baby is born with a severe hæmolytic anæmia (icterus gravis) or is even killed *in utero* (hydrops foetalis).

Rh antibodies and other antibodies to red cells artificially acquired, often fail to agglutinate red cells suspended in saline, but will agglutinate red cells suspended in albumin solution. For this reason albumin as well as saline red cell suspensions are used for routine cross-matching. The suspending medium influences the physical consequences of any antigen-antibody union. An anti-bacterial serum, for example, agglutinates bacteria if they are suspended in saline, but not in distilled water.

Coombs' Test.—The antibodies have combined with the red cells even though they have produced no visible effect, and the coating of antibody can be detected by Coombs' test. The cells are treated with a serum made by immunising animals to human globulin, so that it contains antibodies that combine with human globulin molecules to make a precipitate. The antibody coating red cells is, like all antibodies, a globulin; thus, when the serum binds the globulin molecules together in precipitates, it draws the red cells together into large clumps.

Once antibodies have appeared, every successive stimulus (e.g. pregnancy) increases their amount. Thus one transfusion of Rh-positive blood into an Rh-negative girl

may make it impossible for her ever to produce a live child to an Rh-positive husband. *No female under fifty should ever be transfused with Rh-incompatible blood.*

Apart from this danger to the foetus, any patient, male or female, who has acquired anti-Rh may have a severe, even fatal, reaction if transfused with Rh-positive blood. The anti-Rh will have been produced by a previous transfusion or by an Rh-incompatible pregnancy. To avoid such accidents a second rule must be observed: *No patient should be transfused with Rh-incompatible blood unless it is certain he will never need another transfusion.*

Giving Blood.—Routine intravenous drip transfusion is commenced by: (a) *selecting and preparing the site* (p. 90). (b) *Checking the donor blood:* i.e., the name and number of the patient *must* be that which has been affixed to the bottle after grouping and satisfactory cross-matching have been carried out; (c) *insertion of the giving needle or cannula and setting up the drip* (p. 90); (d) *giving running instructions.* These should be written out clearly on a card attached to the drip stand or bed board. The average flow rate is 40 drips per minute (one bottle of 540 ml. lasting four hours).

In serious emergencies, e.g. operation for bleeding duodenal ulcer, or leaking aneurysm, over 1 litre (2 bottles) can be given in a quarter of an hour. Two or three drips may be set up mainly using arm, or cephalic veins. The rate of transfusion is increased by using a rotary 'Martin' type of pump (fig. 69).

Warming Blood.—If large amounts of blood from the blood-bank refrigerator are being used (e.g. in heart-lung machines (Chap. 30) or for ruptured aneurysm cases), the blood should be warmed to body temperature by running it through several extension coils of the plastic transfusion tubing which are immersed in a heated water-bath. This reduces the risk of ice-cold blood causing excessive hypothermia and favouring cardiac arrest.

Autotransfusion.—This is the obvious procedure when the patient's own blood can be collected, e.g. ruptured ectopic pregnancy. The blood is strained through six layers of gauze into a sterile flask containing 3.8 per cent. sodium-citrate solution, and then returned to the patient.

Exchange Transfusion.—Replacement or exchange transfusion of an infant suffering from hæmolytic disease of the newborn may be life-saving. Rh-negative blood is used, and 5 to 10 ml. of blood are exchanged at a time. The technique takes several hours, and one person should be responsible for nothing else but checking the volumes exchanged. A syringe is used with a four-way adapter (one to baby, one to donor blood, one to citrated saline for rinsing out between each manoeuvre, one to 'waste'). The transfusion is given by polythene catheter via the umbilical vein passed into the vena cava.

Complications of Blood Transfusion.—1. *Incompatibility.*—These dangerous accidents should be prevented by a proper cross-matching technique, but in fact they are most often due to such human errors as wrongly labelled blood, or rightly labelled blood given to the wrong patient. If a patient on transfusion has a rigor, or complains of pain in the back, the transfusion must be stopped immediately and a check of the donor blood made with the laboratory.

2. *Right ventricular failure* is due to over-loading the circulation, especially when the cardiac muscle is enfeebled, as by chronic anæmia. Such a patient, especially if over the age of sixty, should not be given more than 300 ml. at one time. If anæmia is severe, packed cells should be used (p. 73).

3. *Simple pyrexial reactions* may be due to dirty apparatus (avoided by using dis-

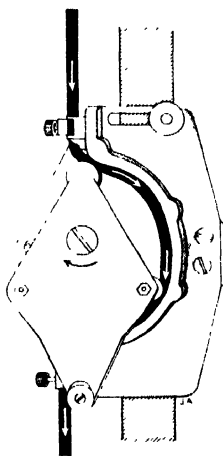


FIG. 69. Martin's pump.

posable plastic apparatus), to pyrogens in the anticoagulant solution, to infected blood, or to a rapid rate of transfusion. Severe pyrexial reactions are accompanied by rigors.

4. *Allergic reactions*, usually urticaria, follow 1 per cent. of transfusions, and are likely if a patient is given repeated transfusions from the same donor.

5. *Hepatitis*.—Probably about 0·8 per cent. of patients receiving blood develop homologous serum hepatitis two or three months later.

6. *Antibody Production*.—Patients who receive repeated blood transfusions sometimes develop antibodies to many red cell antigens, and it may eventually be very difficult to find blood to which they are compatible.

7. *Hypothermia* (see above).

8. *Air embolism* (p. 132).

9. *Thrombophlebitis* (p. 142).

10. *Fibrinopænia* (see below), after massive transfusion.

HÆMOPHILIA AND HÆMOPHILOID DISEASES

Classical hæmophilia (hæmophilia A) is a hæmorrhagic diathesis caused by deficiency in the blood of a specific clotting factor known as the antihæmophilic globulin (AHG) (factor VIII), which is concerned with the generation of thromboplastin, which activates prothrombin to thrombin. Females transmit the condition, but escape the disease, all sufferers being males. The incidence of hæmophilia is highest amongst Anglo-Saxon and Teutonic races; it occurs in Jews, but Latin races are apparently exempt. The well-known affection of the Spanish royal family was transmitted from the Hapsburgs of German origin.

The treatment of this condition is difficult, as the only effective measure is to supply the absent AHG by giving (a) *fresh* blood or *fresh frozen* plasma; (b) human AHG, prepared by special fractionation methods. Stored blood is valueless, except to replace red cells.

*Christmas disease*¹ (*Hæmophilia B*) is clinically indistinguishable from hæmophilia, although usually less severe, and is due to hereditary absence of plasma-thromboplastin component (PTC). Christmas disease will not respond to AHG and can be effectively treated with stored blood. Hæmophilia A and B account for about 90 per cent. of constitutional bleeders.

Rosenthal's syndrome occurs in both sexes, and is due to hereditary deficiency of plasma thromboplastin antecedent (PTA). It is less severe than AHG or PTC deficiencies, but hæmorrhage is often excessive after childbirth or operations.

Clotting and bleeding times are unreliable for control of these hæmorrhagic diseases, but accurate information is gained by assessing the partial prothrombin time (PTT) in conjunction with the Quick prothrombin time.

Fibrinopænia sometimes follows childbirth or thoracic operations with massive hæmorrhage, without previous history of bleeding episodes. Fibrinopænia should be treated with fibrinogen, either as a purified product or as triple-strength plasma.

Hæmophilic Joints.—In hæmophilia, recurrent spontaneous hæmarthrosis is a very common incident. Strangely enough, the hæmarthrosis seems to affect certain particular joints and will often recur repeatedly in one joint, leaving the other joints of the body free. The knee is the commonest site for hæmophilia and frequently recurrent hæmarthroses will occur unilaterally. The joint becomes tense and swollen and sometimes the skin is discoloured over it. Repeated effusions of blood into such a joint result in disorganisation, and X-ray changes may be very puzzling unless the history of hæmophilia is known. The end-result is frequently a fibrous ankylosis.

BLOOD SUBSTITUTES. DEXTRAN. PLASMA

Dextran is a complex polysaccharide of dextrose produced by the action of non-pathogenic bacteria, particularly *Leuconostoc mesenteroides*, on a substrate of sucrose and phosphate. Blood should be taken for grouping before any dextran is administered, as it interferes with accurate grouping.

¹ So-called after the name of the first patient in whom the disease was discovered.

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Armand James Quick, *Contemporary. Professor Emeritus of Biochemistry, Marquette University School of Medicine, Milwaukee, Wisconsin, U.S.A.*

The organisms break sucrose into fructose and dextrose, and synthesise dextran molecules of various sizes. Introduced intravenously, the molecules, if too large, cause untoward reactions, and if too small they are rapidly excreted by the kidneys. The undesirable molecules are eliminated in the manufacture of dextran. Dextran shows little tendency to diffuse through the capillary walls, and at least 50 per cent. of the solution should be retained in the body at the end of twenty-four hours. After that time some of it is excreted, and the remainder is katabolised slowly over about a week. These desiderata are reached by the commercial products listed below.¹

Between 1 and 2 per cent. of patients have a pyrexia and/or urticaria during or following the infusion of dextran. Possibly these patients have been sensitised by *Leuconostoc mesenteroides*; more probably by related pneumococci. Dextran has also a slight nephrotoxic effect, but the condition is usually reversible.

Dextran is administered intravenously and it is safer than plasma, or Group O blood when the time factor prevents cross-matching. One or two bottles can be given comparatively quickly. Infrequently a third bottle may be required, but this amount must not be exceeded, or the patient's blood will become so diluted that its oxygen-carrying properties will become seriously impaired. If more fluid therapy is required, only correctly matched whole blood should be given.

Plasma is valuable as it can be used without delay and cross-matching is unnecessary. Its danger is the 1·3 per cent.² risk of causing virus hepatitis. Dried plasma can be kept almost indefinitely at room temperature, and reconstructed by adding sterile pyrogen-free distilled water, and shaking. Excellent results may be obtained in severely shocked patients by giving as much as 2, or occasionally 3, bottles (1·7 L.) of plasma intravenously in half an hour, but, except in previously healthy individuals, the limit should be two bottles (1·14 L.).

Mannitol, a sugar of low molecular weight, while not a substitute for blood, is of value in preventing the acute renal failure which follows renal arteriolar constriction in all types of shock, and in the crush syndrome (p. 80). It reduces the mortality from renal failure after operations for obstructive jaundice (Dawson). It promotes an osmotic diuresis and may have a specific renal vasodilator action. Commencing preoperatively, up to 1·5 litres of a 10 per cent. solution is given intravenously for twenty-four hours. Test doses of mannitol may be used in cases of diminished urine output to determine whether hydration therapy or the strictly-limited-fluid regimen (Chap. 44) is to be adopted. Catheterisation (indwelling) is helpful to prevent retention due to rapid overfilling of the bladder and to provide means of accurate recording of the response to therapy.

SHOCK

Shock is a combination of a threat to existence and the response of the body to this threat. The types of shock are listed below.

The common factors in shock are: 1. *A loss or withdrawal of blood volume* from the main vascular channels of the body, affecting cardiac output, central blood pressure and the perfusion pressure of oxygen into the tissues, particularly the heart and brain. 2. *Vasoconstriction* (Nature's first aid for shock), which serves to maintain the peripheral vascular

¹ Plasma substitutes: 'Dextraven', Benger Laboratories Ltd., 'Intradex', Crookes Laboratories Ltd.; 'Plasmosan', May and Baker Ltd.) A satisfactory low molecular weight dextran ('Rheomacrodex', Pharmacia (G.B.) Ltd.) is available which prevents red cell sludging in cases of shock, and is useful in the crush syndrome.

² If the plasma is derived from a pool of more than 10 donors, the risk rises proportionately.

resistance, the arterial pressure, and therefore the oxygen perfusion of the vital structures. This is a protective mechanism which plays for time—time for the threat to cease (e.g. arrest of hæmorrhage and blood transfusion), so that full recovery is possible (reversible shock). If the threat persists, is repeated, or replacement of blood volume is not carried out, the anoxia to heart muscle and brain stem causes failure of these organs, thus impairing oxygen perfusion of tissues still further and causing a vicious circle (irreversible shock) ending in death (p. 66). Contributory causes are exposure, fatigue and starvation, and the extremes of life.

Clinically the condition of (1) loss of blood volume and (2) vasoconstriction is manifest as pallor, empty veins, cold skin, sweating and increased pulse. Restlessness and air hunger indicate hypoxia of the brain stem.

The Types of Shock

Vasovagal Shock. Neurogenic Shock. Psychogenic Shock.—Occurs immediately as a result of a sudden fright (e.g. bad news) or severe pain (e.g. a blow on the testes). The effect varies in intensity from a slight faint to sudden death. The expression 'I nearly died of fright' is not necessarily hyperbolic. The hypoxia of the vital centres is brought about by pooling of blood in the large vascular reservoirs (limb muscles), and by dilatation of the splanchnic arteriolar bed, with consequential low cardiac output and fall of arterial pressure until restored by peripheral vasoconstriction. A similar phenomenon of pooling and fainting occurs in guardsmen standing to attention for long periods.

Hæmorrhagic Shock.—See hæmorrhage, p. 65 and p. 77.

Burns Shock (p. 110).—The loss of blood volume is due to rapid plasma loss from the damaged tissues and whole blood destruction.

Dehydration, Diarrhæa, and Vomiting.—Severe extravascular fluid loss is usually associated with infection (exotoxins and endotoxins) and loss of protein as a result of inflammation of the intestinal mucous membrane (e.g. staphylococcal enterocolitis (p. 5), cholera, etc.).

Pleural, Peritoneal, Mediastinal, Retroperitoneal Shock.—Sudden leakage of blood, or alimentary secretions into these compartments will cause sudden collapse. Examples are penetrating chest wounds and hæmothorax, a leaking œsophageal or intestinal anastomosis, acute pancreatitis, biliary peritonitis.

Bacillus Coli Shock (Intestinal) (syn. Gram Negative Shock).—Multiplication of bacillus coli occurs in segments of strangulated intestine (strangulated hernia, mesenteric thrombosis), and in peritonitis. The endotoxin from the bacilli causes splanchnic arteriolar dilatation and blood volume discrepancy. Release or removal of the strangulated segment, or the drainage of pus is the important practical factor in obtaining recovery of the patient.

Bacteræmic Shock from other Infections.—Gas gangrene is a typical example, causing decreased blood volume and also anæmia from hæmolysis.

Anaphylactic shock, histamine shock, may follow injection of serum (e.g. A.T.S.), in spite of precautions (p. 11), into patients who have had serum before, or who have a history of allergy. There is wide dilatation of the splanchnic vessels with consequential fall of venous return and cardiac output, necessitating immediate injection of

adrenalin (0.5–1.0 ml., 1:1,000 soln.), which may need to be repeated in doses of 0.5 ml. every 20–30 minutes if the systolic pressure remains below 100 mm. Hg.

Diagnostic Difficulties in Shock.—Difficulties occur particularly with intra-abdominal catastrophes, such as peritonitis and bacillus coli shock. Because of a thready and irregular pulse, cardiac infarction and even pulmonary embolism are notoriously confusing. In both there is a shock-like state from a degree of failure of cardiac output and compensatory vasoconstriction. An E.C.G. and an X-ray of the chest do not always determine the diagnosis. The measurement of the central venous pressure (C.V.P.) (p. 69) and its response to a small rapid transfusion may be of assistance. However, the surgeon cannot escape the responsibility of performing an exploratory operation for suspected peritonitis, and after adequate operative treatment recovery starts immediately, and intravenous fluids, hitherto ineffective, have their desired effect.

Treatment of Shock

Treatment, in principle, is the removal of the cause and the replacement of lost blood-volume. In hæmorrhage this implies the arrest of hæmorrhage and blood transfusion. In burns the loss of plasma is replaced by plasma and dextran (p. 111). Bacillus coli and bacteræmic shock require removal of the causal lesion and transfusion with blood or substitutes, as indicated in the preceding paragraphs.

Position and Limb Compression.—Venous return is improved, and pooling of blood in the large muscles of the legs is reduced by raising the foot of the bed and bandaging the limb so as to compress the peripheral blood vessels.

Overheating is harmful—for the vasodilation so caused will nullify the compensatory reflex vasoconstriction.

Relief of pain is achieved by giving morphine 10–15 mg. ($\frac{1}{4}$ – $\frac{1}{2}$ grain) I.M. or I.V. It is not given subcutaneously as it is not easily absorbed, and if repeated doses are administered, an excessive amount is liable to be absorbed when the circulation improves. It is not to be given in head injuries.

Sedation for a frightened patient may be by intravenous phenobarbitone, 60–100 mg. (2 grains) or paraldehyde 5–10 ml. I.M.

Vasoconstrictor Drugs.—These should be given early. Given late, their action, if any, will only serve to accelerate the damage caused to kidneys and liver by intense prolonged vasoconstriction. The substances used include intravenous methylamphetamine (Methedrine) 15 mg. and noradrenaline 4 mg. per litre of intravenous fluid—the rate of flow being adjusted according to the response obtained. Hydrocortisone hemisuccinate in doses of 100 mg. may be added to the intravenous fluid. It raises the blood pressure possibly by sensitising the peripheral vascular nerve endings to naturally circulating noradrenaline. Care is always taken to wean the patient from vasoconstrictor drugs as soon as possible, or he may become drug-dependent.

OXYGEN THERAPY

Indications in Surgery.—In shock its value has been disputed, but it is of special value in chest injuries, in severe hæmorrhage, in toxic hæmolysis, e.g. gas gangrene, in coal gas poisoning and in over-morphinisation. It is also to be used in pulmonary embolism, fat embolism, spontaneous pneumothorax, acute pulmonary œdema, and cardiac infarction, and in cases of acute

bronchitis, pneumonia, and anoxic cor pulmonale. The usual method employs a disposable polythene mask, the Polymask or the Pneumask (fig. 70), delivering 4–6 litres per minute. For patients with chronic bronchitis and emphysema there is a risk of a high concentration of oxygen reducing the carbon dioxide drive for respiration, so causing carbon dioxide narcosis.



FIG. 70.—Pneumask.



FIG. 71.—A disposable 'Ventimask'.

(Courtesy Oxygenaire Ltd.)

The safe concentration (Campbell) is about 27 per cent. oxygen delivered by a Venturi or disposable Ventimask (fig. 71) in which a jet of oxygen at 4 litres per minute strikes the side walls of the air intake aperture and sucks in about 50 litres of air per minute thus giving the necessary mixture.

Oxygen tents may also be used if a high concentration of oxygen is required. Children should be reassured, as they are likely to be frightened by confinement in an enclosed space, and, if possible, nervous patients should be accustomed to a tent before the necessity arises. Nurses are warned against the risk of explosion, and naked lights and smoking are forbidden in the proximity of the tent.

Hyperbaric Oxygen. Oxygen Drenching.—Oxygen breathed through a mask at 2 or 3 atmospheres pressure in a compression chamber will at least double the arterial plasma oxygen saturation and so the oxygen perfusion of tissues can be increased. When such a chamber is available it can supplant the use of carbogen (oxygen with 5 or 7 per cent. carbon dioxide) in the treatment of carbon monoxide poisoning, and may be used for the treatment of anaerobic infections, e.g. tetanus and gas gangrene, and ischaemic ulcers and pre-gangrene in peripheral vascular disease. Oxygen is also a radiosensitiser in the treatment of cancer. Drenching has been used to treat paralytic ileus and meteorism, for since the gas in the distended bowel is nitrogen, breathing pure oxygen so reduces the partial pressure of nitrogen in the lungs and plasma that reabsorption from the bowel takes place.

Topical Oxygen Therapy.—Hydrogen peroxide 5 or 10 volumes, and 2 per cent. zinc peroxide, giving off nascent oxygen are used locally on wounds infected with anaerobic or microaerophilic organisms, e.g. spreading symbiotic gangrene of the abdominal wall (Chap. 42). Hydrogen peroxide is also used to free the sloughs of ulcers.

CRUSH SYNDROME

This syndrome which was commonly associated with air-raids, sometimes occurs in connection with mining and other accidents. As a result of massive crushing of

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muscles, oligæmic shock occurs, due to extravasation of blood mainly into adjacent muscles, and myohæmoglobin enters the circulation. Acute renal tubular necrosis is likely to result. The degree of shock associated with the injury has no relation to the development of the syndrome. A similar effect may follow the application of a tourniquet for too long a period, or one that has been forgotten (p. 70)!

First-aid treatment may necessitate the application of a tourniquet to the affected limb, which is gradually released so that deleterious substances are admitted to the circulation in small quantities. The patient may appear to be comparatively well for two or three days following the accident, but excretion of urine is scanty. Apathy, restlessness, and possibly mild delirium indicate deficient renal function, and uræmia supervenes. The crushed muscle often swells considerably, and being confined within unyielding deep fascia, tension develops, impeding the circulation and increasing the extent of ischæmic damage. The limb feels tense and pain is severe. Tension should be relieved by parallel incisions, through which the swollen muscle protrudes. If oliguria develops, the fluid intake must be restricted to 'x' plus 400 ml., 'x' being the urinary output for the previous twenty-four hours. The urine should be rendered alkaline by the administration of sodium citrate and sodium bicarbonate. *Rheomacrodex* (p. 77), by clearing blood sludging and myohæmoglobin, or *mannitol* (p. 77), help to re-establish renal cortical blood flow. If there is no response to these measures, the use of an artificial kidney may be life-saving.

CHAPTER 6

FLUID, ELECTROLYTE AND ACID-BASE BALANCE

DAILY WATER BALANCE OF A HEALTHY ADULT (70 kgm)

Intake		Output	
Water from beverage	. 1,200 ml.	1,500 ml.	. Urine
Water from 'solid food'	. 1,000 ml.	900 ml.	. Insensible loss from skin and lungs
Water from oxidation	. 300 ml.	100 ml.	. Faeces

It will be noted that:

The Intake is derived from two sources (a) exogenous, and (b) endogenous.

Exogenous water is either drunk or ingested in solid food. The quantities vary within wide limits,¹ but average 2 to 3 litres per twenty-four hours, of which nearly half is contained in solid food.

Taking into consideration their body-weight, the water requirements of infants and children are relatively greater than those of adults because of (a) the larger surface area per unit of body-weight; (b) the greater metabolic activity due to growth; (c) the comparatively poor concentrating ability of the immature kidneys of the neonate.

Endogenous water is released during the oxidation of ingested food and the amount is less than 500 ml. per twenty-four hours. During starvation this amount is supplemented by water released from the breakdown of body tissues.

The Output.—Water is lost from the body by four routes :

1. *By the Lungs.*—About 400 ml. of water is lost in expired air each twenty-four hours. In a dry atmosphere, and when the respiratory rate is increased, the loss is correspondingly greater.

2. *By the Skin.*—When the body becomes overheated there is visible perspiration, but throughout life invisible perspiration is always proceeding. The cutaneous fluid loss varies within wide limits in accordance with the atmospheric temperature and humidity, muscular activity, and body temperature. In a temperate climate the average loss is between 600 and 1,000 ml. per twenty-four hours.

3. *Faeces.*—Between 60 and 150 ml. of water are lost by this route daily. In diarrhoea this amount is multiplied not only by the number of stools, but according to their fluidity and size.

4. *Urine.*—The output of urine is under the control of a secretion of the

¹ The wide individual variation of patients' habitual water intake deserves greater consideration than it receives at present. It may be as great a hardship, and actually harmful, for an elderly patient to drink 5 to 6 pints (3·4 litres) as for others to do without water entirely (A. W. Wilkinson).

posterior lobe of the pituitary gland (the anti-diuretic hormone) which controls the tonicity of the body fluids, a function that it performs by stimulating the reabsorption of water from the renal tubules, thus varying the amount excreted after the requirements of the first three routes have been met. The normal urinary output is 1,500 ml. per twenty-four hours, and provided the kidneys are healthy, the specific gravity of the urine bears a direct relationship to the volume.

Water Depletion.—*Pure water depletion*, apart from the concomitant electrolyte depletion which follows diarrhoea or vomiting, is usually due to a diminished intake. The causes include: (a) weakness, exhaustion, mental changes, and cerebrovascular states, (b) because intake is restricted after gastro-intestinal operations, or (c) inability to swallow as in painful conditions of the mouth or throat or in oesophageal obstruction. The main symptom is intense thirst and the leading sign is oliguria. As the dehydration progresses, these features (and weakness) increase, but the patient still remains capable of physical and mental exertion. Pure water depletion may also follow the increased loss from the lungs after tracheostomy and may amount to as much as 500 ml. in twenty-four hours in excess of the normal insensible loss. After tracheostomy humidification of the inspired air is an important preventative measure.

Treatment.—The intake of water must be increased. If swallowing is possible, much depends upon the nursing staff ensuring a regular half or hourly intake. Intravenous 5 per cent. dextrose or dextrose saline (p. 89) is given if swallowing is impossible (water is not isotonic and would hæmolyse blood). Careful charting of intake and output is essential. A diuresis signals that enough has been given, and thereafter care must be taken not to overload and cause water intoxication.

Thirst.—The seat of the osmoreceptors (drinking centre) is in the supra-optic nucleus of the anterior hypothalamus (E. B. Verney). Thirst is most intense about six to twelve hours after operation and it lasts forty-eight hours or more. This period corresponds to the rapid loss of potassium from the cells of the body. If necessary, the urge to satisfy thirst can be assuaged by sham drinking—contact of water with the pharynx—by washing out the mouth with ice-cold water.

Water Intoxication, which can occur without sodium loss, especially if the excessive intake of water is rapid, is more likely to supervene when impaired renal function leads to slowing of diuresis. There is always a danger of this happening when there is (1) failure to keep accurate intake and output fluid-balance charts, (2) failure to add up the twelve- or twenty-four-hourly totals on the charts (fig. 77), as well as (3) failure to be aware that water intoxication can actually occur. It is particularly likely (a) when isotonic (5 per cent.) dextrose solution is being administered intravenously, (b) when fluid therapy is being undertaken by the administration of tap water per rectum and (c) following recto-colonic washouts of plain water or sodium-free solutions, particularly in children with Hirschsprung's disease (Chap. 38).

Clinical Features.—These include drowsiness, apathy, weakness, sometimes periods of excitement and delirium, and later convulsions and coma. The patient is pale and cold and bathed in sweat and the patient appears to be in shock—which is

incorrect, because the blood-pressure is not low and the pulse is not unduly rapid until the patient is moribund. Nausea and vomiting of clear fluid are common, and usually the patient passes a considerable amount of dilute urine. The diagnosis can be confirmed biochemically by finding a reduced plasma-sodium value.

Treatment.—The intake of water having been stopped, the best course is masterly inactivity. If after several days the water-logged patient is still stuporous (due, no doubt, to renal inactivity), it is permissible to inject *very slowly* not more than 200 ml. of hypertonic (5.85 per cent.) saline solution intravenously. If this brings about improvement and a diuresis begins the injection should be stopped forthwith, otherwise circulatory failure from overloading (due to the sudden shift of fluid from cells and tissue spaces) or cardiac arrest is liable to ensue.

ELECTROLYTE BALANCE

When inorganic salts are in solution, as in the extracellular or intracellular fluids of the body, they dissociate into ions. Ions are of two kinds (*a*) *Cations*, which are electropositive and (*b*) *Anions*, which are electronegative: collectively these are the electrolytes. The most accurate way of describing the chemical concentration, reactivity, and osmotic power of these ions is in terms of their milli-equivalents per litre (mEq./l). The cations include sodium, potassium, calcium, and magnesium; the anions chloride, phosphate, bicarbonate, and sulphate. The distribution of the salts within the fluid compartments of the body controls the passage of water through the cell walls and maintains acid-base equilibrium.

Sodium Balance

Sodium is the principal cation content of the extracellular fluid. The total body sodium amounts to approximately 5,000 mEq., of which 44 per cent. is in the extracellular fluid, 9 per cent. in the intracellular fluid, the remaining 47 per cent. being in bone. The sodium housed in bone merits special notice: a little more than half of it is osmotically inactive, and requires acid for its solution; the remainder is water-soluble and exchangeable. Thus there is a large storehouse of sodium ready to compensate abnormal loss from the body. The daily intake of sodium is inconstant. On an average it is 80 to 100 mEq. (5 to 6 G.) sodium chloride or 1 pint (568 ml.) of isotonic 9.9 per cent. saline solution. An equivalent amount is excreted daily, mainly in the urine, also a little in the faeces. The loss in perspiration normally is negligible; however, in persons not acclimatised to tropical heat prolonged profuse sweating results in a considerable loss of sodium—as much as 85 mEq. per hour (J. Nash). If water alone is given to counterbalance the fluid loss, serious sodium depletion can occur from excessive sweating. (See also mucoviscidosis, Chapter 36).

Control by Adrenal Corticoids.—The output of sodium, governed by the variation in the avidity with which the renal tubules reabsorb sodium from the glomerular filtrate and the amount of sodium excreted by the sweat glands, is under the control of the adrenal corticoids, the most powerful conservator of sodium being *aldosterone*. When the adrenal glands have been destroyed by disease or extirpated, there is an unbridled loss of sodium in the urine. However, this inability on the part of the renal tubules to reabsorb sodium from the glomerular filtrate does not become implemented for several days, the reason being:

The Sodium Excretion Shut-down of Trauma.—Following trauma of any kind (including operation trauma) there is a spell, the length of which varies directly with the degree of tissue damage, of almost non-excretion of

sodium. During this period the output of sodium, reduced to not more than 10 mEq. *per diem*, cannot be increased by a supplemented intake of sodium. For this reason it is most inadvisable to administer large quantities of isotonic (0.9 per cent.) saline solution soon after an operation. The period of sodium shut-down lasts for at least forty-eight hours.

Sodium Depletion (*syn.* Hyponatræmia).—The most frequent cause of sodium depletion seen in surgical practice is obstruction of the small intestine, with its rapid loss of gastric, biliary, pancreatic, and intestinal secretions by antiperistalsis and ejection, whether by vomiting or aspiration. Duodenal, total biliary, pancreatic, and high intestinal external fistulæ also are all notorious for bringing about early and profound hyponatræmia.

There is one other less obvious, nay surreptitious, means whereby the patient is robbed of sodium, and that is by gastric aspiration combined with allowing the patient to drink as he pleases and promptly aspirating the fluid swallowed. The act of drinking excites the flow of gastric juice, and this is aspirated also. During this form of therapy, should the patient be receiving intravenous dextrose solution, as opposed to dextrose-saline solution, to maintain fluid balance, he will soon become a victim of hyponatræmia.

Clinical features of hyponatræmia are due mainly to sodium depletion, but in part to depletion of body water.

In established cases the eyes are sunken and the face is drawn. In infants the anterior fontanelle is depressed. The tongue is coated and dry; in advanced cases it is brown in colour. Unlike the dehydration produced by loss of water only, in water + salt depletion thirst is not particularly in evidence. The skin is dry and often wrinkled, making the patient look older than his years. The subcutaneous tissue feels lax. Peripheral veins are contracted and contain dark blood. The arterial blood-pressure is likely to be below normal. The urine is scanty, dark in colour, of a high specific gravity, and except in cases of salt-losing nephritis contains little or no chloride.

Presuming that the erythrocyte count before the dehydration commenced was normal, the hæmatocrit reading (P.C.V.) provides an index of the degree of hæmoconcentration. On the other hand, hæmoconcentration can be masked by pre-existing anæmia (p. 68).

Treatment.—The sodium deficit is replaced by an intravenous infusion of 0.5 to 2 litres of isotonic saline or Ringer's solution, depending on the severity of the hyponatræmia. It is important to watch the plasma protein levels in order to avoid overdosage, thus causing hypoproteinæmia and œdema. In severe cases the first step is to restore the circulating blood volume by a rapid infusion of plasma or plasma substitute.

Post-operative Hyponatræmia.—When hyponatræmia arises as a result of too prolonged administration of a sodium-free solution (5 per cent. dextrose) intravenously, symptoms seldom present until such treatment has been in progress for more than forty-eight hours (*cf.* Water Intoxication, p. 83). Early symptoms are headache and giddiness. Hyponatræmia causes pylorospasm and the resulting vomiting adds to the sodium deficit. Peripheral circulatory impairment (shock) follows.

Laboratory Tests.—If renal function is impaired, there will be a fall in urinary chlorides, and the plasma-sodium level will have fallen.

Treatment.—If shock is present, that must be treated first. Isotonic (0.9 per cent.) saline solution is then administered very cautiously, and discontinued as soon as symptoms abate.

Sodium excess is likely to arise if a patient is given an excessive amount of 0.9 per cent. saline solution intravenously during the early post-operative period when, as has been described, some degree of sodium retention is to be expected. The result is an overloading of the circulation with salt and its accompanying water. Even a sub-clinical degree of sodium excess is harmful, for it results in œdema of intestinal and other suture lines. In addition there is danger of pulmonary œdema.

Clinical Features.—Slight puffiness of the face is the only early sign. The patient himself makes no complaint. Pitting œdema should be sought, especially in the sacral region, but for pitting œdema to be present at least 4.5 litres excess fluid must have accumulated in the tissue spaces (Marriott). The patient's weight increases *pari passu* with the water-logging, and the aid of bed-scales should be sought. Signs of overhydration in infancy (infants are very susceptible) are increased tension in the anterior fontanelle, increased weight, increase in the number of urinations, and œdema.

Treatment.—The infusion must be stopped. In established cases with distended jugular veins and pulmonary œdema, treatment should follow the lines suggested on p. 675.

Potassium Balance

Potassium is almost entirely intracellular. No less than 98 per cent. is intracellular, and only 2 per cent. is present in the extracellular fluid. Three-quarters of the total body potassium (approximately 3,500 mEq.) is found in skeletal muscles. When the body needs endogenous protein as a source of energy, potassium, as well as nitrogen, is mobilised. The mobilised potassium passes to the extracellular fluid, but the surplus over and above the normal content is so rapidly excreted by healthy kidneys that the concentration of potassium in the plasma remains unaltered. Each day a normal adult ingests 2 to 3 G. (52 to 78 mEq.) of potassium in food; fruit, and especially milk, are rich in this cation. Except for a very small quantity in formed fæces and a still smaller quantity in sweat, an amount corresponding to the intake is excreted in the urine.

Potassium Depletion

The Augmented Potassium Excretion of Trauma.—Following trauma, including operation trauma, there is a spell, varying directly with the degree of tissue damage, of increased excretion of potassium by the kidneys. This loss is greatest during the first twenty-four hours and lasts, for example in the case of partial gastrectomy, about three or four days. It will be noticed that the behaviour of the body's potassium in the post-trauma phase is the exact opposite to that of sodium—the potassium flees; the sodium stays. So great are the body's reserves of potassium that, unless the patient was

severely depleted at the time of the operation, the therapeutic administration of potassium (always a responsible undertaking) does not arise until the fourth day of intravenous alimentation.

The Increased Potassium Loss of Alkalosis.—Alkalosis also brings about abnormal loss of potassium in the urine. It is hypothesised that the cause of this potassium loss is increased permeability of cell membranes resulting from the alkalosis. In addition to the evil of letting the potassium out, the permeable membranes let the sodium in, viz. $\rightarrow \text{Na}^+$ $\leftarrow \text{K}^+$. This results not only in hypokalaemia from loss of potassium in the urine, but in hyponatraemia without any *external* loss of sodium.

Hypokalaemia can occur suddenly or gradually.

Sudden hypokalaemia is unlikely to be encountered in surgical practice. It occurs most frequently in diabetic coma treated by insulin and prolonged infusion of saline solution.

Gradual hypokalaemia is the type met with in surgical practice. The diarrhoea from ulcerative colitis and the loss from external fistulae of the alimentary tract are fruitful causes (e.g. duodenal fistula, ileostomy); the potassium content of the discharge from some of these fistulae is twice that of the plasma potassium concentration. Another frequent cause of hypokalaemia is prolonged gastroduodenal aspiration with fluid replacement by intravenous dextrose-saline solution. It is also prone to occur in the post-operative period following extensive resections for carcinoma of the alimentary tract, because often the operation has to be undertaken after months of weight-loss and potassium depletion.

Clinical Features.—The patient lies listlessly in bed. Speech is slurred and slow; often the patient fails to complete a sentence. Intense drowsiness soon follows and all the patient desires is to be left in tranquillity to sleep. Muscular hypotonia is the outstanding physical sign. Reflexes are lost and incontinence of urine is common. Abdominal distention amounting to paralytic ileus is a constant accompaniment, and in all cases of paralytic ileus the plasma-potassium value should be investigated.

Weakness of the respiratory muscles frequently occurs and results in rapid, shallow, gasping respirations; these are conducive to post-operative pulmonary complications. The diastolic blood-pressure is low, but there is usually a bounding pulse and a presystolic murmur. The diagnosis should be confirmed by electrocardiography, which may show a prolonged QT interval, depression of the ST segment, and a lowering or inversion of the T wave (fig. 72).

Treatment

Oral Potassium.—

Potassium is usually given in the form of milk, or of meat extracts and fruit juices. In more advanced cases potassium chloride,

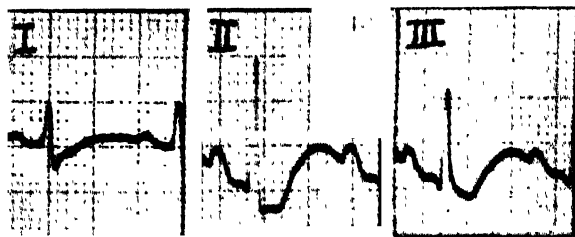


FIG. 72.—Electrocardiographic changes in severe hypokalaemia. The plasma potassium estimation was 1.6 mEq./litre. (W. F. Walker, F.R.C.S., Dundee.)

30 grains (2 G. effervescent tablets), is given by mouth six-hourly. Should the patient be so sleepy as to make swallowing difficult, the potassium is given by a transnasal intragastric drip.

Intravenous Potassium.—Should ingestion be inadequate or contraindicated, this route must be used. It is not without the danger of giving too much too quickly (especially when renal function is impaired), thus raising the plasma concentration to a dangerous level and causing cardiac arrest. Administration should be properly controlled; the plasma level of potassium should be checked daily; the urine output must be adequate (500 ml. per twenty-four hours—20 ml. per hour).

When there is associated alkalosis, an ampoule containing 2 G. (52 mEq.) of potassium chloride is added to 500 ml. of 5 per cent. dextrose solution, and this is gravitated into a vein at the rate of 20 drops per minute. Potassium solution must be given slowly.

When there is no associated alkalosis, the potassium deficit can be restored safely by Darrow's solution (p. 89).

It is of paramount importance to record the pulse-rate half-hourly during the administration of potassium, and if the pulse becomes slow, to substitute 5 per cent. dextrose solution for the potassium infusion. Not more than 3 G. (80 mEq.) of potassium is given in twenty-four hours.

Estimation of Electrolyte Balance by Flame Photometry.—Rapid and frequent estimations of plasma sodium and potassium can be made by this method. For full electrolyte determinations it is best to take sufficient blood from a vein to fill to the brim a small tube (6-7 ml.) into which a small quantity of standard lithium heparin solution has been introduced and dried. Replace the stopper, mix by repeated inversion and dispatch to the laboratory immediately. Every effort must be made to avoid hæmolysis. The plasma separated from hæmolysed blood is useless for potassium estimations.

The Plasma-Sodium Level.—Sodium, with its equivalent anions, accounts for about 90 per cent. of the osmotic pressure of the plasma. Changes in the sodium content coincide with changes in the osmolarity of the plasma, and so of all the body fluids. The best index of the total electrolyte concentration of the plasma is the sodium value, which normally is between 137 and 147 mEq. per litre. Whenever possible the *plasma chloride* and *bicarbonate* should be estimated simultaneously, since variations in the one may be accompanied by opposite changes in the other. The normal level of chloride is 95 to 105 mEq. per litre, and of bicarbonate 25 to 30 mEq. per litre; the sum of the two remains roughly constant at 125 to 135 mEq. per litre.

Potassium deficiency is present if the plasma-potassium value is less than 3.5 mEq. per litre. The normal range is 4.0 to 5.5 mEq. per litre. It must be remembered that intracellular potassium deficiency may be present although the plasma concentration is normal, and that deficiency is to be expected if oral feeding has been withheld for more than four days. Estimation of potassium in the urine or aspirated gastro-intestinal contents serve as a guide to the rate of depletion and the replacement necessary (W. J. Griffiths). Electrocardiography may also be used.

NORMAL ELECTROLYTE VALUES FOR PLASMA

Na . . .	137-147 mEq./litre = 315-338 mg./100 ml.
K . . .	4-5.5 mEq./litre = 16- 22 mg./100 ml.
Cl: . . .	95-105 mEq./litre = 550-620 mg./100 ml.
HCO ₃ : . .	25- 30 mEq./litre = 55- 70 vol. CO ₂ /100 vol.

PARENTERAL FLUID THERAPY

To overload the circulation is a grievous fault, and grievously does the patient pay for it.

The administration of fluid by any route other than the alimentary canal, i.e. intravenous, intramuscular, subcutaneous, or into the bone marrow, is known as parenteral administration.

The sterility of a solution for parenteral use must be assured. Furthermore, the solution must be free from dead bacteria and other particulate matter, as also must the delivery tube. For these reasons proprietary solutions manufactured on a large scale, with facilities for the prevention of contamination that cannot be emulated by a hospital pharmacy, are now used extensively. (fig. 73).

Solutions mainly in use:

1. **Dextrose 4.3 per cent. with Saline 0.18 per cent. (one-fifth normal saline).**—This solution, which is isotonic, may be regarded as the standard solution to employ. Usually it is referred to as 'dextrose-saline'.

2. **Isotonic (0.9 per cent.) saline solution** is needed in the following conditions: when a large amount of sodium has been lost by vomiting, or by gastric, duodenal, or intestinal aspiration or through an alimentary fistula. Possibly, on occasions, excessive sweating may justify its use.

These two solutions meet all the requirements in 95 per cent of cases.

3. **Double-strength saline (1.8 per cent.)** is used in doses of 500 ml. when there is severe sodium and chloride depletion. The plasma-chloride level will rise more quickly than sodium. *Sodium lactate* (m/6 or 1.86 per cent.), or *sodium bicarbonate* (2.25 per cent.) may then be used to raise the sodium level. These latter solutions are used in conjunction with normal saline in the treatment of metabolic acidosis (p. 96).

4. **Dextrose 5 per cent.** is an isotonic solution that supplies calories without electrolytes. It is useful mainly in the very early post-operative period when sodium excretion is reduced (p. 84). It is also valuable when the salt requirements of a patient needing much fluid have been satisfied on a particular day. Prolonged administration of 5 per cent. dextrose solution is liable to result in hyponatraemia, and may cause thrombosis of the vein used.

5. **Darrow's solution** contains sufficient potassium to combat hypokalaemia and in surgical practice it is a safe and convenient method of supplying this cation provided alkalosis is not present (p. 95). After four days of parenteral fluid therapy a litre of Darrow's solution can be given as a prophylactic measure against hypokalaemia. The composition of the solution is as follows:

B	Potassium chloride . . .	2.7 G.	Molar sodium lactate . . .	50 ml.
	Sodium chloride . . .	4.0 G.	Water for injection to . . .	1,000 ml.



FIG. 73.—Plastic bags are replacing glass bottles for intravenous therapy. The quantity given is registered on the spring balance which is graduated in ml. or G.

The rate of infusion should not exceed 60 drops a minute.

Continuous Intravenous Infusion (Venoclysis) (fig. 74).—Because of the certainty with which the fluid enters the circulation without the necessity of being absorbed, this is by far the most usual method of parenteral administration. If a suitable vein can be

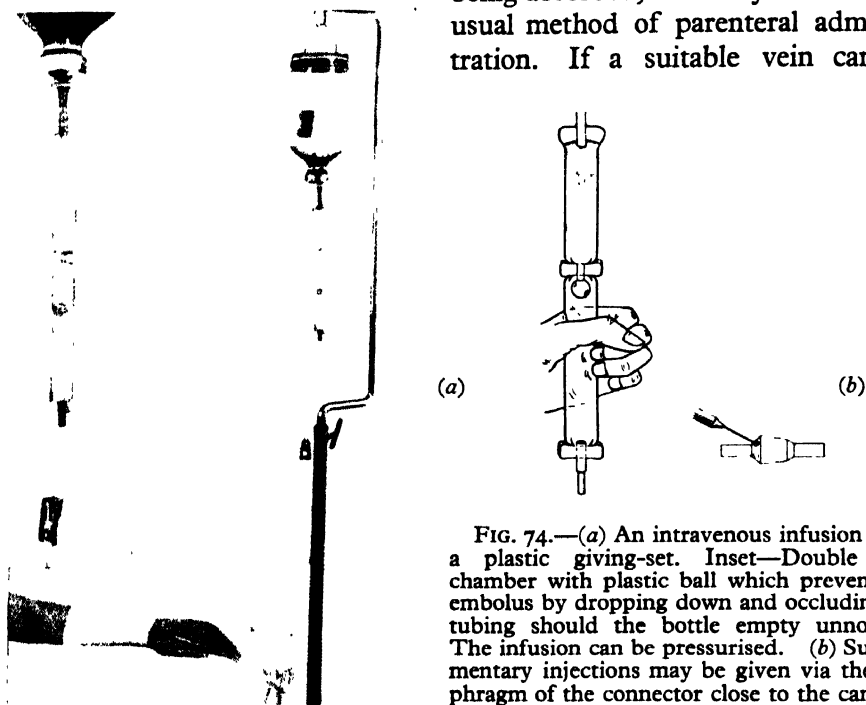


FIG. 74.—(a) An intravenous infusion using a plastic giving-set. Inset—Double drip chamber with plastic ball which prevents air embolus by dropping down and occluding the tubing should the bottle empty unnoticed. The infusion can be pressurised. (b) Supplementary injections may be given via the diaphragm of the connector close to the cannula.

rendered prominent by the application of a light tourniquet, it can be entered by a hollow needle (venepuncture) (fig. 75). The limb is then immobilised by placing it on a light splint or, if a vein of the forearm is used, with a few strips of adhesive plaster. Frequently a length of silicone-treated P.V.C. catheter¹ is threaded through a wide-bore needle up into a large vein.

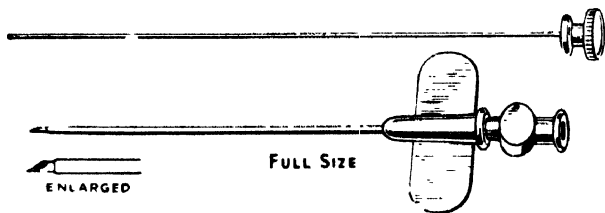


FIG. 75.—Intravenous Guest cannula. The modern tendency is to replace the metal cannula by a plastic one or a length of P.V.C. silicone-treated tube (e.g. 'Intracath').

Site.—As a routine measure, a vein in the upper third of the forearm, or one on the back of the hand is chosen (fig. 76). In women, young children, and even some men, especially when the venous pressure is low, a visible

¹ The manufacturer's instructions concerning the insertion and fixation of a plastic catheter must be followed carefully, as fracture of the tube and disappearance of part of it up a vein has occurred. The catheter must never be withdrawn through the needle as it is likely to be stripped or cut by the sharp edge of the bevel. If difficulty is encountered, both needle and catheter should be withdrawn at the same time.

or palpable vein may not be found in the arm; consequently another site must be selected. When this is the case, if a transverse incision is made over the surface marking of the long saphenous vein 2 cm. above and in front of the medial malleolus, the commencement of the long saphenous vein can always be found. Nevertheless, because of the risk of thrombophlebitis and pulmonary embolism, a vein of the leg should not be selected if it can reasonably be avoided. This injunction does not hold good for infants and small children, who are singularly immune to thrombophlebitis. If, in cases where the forearm veins are not visible, the cephalic vein in the delto-pectoral groove is selected, almost every requirement will be satisfied (Antia).

Contraindications to Intravenous Therapy.

1. *The Failing Heart.*—A history of dyspnoea on exertion, uncompensated valvular disease, or any possibility of cardiac weakness should call for hesitation in increasing the bulk of circulating fluid.
2. *Pulmonary Congestion.*—In all conditions where there are signs of œdema of the bases or consolidation of the lungs the method should be absolutely forbidden.
3. *Hypertension.*—If the blood-pressure is high, it is obviously courting danger to burden the circulation still further.
4. *Poor Renal Function.*—Severe, acute, or chronic pyelonephritis; bilateral (back pressure) hydronephrosis; nephritis in all its stages (salt-losing nephritis excepted). In cases falling into any of the above categories, the necessary fluid must be administered by another route.



FIG. 76.—Hand or forearm veins are used to give the patient maximum mobility.

Clinical Guide to Fluid Requirements.—When a patient is admitted in a dehydrated condition it is of course impossible to measure the loss of fluid and electrolytes he has sustained, but a detailed history of the nature and quantity of the fluid lost (usually, in surgical practice, by vomiting), and particularly the appearance of the patient (p. 93), are the means by which the amount of electrolytic solution he should receive is estimated. The hæmatocrit reading (p. 68) may be a help in this direction. In most instances the replacement is commenced by giving isotonic (0.9 per cent.) saline solution intravenously. When the bulk of the loss has been made up in this way, the *maintenance* of fluid balance is effected with dextrose-saline solution. In cases of extreme dehydration, where peripheral circulatory impairment (shock) is in evidence, it is necessary to commence by supplementing the volume of circulating fluid by dextran or plasma infusion (p. 76), and to follow this by substituting isotonic saline solution. When the fluid loss is considered to be rectified and fluid by mouth is contraindicated,

it is again advisable to supply the maintenance fluid requirements in the form of dextrose-saline solution.

In infants with dehydration, the amount of fluid required will be approximately the same as the weight loss, i.e. the difference between the estimated and the actual weight. (1 G. equals 1 ml.)

Rate of Flow.—Using a plastic transfusion set, 15 drops of fluid are approximately equal to 1 ml. At a rate of 30 drops per minute 1 litre will be given in eight hours, at 45 drops per minute it will take six hours, and at 60 drops some four hours. In severely dehydrated patients the rate of the first hour can be accelerated to well over 100 drops per minute, after which it should be cut to 50 or 60 drops. The maintenance dose rate is about 30 drops (3 L. in twenty-four hours).

The programme of rates to be used should be written down on the treatment card or fluid balance chart, and without further instructions the nursing staff should not exceed this rate.

Charting Fluid Intake and Output (fig. 77).—When fluid is administered intravenously, or by any alternative route, it is absolutely essential that

FLUID BALANCE CHART (24 Hour).

OXF. 59

TIME	INTAKE (Type and Quantity in ml.)				OUTPUT (In ml.)		
	ORAL	RECTAL OR GASTRIC DRIP	INTRAVENOUS		GASTRIC ASPIRATION VOMIT	URINE	OTHER Drainage Fistula, etc
			TYPE	Vol			
8 a.m.							
9 a.m.							
10 a.m.							
11 a.m.							
12 a.m.							
1 p.m.							
2 p.m.							
3 p.m.							
4 p.m.							
5 p.m.							
6 p.m.							
7 a.m.							
24 hr. Total							
24 HOUR INTAKE Oral + Rectal + IV				24 HOUR OUTPUT Aspiration + Urine + Other			
ml				ml			
24 HOUR BALANCE				ml			
				ORDERS FOR NEXT 24 HOURS			
INTRAVENOUS RECORD TYPE OF FLUID ONLY AT COMMENCEMENT OF EACH BOTTLE & AMOUNT ACTUALLY GIVEN AT COMPLETION OF EACH BOTTLE				Na			
				K			
				Cl			
				CO ₂			
				Protein			
				Urea			
Name							
HOSPITAL NUMBER				Ward Date			

FIG. 77.—The Westminster Hospital fluid balance chart.¹ There is no column for insensible loss because the nurse is unable to measure this. Insensible loss is taken into consideration by the Medical Officer when he writes up orders for the ensuing twenty-four hours.

the amount of fluid the patient receives should be recorded. It is equally important that all urine passed during each twenty-four hours should be measured and recorded. To this figure is added fluid recovered by gastrointestinal aspiration, and that lost from an intestinal, urinary, biliary, or pan-

¹ Obtained from Norman Starbuck and Co. Ltd., Bank Buildings, Cranleigh, Surrey.

creatic fistula plus 1,000 ml. for insensible loss from the lungs and skin. If the patient has been sweating, the last figure is increased.

Other Aids to Maintaining Correct Fluid Balance.—In addition to the fluid balance chart, the following also are important :

1. **Clinical.**—Much information can be gleaned at the bedside.

If the fluid balance is correct, a warm, dry, pink, elastic skin denotes a good circulation and a satisfactory blood volume.

Circulatory overloading requires constant watchfulness, for the initial symptoms are nil and the signs non-proclamatory. A raised jugular venous pressure (fig. 78) indicates that the patient has received too much fluid, or too much salt.

2. **Blood Indices.**—Plasma electrolyte estimations, as well as those of the hæmoglobin and the hæmatocrit and the plasma-proteins, are all helpful in ascertaining that the amount of fluid the patient is receiving is adequate, yet not excessive.

3. **Weight.**—Daily weighing is extremely useful in assessing that the amount of fluid given is correct. In patients subsisting entirely on parenteral fluid there should be a daily loss of weight of 150 G. (5 oz.).

4. **Blood Volume Estimations**

(p. 68).—These are valuable especially if a preoperative base line reading has been obtained in patients undergoing operations that are likely to lead to circulatory disturbances.

Vitamin Requirements.—Unless total intravenous therapy continues for more than a week, there is no need to supply fat-soluble vitamins, of which there is an adequate store in the body. The requirements of the vitamin B complex vary considerably, but more is required if there is any disturbance of dextrose metabolism. Antibiotics produce vitamin deficiency, notably of complex B. Therefore it is best to err on the side of safety and assume that a liberal amount of vitamins B will be required. Vitamin C should be given in large doses, to ensure saturation.

Twin ampoules of Parenterovite¹ are recommended daily. They should be mixed before being injected intramuscularly or via the intravenous drip.

Hypoproteinæmia.—Except in the presence of a profuse discharge from a duodenal or jejunal fistula, or extensive burns, hypoproteinæmia is most unlikely to develop within five days of total parenteral sustenance with crystalloid solutions. As a rule the patient has a lean and hungry look. Œdema is likely to develop, especially in the area of the wound, leading to delayed healing. Œdema of the pyloric mucous membrane, causing delay in gastric emptying, is frequently present in this condition. Œdema does not develop until the plasma albumin² is grossly depleted and the body reserves of mobile proteins are exhausted. Once present, hypoproteinæmia takes a long time to remedy, even when the patient can take nourishment by mouth. Milk



FIG. 78.—Fullness of the external jugular vein in a patient undergoing fluid therapy indicates that too much fluid has been given (ref. Central Venous Pressure, p. 69).

¹ Contains large doses of vitamin B complex and ascorbic acid (Vitamins Ltd.).

² Normal value 3.6 to 4.5 G. per 100 ml.

fortified with powdered skimmed milk is an excellent source of protein. In other circumstances protein hydrolysates (e.g. Aminosol) must be given intravenously. As an emergency measure the administration of 1 pint (568 ml.) of plasma will supply the patient with his immediate protein needs.

ALTERNATIVE ROUTES FOR PARENTERAL FLUID THERAPY

The introduction of hyaluronidase has made the use of continuous subcutaneous infusion popular once again.

Hyaluronidase, which causes diffusion, and therefore acceleration of absorption of fluids injected or gravitated subcutaneously or intramuscularly, is a mucolytic enzyme manufactured from bulls' and whales' testes, and is available as a sterile powder in ampoules of 1,000 units. The powder is readily soluble in sterile water, and the solution must be prepared immediately before use. Hyaluronidase must be injected before the fluids to be infused, and the most convenient method is to inject the enzyme through the rubber tubing of the infusion apparatus about 1 inch (2.5 cm.) above the needle as soon as the fluid has commenced to gravitate into the chosen site. Usually 1,000 units of hyaluronidase are sufficient to accelerate the absorption of 1,000 ml. of fluid.

Contraindications to the use of Hyaluronidase.—Note that hyaluronidase is completely ineffective if the patient is in a state of severe shock or is suffering from hypoproteinæmia or venostasis. It is also important to remember that with this enzyme it is dangerous to give dextrose solution without sodium chloride to patients with electrolyte deficiency. In this circumstance diffusion of the already depleted sodium and potassium salts from the blood-stream into the infusion area may be sufficient to induce a shock-like state.

Continuous subcutaneous infusion (hypodermoclysis), administered correctly, does not cause much discomfort. Should the infusion be given into an unsuitable site, or too rapidly, discomfort becomes pain.

Because of its loose subcutaneous tissue and rich lymphatic network, the site of election for subcutaneous infusion is the axilla. With a Y-shaped connection, both axillæ can be utilised simultaneously. For an infant, the region of the scapula is suitable.

Continuous rectal infusion (proctoclysis) was introduced by the celebrated J. B. Murphy, of Chicago, yet to-day it is seldom employed in N. America. The administration of fluid via the rectum has the advantage of simplicity. It requires but little apparatus, and neither asepsis nor isotonicity.

Avery Jones advises *dilute* saline solution for proctoclysis, viz. to 1 pint (568 ml.) of isotonic saline solution is added 4 pints (2.3 litres) of *tap water*. Dilute saline given per rectum avoids the absorption of an excessive quantity of water that may well result in hydræmia. Via a catheter in the rectum, the fluid is given by the drip method according to the needs of the patient. The rate of administration should not be greater than 50 drops a minute. It should be noted that there is no point in administering dextrose per rectum for its calorie value, because too little of it is absorbed.

Transnasal Intra-gastric Drip.—When it is unnecessary to keep the alimentary tract at rest and the patient cannot or will not drink a sufficient quantity of fluid, a simple and effective method of ensuring an adequate fluid intake is to gravitate the fluid by the drip method into the stomach through a transnasal intra-gastric polythene or Ryle's tube.

ACID-BASE BALANCE

In health the reaction of the blood lies within the range of pH 7·36 to 7·44. The terms *acidæmia* and *alkalæmia* imply a hydrogen ion concentration respectively above and below the normal range.

Acidosis is now defined as a condition which would cause acidæmia if uncompensated and *alkalosis* as a condition which would cause alkalæmia if uncompensated. The pH of the blood is regulated and controlled by various buffering systems essentially consisting of weak acids (and bases) of which the most important is carbonic acid (and the bicarbonate base). It is also regulated by the removal of CO₂ by the lungs and by the excretion of both acids and bases by the kidneys.

The fraction $\left[\frac{\text{HCO}_3^-}{\text{H}_2\text{CO}_3} \right]$ is the variable factor determining the pH. A decrease in the fraction leads to increased acidity, and vice versa.

[HCO₃⁻] can be altered by metabolic factors, while [H₂CO₃] is subject to alteration by respiratory factors, alteration of the one being followed automatically by a compensatory alteration of the other, so that in health the ratio $\left[\frac{\text{HCO}_3^-}{\text{H}_2\text{CO}_3} \right]$, and therefore the pH of the medium, remains constant.

ALKALOSIS

1. **Metabolic alkalosis** (a condition of base excess or a deficit of any acid other than CO₂) can be caused by:

- (a) Excessive ingestion of absorbable alkali. Such is not uncommon in patients who take proprietary indigestion remedies without medical supervision.
- (b) Loss of acid from the stomach by repeated vomiting or aspiration.
- (c) Cortisone excess, usually the result of over-administration of adrenal corticoids, but occasionally due to Cushing's syndrome (p. 579).

Compensation is effected by (i) retention of carbon dioxide by the lungs and (ii) excretion of bicarbonate base by the kidneys (alkaline urine).

Alkalosis due to loss of acid from the stomach is by far the most common and most important. In its most typical form it is seen in patients with pyloric stenosis in whom the loss of acid by repeated vomiting is often accentuated by the taking of medicines containing sodium bicarbonate. The most striking feature of severe alkalosis is Cheyne-Stokes' respiration with periods of apnoea lasting from five to thirty seconds. Tetany sometimes occurs. Latent tetany (in which the serum calcium remains just above the low value necessary for tetanic muscular contractions) is more common, and can be unveiled by Trousseau's sign (p. 569). Regarding other signs, the dual phenomenon of severe alkalosis and hypokalæmia (p. 87) are so interwoven that their clinical separation is well-nigh impossible. Subclinical degrees of alkalosis are recognisable only by a raised plasma-bicarbonate concentration (Table of Normal Values, p. 88). Severe alkalosis may result in renal epithelial damage, and consequent renal insufficiency.

Treatment.—The replacement of potassium is the primary consideration, and it is administered first in the same manner as has been described under

hypokalaemia (p. 87), to be followed by isotonic saline, according to the cause and degree of loss.

2. **Respiratory alkalosis** (a condition where the CO_2 tension in the arterial blood P_{CO_2} is below the normal range of 36–44 mm. Hg.) is caused most commonly in surgical practice by excessive manual pulmonary ventilation carried out upon an anaesthetised patient who has been given a muscle relaxant. Other causes are hyperventilation occasioned by high altitudes, hyperpyrexia, a lesion of the hypothalamus, and hysteria. Compensation, which depends on increased renal excretion of bicarbonate, usually is inadequate. During anaesthesia alkalosis is accompanied by pallor and a fall of blood-pressure. In severe cases respiratory arrest follows.

Treatment.—Respiratory arrest due to alkalosis is rectified by insufflation of carbon dioxide.

ACIDOSIS

1. **Metabolic acidosis** (a condition where there is a deficit of base or an excess of any acid other than CO_2) occurs as a result of:

(a) *Loss of bases* such as occurs in sustained diarrhoea, ulcerative colitis, gastrocolic fistula, a high intestinal fistula, or prolonged *intestinal* aspiration.

(b) *Increase in fixed acids* due to the formation of ketone bodies as in diabetes or starvation, the retention of metabolites in renal insufficiency, and the rapid increase of lactic and pyruvic acids which follows cardiac arrest (Chap. 30), or the release of the clamped aorta in the surgery of abdominal aneurysm (p. 127). Acute acidæmia with pH levels of 7·1 are frequently encountered in such cases.

Clinical Features.—In severe acidosis the leading sign is rapid, deep, noisy respirations which are unremitting save, perhaps, momentarily while the patient endeavours unsuccessfully to moisten his dry lips with his parched tongue. This hyperpnoea is due to over-stimulation of the respiratory centre by the reduction of pH of the blood, and the physiological purpose of over-breathing is to eliminate as much as possible of the acid substance CO_2 . Except in renal acidosis, the pulse-rate and the blood-pressure are raised. The urine is strongly acid. In all cases the plasma-bicarbonate content, or alkali reserve, is lowered (Table of Normal Values, p. 88).

Treatment.—Cases belonging to class (a) are readily rectified by the administration of Darrow's solution. When dehydration is in evidence the prelude to treatment by Darrow's solution should be rapid infusion of sufficient isotonic saline solution to restore the extracellular fluid volume. One-sixth molar lactate solution may be required. The treatment of acidosis due to renal failure is discussed in Chapter 44.

The acute acidæmia after releasing a cross-clamped aorta or in cardiac arrest requires the infusion of 50–150 ml. of 8·4 per cent. sodium bicarbonate solution (see 'bedside biochemistry' below).

Acidosis due to transplantation of the ureters into the colon is discussed in Chapter 45.

2. **Respiratory Acidosis** (a condition where the P_{CO_2} is above the normal range) is caused by impaired alveolar ventilation.

In practice this problem most commonly occurs when there is inadequate ventilation of the anaesthetised patient, or when the effects of muscle relaxants have not worn off or been reversed at the end of the anaesthetic. There is also a risk of respiratory acidosis when the patient undergoing surgery already has pre-existing pulmonary disease (e.g. chronic bronchitis or emphysema) and this is accentuated by thoracic and upper abdominal incisions.

MEASUREMENT OF ACID-BASE DISTURBANCES (BEDSIDE BIOCHEMISTRY)

In addition to the older and better known methods of assessing the metabolic balance of the patient (such as the standard bicarbonate), it is important to know the respiratory balance of a patient. This increasing need for complex 'bedside biochemistry' (C. F. W. Illingworth) is met with to some extent by direct estimations of the pH, P_{CO_2} and P_{O_2} of arterial blood, though in most situations the pH and P_{CO_2} can be measured using capillary blood. The Astrup apparatus (Radiometer) (fig. 79), is commonly used for such estimations in the surgical laboratory.

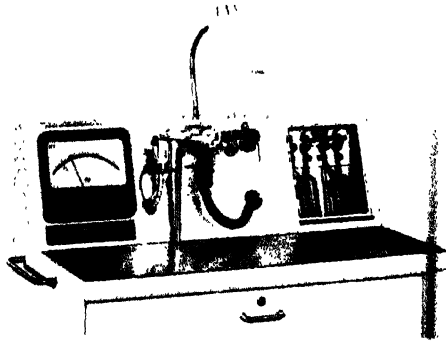


FIG. 79.—The micro-Astrup blood pH equipment consists of a pH meter calibrated from pH 6.8 to 8.2. In the centre is the micro-electrode unit which includes glass and calomel electrodes and the micro-tonometer which equilibrates heparinised blood samples with two CO_2 and O_2 mixtures of known carbon dioxide tensions. Both the electrode unit and tonometer are enclosed in a water jacket thermostatically controlled at $38^\circ C$. To the right of the instrument is found the humidifier assembly where the two gas mixtures are bubbled through water at $38^\circ C$. The cabinet underneath contains the cylinders of CO_2 - O_2 mixtures.

Sir Charles Frederick Wilham Illingworth, *Contemporary*. Emeritus Regius Professor of Surgery, University of Glasgow.

Poul Astrup *Contemporary*. Department of Clinical Chemistry, Rigshospitalet, Copenhagen.

CHAPTER 7

SKIN. BURNS. SKIN GRAFTING

CALLOSITIES, CORNS, AND WARTS

A **callosity**¹ (*French: callosité*) is a localised thickened or hardened part of skin caused by friction. It is commonly occupational, e.g. on a gardener's hand or the finger of a violinist.

A **corn** (*Old French: corn = grain*) is a horny induration of the cuticle with a hard centre, caused by undue pressure, chiefly affecting toes and feet.

Most corns yield to the chiropodist, provided that footwear is suitable. Skilled treatment is important in patients with diabetes or a poor peripheral circulation, as secondary infection may precipitate gangrene. Salicylic acid in collodion (20 per cent.) applied for a few nights, followed by soaking in hot water, is often effective in removing a corn.

A **wart** (*Old English: wearte*) is a dry rough excrescence on the skin. *The common wart* usually occurs on the hands, particularly in children. It is due to a virus and is infectious. As warts often disappear spontaneously, any treatment which is in use at the time will gain an undeserved reputation as a wart 'cure'. Curetting and cauterisation of the centre is the radical treatment, but further crops may appear.

Plantar warts occur in the sole, and are usually multiple. They may be so tender as to render standing or walking exceedingly uncomfortable.

Treatment.—The nightly application of formaldehyde lotion for six to eight weeks is effective in 60 per cent. of cases. Curettage is more expeditious and certain. Under a local or general anæsthetic, a scalpel, held vertically to the skin, incises the hard skin at the boundary of the lesion. A sharp curette can be inserted beneath the wart to remove it.

The condition is due to infection with a virus, and the footwear must be treated if re-infection is to be avoided. Cotton socks should be worn, as these can be boiled. Shoes can be disinfected by formalin vapour.

Seborrhæic (syn. 'Senile') warts, see p. 102.

Venereal warts and moist warts (*papillomata acuminata*), see Chapter 48.



FIG. 80.—Warts on the hand.

INFECTIONS

Impetigo is an intradermal infection. When caused by the streptococcus the primary lesion is bullous, which soon ruptures to form an erosion and then a crust. In the staphylococcal type, the primary lesion is also bullous, but the bullæ are more durable (e.g. in neonatal pemphigus). The infection is contagious, and in rugby football one player so infected can spread the disease amongst his team mates and the

¹ Other definitions of names of skin lesions in common usage include:

Nævus (*Latin: Nævus*) = A birth mark, but often used to mean simply a mark or blemish on the skin (see *Hæmangioma* and *Melanoma*).

Mole (*Anglo-Saxon: Mâl = a lump*) = A spot or blemish on the skin, sometimes hairy.

Mollusc (*Latin: Molluscus = soft*) = A soft protuberance on the skin.

opposing side (the condition being known as 'scrum-pox'). *Treatment* includes the careful washing of the face to remove crusts, using hexachlorophene soap and lotion (1 per cent.). Systemic antibiotics, according to the sensitivity tests, are only used in those cases which are resistant to local treatment.

Boil (syn. Furuncle).—A boil is an acute staphylococcal infection of a hair follicle, with perifolliculitis, which usually proceeds to suppuration and central necrosis. A painful and indurated swelling appears which gradually extends. After two or three days, the centre softens and a small slough is discharged with a bead of pus, and in the large majority of cases the condition then subsides. A 'blind boil' is one which subsides without suppuration. Boils are common on the back of the neck and in other hairy areas. A *stye* (syn. *hordeolum*) is due to infection of an eyelash follicle. Infection of a perianal hair follicle (perianal abscess), with consequent suppuration, is likely to result in a sinus. Furunculosis of the external auditory meatus is extremely painful, as the skin is attached to the underlying cartilage, and swelling is accompanied by considerable tension.

Complications of Boils.—(i) Cellulitis, especially in debilitated subjects. (ii) Infection of the lymph nodes draining the affected part. (iii) Secondary boils due to infection of neighbouring hair follicles.

Treatment consists in improving the general health of the patient, since boils are frequently associated with overwork, worry, debility, examinations, or other undermining influences. Incision is unnecessary, as a touch of iodine or liquid phenol on a skin pustule will hasten necrosis of the overlying skin so that pus can then escape. Should softening occur around a hair follicle, particularly an eyelash (*stye*), removal of the appropriate hair allows the ready escape of pus. The sensitivity of the organisms is determined and the appropriate antibiotic may be given. Washing the surrounding skin twice a day with suitable disinfectant, e.g. hexachlorophene, discourages the development of secondary boils. A paste, composed of anhydrous magnesium sulphate (24 parts) and glycerin (11 parts) exercises a valuable osmotic effect.

Carbuncle.—This is an infective gangrene of the subcutaneous tissues, due to staphylococcal infection. It is uncommon before the age of forty, and males are the usual sufferers. *Diabetes* may be present.

A carbuncle often occurs in the nape of the neck, where the skin is coarse and ill-nourished.

The patient complains of tenderness and stiffness at the site of origin of the carbuncle. The subcutaneous tissues become painful and indurated, and the overlying skin is red (fig. 81). Unless the condition is aborted by prompt treatment, extension will occur, and after a few days areas of softening appear. The skin then gives way and thick pus and sloughs discharge. Usually there is one central large slough, surrounded by a 'rosette' of smaller areas of necrosis. In-



FIG. 81.—Carbuncle of the neck.
(Herbert Bourns, F.R.C.S.)

Carbunculus in Latin, *Anthrax* in Greek, is the word for charcoal. The Ancients saw in these conditions burning sores upon the skin—hence they likened them to charcoal.

fection sometimes extends widely, and fresh openings appear on the surface and coalesce with those previously formed.

A carbuncle on the cheek or upper lip is particularly dangerous, owing to the risk of cavernous sinus thrombosis (p. 458).

Treatment.—The general treatment and organism identification is similar to that described for boils. However, many carbuncles are aborted if penicillin is used adequately in the early stages. Local treatment by osmotic pastes is often supplemented by infra-red or short-wave diathermy.

Erysipelas and Cellulitis are described on pp. 6 and 14.

Lupus vulgaris (tuberculosis of the skin) usually occurs between the ages of ten and twenty-five, the face being the site of election. One or more cutaneous nodules appear, with congestion of the surrounding skin. On applying pressure with a glass slide the nodules are seen to be the colour of apple jelly. Extension occurs very slowly, but ulceration is likely to follow sooner or later (fig. 82). The resulting ulcer tends to heal in one situation as it extends to another. The

FIG. 82. — Early lupus vulgaris showing cutaneous nodules.

mucous membranes of the mouth and nose are sometimes attacked, either primarily or by extension from the face. Œdema occurs if the fibrosis caused by the lupus obstructs the normal lymphatic drainage. Infection in the nasal cavity may be followed by necrosis of underlying cartilage. Epithelioma is prone to occur in a lupus scar (fig. 83).

Treatment is by chemotherapy (p. 2). If healing is slow, the lesion should be excised.

CYSTS

A **sebaceous cyst** (*syn.* a wén) follows obstruction to the mouth of a sebaceous duct, and is therefore a retention cyst. Pathologically, it is classed as an epidermoid cyst (p. 50). It commonly occurs on the face or scalp (p. 352), but can occur anywhere except on the palms and soles, which are devoid of sebaceous glands.

A typical cyst appears as a hemispherical swelling, firm or elastic in consistency, and with no definite edge. It is more or less adherent to the skin, especially if it has been previously inflamed or is subjected to pressure. The punctum of the obstructed duct can sometimes be seen on the summit of the cyst,



FIG. 83. — Advanced lupus vulgaris. Carcinoma has developed on the upper lip.

and sebaceous material may be expressed from the duct. An uncomplicated cyst contains yellowish-white material composed of fat and epithelial cells, of a putty-like consistency, so it can often be indented by a finger-tip. Rarely, a minute worm, *Demodex folliculorum*, which is harboured in sebaceous glands, may be seen on microscopy of the cyst wall.

Complications

(i) *Infection*.—The cyst becomes enlarged and painful, and the overlying skin is red. After a few days the inflammation usually subsides, but recurrence is the rule. Recurrent attacks cause the cyst wall to become adherent to surrounding subcutaneous tissue, and consequently more difficult to remove. The contents of an infected cyst become semi-liquid and usually very foetid.

(ii) *Ulceration*.—An infected cyst may break down and discharge its contents. A foul ulcerated surface remains, which resembles an epithelioma, and to which the term Cock's 'peculiar tumour' is applied (fig. 84).

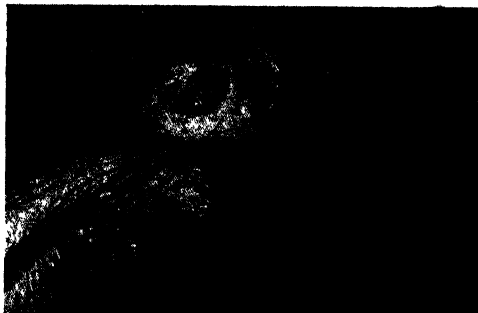


FIG. 84.—Cock's 'peculiar tumour.'

(iii) *Sebaceous Horn*.—The contents of a cyst sometimes escape slowly from the duct orifice and dry in

successive layers on the skin, forming a sebaceous horn (fig. 85 and facing title page).



FIG. 85.—Sebaceous horn.

Treatment.—(a) *Incision-Avulsion*.—Under local anæsthesia an incision is made through the skin, into the cyst. The contents are squeezed out and the cyst wall is seized with artery forceps and avulsed.

(b) *Dissection* is necessary for cysts which have been previously inflamed. An incision is made over the cyst, the wall is defined, and the cyst is dissected from adjacent

tissue and removed intact. Unless the wall is completely removed recurrence is probable.

Sequestration and Implantation Dermoid Cysts are described on p. 50.

PAPILLOMAS AND MISCELLANEOUS SKIN LESIONS.

Papilloma

A papilloma of the skin may be derived from either the squamous or the basal cell layers.

The *squamous cell papillomas* include the following types: (1) *Congenital* (*syn. Nævus Verrucosus*), which may be single or multiple, and it appears either at birth or early in life. It is a warty growth of brownish colour, but

Edward Cock, 1805-1892. Surgeon to Guy's Hospital, London.

Sir Astley Cooper, 1768-1841. Surgeon to Guy's Hospital; received a baronetcy and one thousand guineas for successfully removing an infected wen from the head of King George IV at Brighton in 1821.

large horny excrescences may be present. (2) *Infective Wart (verruca vulgaris)*—the common wart—described on p. 98. (3) *Soft Papilloma*, which often occurs on the eyelids of elderly people (fig. 86). (4) *Keratin Horn*.—This is also seen in old people and is a papilloma with excess keratin formation.



FIG. 86.—Soft papilloma.

Molluscum Fibrosum.—Soft fleshy skin tags which may be polypoid or filiform and which may occur in large numbers on the neck, trunk, and face in persons past middle age.

Molluscum Sebaceum (*syn. Kerato-acanthoma*).—Most commonly occurs on the face during the fifth and sixth decades. It presents as a firm hemispherical nodule, which may reach a size of up to 2 cm. in diameter in six weeks (fig. 88). The summit of the tumour ulcerates and forms a crust beneath which is a

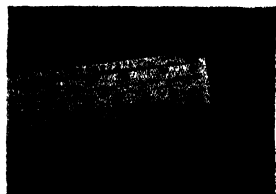


FIG. 88.—Molluscum sebaceum.

crater. After a time, the crust is shed and spontaneous healing occurs. This may leave a scar. The tumour may be curetted and a caustic (e.g. carbolic or silver nitrate) applied to the crater. A quicker cure is obtained by excision, which permits adequate microscopy. Undoubtedly in the past many cases were regarded as epitheliomas and helped to swell the number of

'cures' for this condition. Radiotherapy also is an effective treatment.

Benign Calcifying Epithelioma.—These are solitary, hard, tumours that appear to be growing from the deep surface of the skin. They occur most frequently on the face and upper extremities. The size usually varies between 0.5 to 3 cm. in diameter. The tumour may arise at any age but is common in childhood. Clinically it resembles a rather hard sebaceous cyst (fig. 89).



FIG. 90.—Sclerosing angioma.

Sclerosing Angioma (*syn. Dermatofibroma; Subepidermal Nodular Fibrosis*).—This occurs in skin as firm, indolent, single, or multiple nodules. The nodules, in adults, are situated most commonly on the extremities. As a rule they are only a few millimetres in diameter. Most are reddish, others are yellowish-brown (due to lipid) or blue-black (due to hæmosiderin). The latter may resemble melanoma (fig. 90).

Granuloma Pyogenicum (fig. 91).—This is a traditional

Basal Cell (Seborrhæic, or Senile, Wart).—Often develops in large numbers on the trunk, face, and arms of persons in or past middle life. Circular, slightly raised, warty, often brownish, they look as if they are stuck on the skin (fig. 87).



FIG. 87.—Basal cell seborrhæic wart.



FIG. 89.—Benign calcifying epithelioma.

but misleading name. The lesion looks like a hæmangioma but has a typical natural history. Usually single, it consists of a dull red, soft or moderately firm, more or less pedunculated nodule which grows rapidly to a size varying from 0.5 to 2 cm. in diameter. The surface shows atrophic epidermis, but often has crusts. It may occur anywhere on the skin, but most commonly it is seen on the face and the fingers. Treatment is excision.

Tumours of Accessory Skin Structures.—These are rare. (1) *Sebaceous nævus*: A congenital condition presenting as a pinkish-yellow plaque in the scalp. (2) *Sebaceous adenoma*: Isolated lesions are very rare. Multiple sebaceous adenomata occur in association with epilepsy and mental deterioration. (3) *Tumours of sweat glands*: Are closely related to the basal cell carcinomata but present deep to the skin and are often cystic (Papilliferous cystadenoma).

Sarcoid (Boecks) (p. 158).—This is a generalised disease which may affect skin. In the skin it occurs as reddish-brown nodules which are soft, and rarely ulcerate. Giant cells are found, but tubercle bacilli can never be isolated.

Rhinophyma (syn. Potato nose) (fig. 92).—This is a glandular form of Acne Rosacea. The skin of the nose, particularly the distal part, becomes immensely thickened and the openings of the sebaceous follicles are easily seen. The capillaries become dilated and the nose assumes a bluish-red colour. Surgical treatment, by paring away the excess tissue gives a great improvement.



FIG. 92.—
Rhinophyma.
(Dr. E. B. Ash,
Birmingham.)

HEMANGIOMA

A hæmangioma is a developmental malformation of blood-vessels rather than a true tumour, and is therefore an example of a hamartoma (p. 57). It may occur in any tissue of the body, but is most common in the skin and subcutaneous tissues. A hæmangioma is either capillary, venous (cavernous), or arterial in type.

Capillary Hæmangioma

(1) **Salmon Patch**.¹—This is present at birth over the forehead in the midline, and over the occiput. It disappears by the age of one year.

(2) **Port Wine Stain** (Nævus flammeus) (fig. 93).—Present at birth, it changes very little throughout life, although the colour may alter a little and it may become nodular in some areas. *Treatment* is for reason of appearance. The texture of the skin is quite normal and in a girl the blemish can be disguised by the skilful use of cosmetics. In a boy, treatment by excision and grafting may be considered. Radiation and other destructive treatment has been disappointing.



FIG. 94.—Strawberry
angioma.



FIG. 93.—Port
wine stain.

(3) **Strawberry Angioma** (fig. 94).—This common condition has a typical history. The baby

¹ Also called 'stork bites'!

is normal at birth and at the age of one to three weeks is noted to have a red mark. This increases rapidly for some weeks or even up to three months, until the typical strawberry or raspberry-like swelling is present. Clinically, the sign of emptying may be demonstrable. The lesion is composed of immature vaso-formative tissue. The subcutaneous tissue as well as the skin is often involved, and in severe cases the muscle layer may be affected. Sub-mucous nævi are prone to hæmorrhage, which is sometimes alarming (fig. 625).

From the age of three months to one year the nævus grows with the child. Then it ceases to grow. Eventually the colour fades and flattening occurs so that at the age of seven or eight years involution is complete. *Treatment*.—

The present evidence suggests that the final result (fig. 95) is better if natural involution is allowed to occur rather than if regression is hastened (as it can be) by any operative or physical methods (e.g. carbon dioxide snow, injection of hot water or sclerosant fluid, or hypertonic saline, etc). X-ray therapy carries a high risk of disturbance of growth and is dangerous.



FIG. 95.—Strawberry angioma. Same case as fig. 94 with conservative treatment, about 7 years.

(fig. 96). Usually it shows no tendency to involution and may become larger and more troublesome later. Sometimes the whole of one limb and the adjacent part of the trunk is affected (fig. 157). Occasionally the nævus is associated with a lipoma (nævo-lipoma). In some cases arterio-venous communications are present (p. 128). The skin overlying the nævus may be atrophic, and besides being in danger of severe hæmorrhage from trauma, the patient may suffer from septicæmia if organisms gain entry, in which case the energetic use of antibiotics is an urgent matter. *Treatment*.—In general this is conservative. The repeated injection of hot water and other sclerosing agents may have some effect, but the method is not generally employed.

Arterial (plexiform) Angioma is in fact a type of congenital arteriovenous fistula (p. 128). The swelling consists of arteries and arterialised veins which pulsate. The usual form is a *cirroid aneurysm* (p. 352).

Spider Nævus.—These may be associated with liver disease (Chap. 34) if present in the skin over the manubrium sterni, but they may occur quite innocently and commonly as isolated lesions. They show the characteristic sign of emptying.

Nævus tardes are small angiomas occurring in *adults* particularly around the mouth. They may be associated with vasospastic conditions and scleroderma (p. 139).

Lymphangioma are described on p. 153. Sometimes they are associated with a hæmangioma (hæmo-lymphangioma).

Venous Angioma (cavernous) is relatively uncommon. It is present at birth and consists of a multiplicity of venous channels of varying calibre



FIG. 96.—Venous angioma.

The usual form

PREMALIGNANT CONDITIONS OF THE SKIN

(1) **Bowen's disease** is an intradermal precancerous condition. A brownish induration with a well-defined edge appears in the skin (fig. 97). Microscopically large clear cells similar to those found in Paget's disease of the nipple (p. 606) are in



FIG. 97.—Bowen's disease of the abdominal wall, in which epithelioma has developed.

evidence. Sooner or later carcinoma develops, and wide excision is then necessary.

(2) **Paget's Disease of the Nipple** (p. 606).

(3) **Leukoplakia** (p. 496).

(4) **Senile (or Solar) Keratosis**.—Occurs frequently as multiple lesions on the face and dorsa of hands (fig. 98) in persons past middle life. Exposure to sun is the important predisposing factor. Usually the lesions are less than 1 cm. and have a dry, hard scale with little or no infiltration. Squamous cell carcinoma may develop.



FIG. 98.—Senile keratosis.

(5) **Radiodermatitis**.—(a) *Early*: Erythema occurs which goes on to desquamation and pigmentation. If the dose is very great, ulceration may occur. (b) *Late*: Atrophy, irregular hyper-pigmentation and telangiectasis and hair loss occur. Eventually, squamous cell carcinoma may develop.



FIG. 99.—X-ray scar of the neck in which carcinoma has developed (Marjolin's ulcer).

(6) **Chronic Scars** (Marjolin's Ulcer).—A carcinoma which develops in a scar (fig. 99) presents the following characteristics:

(i) It grows slowly, as the scar is relatively avascular.

(ii) It is painless, as scar tissue contains no nerves.

(iii) Secondary deposits do not occur in the regional lymph nodes, as lymphatic vessels have been destroyed. If the ulcer invades normal tissue surrounding the scar, it extends at a normal rate, and lymph nodes are then liable to be involved.

MALIGNANT DISEASE OF THE SKIN

Basal Cell Carcinoma (Rodent Ulcer).—A common tumour of low-grade malignancy which occurs in white-skinned people. Exposure to sun-

light is a predisposing factor and it is therefore common in Australia. It occurs in middle or late age and ninety per cent. of lesions are found on the face, usually above a line from the lobe of the ear to the corner of the mouth, the commonest site being around the inner canthus of the eye. Although often called a rodent 'ulcer' many of the lesions are non-ulcerated, and have a nodular appearance with a pearly or darkly translucent colour as if containing water, and with a network of fiery red blood-vessels on the surface. Thus the common types are: nodular, cystic, and ulcerated (figs. 100 and 101). An unusual type is the 'field fire' rodent (fig. 102).

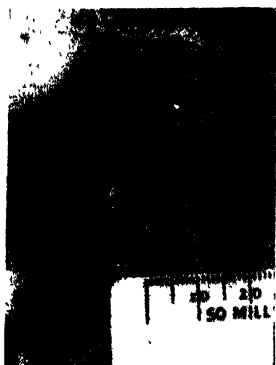


FIG. 100.—Rodent ulcer.

The ulcerated type has a typical appearance—a raised rolled edge, like a motor car tyre, with central ulceration (fig. 100). Temporary healing often takes place, to be followed by further ulceration with serous discharge and bleeding. The patient gives a history of a 'spot' which never really heals: 'it keeps scabbing over and breaking down'.

Though the tumours are slow-growing, if ulcerated they will, as the name implies, gradually erode deeper tissues, such as muscles, cartilage, and bone, producing severe disfigurement (fig. 101). Dissemination by lymphatics or the bloodstream does not occur. Rarely the ulcer takes on a squamous-cell carcinomatous change.

Microscopic examination shows masses of darkly staining cells with a characteristic arrangement—an outer palisade layer of columnar cells



FIG. 101.—Disfigurement caused by rodent ulcer.

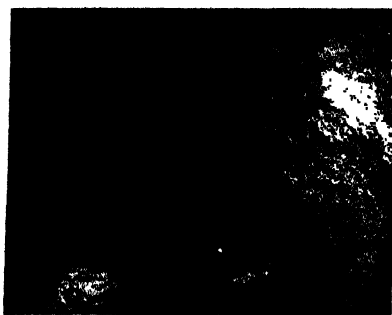


FIG. 102.—'Field-fire' type of rodent ulcer.

surrounding a central mass of polyhedral cells. Cystic spaces may be seen.

Treatment.—Surgery or radiotherapy is necessary. Excision allows the whole lesion to be examined. With small lesions the operation can be described as an excision-biopsy. It is essential to excise a margin of healthy tissue around and beneath the tumour. A skin graft (p. 114) may be needed to cover the defect. In large lesions some form of plastic repair by a pedicle-flap graft may be required—

cosmetic results should be good (fig. 114).

Cylindroma (*syn.* 'turban' tumour) is so called from the arrangement of the stroma in peculiar transparent cylinders. It is considered by some to be a basal-

celled carcinoma, although other authorities classify the tumour as an endothelioma. The tumour gradually forms an extensive turban-like swelling extending over the scalp. Ulceration is uncommon, and the tumour is relatively benign.

Squamous Cell Carcinoma (Squamous Cell Epithelioma, Epithelioma) (fig. 103).—This is less common, but it is more malignant and more rapidly growing than rodent ulcer. A squamous cell epithelioma can occur *de novo* in the skin of the face of elderly people, but more often it occurs in a pre-existing skin lesion, or as a result of past irradiation. The pre-malignant conditions are listed above, and other conditions include long-standing venous ulcers, chronic lupus vulgaris lesions, and the prolonged irritation of the skin by various chemicals, e.g. dyes, tar, or soot (p. 49).

A typical carcinomatous ulcer is irregular in outline, and the edges are raised and everted. The base is indurated and sooner or later becomes attached to the deeper structures. A blood-stained discharge occurs, which is increased in amount with the advent of secondary infection. The regional lymph nodes become involved, and the deposits are liable to undergo mucoid degeneration, to which secondary infection is sometimes added.

Treatment.—Treatment must be adequate. Immediately after biopsy confirmation of the diagnosis, the surgical treatment is wide excision, which may require some form of skin graft to the defect. Whether radiotherapy should be employed instead of surgery depends upon the condition of the patient, the size and attachments of the tumour, and the radiotherapy facilities.

The presence, or subsequent appearance, of mobile lymph nodes is an indication for block-dissection, unless it is considered that secondary infection is responsible for their enlargement, in which case the decision to operate will await the effect of the appropriate antibiotic. Fixed enlarged lymph nodes are not removable; some regression may be obtained by radiotherapy.

MELANOCYTIC TUMOURS AND MALIGNANT MELANOMA

Simple Melanocytic Tumours.—The melanocyte is either a specialised epithelial cell, or it is of neural origin. In normal skin the melanocytes appear as clear cells in the epidermis. In melanotic conditions they become increased in number in the layers of the skin, as follows : (1) in the epidermis (*Lentigo*), (2) in the deepest layer of the epidermis against the basement membrane (*Junctional naevus*), (3) in the dermis (*Dermal naevus*), and (4) when (1) and (2) are combined (*Compound naevus*). These simple melanocytic tumours usually arise in the skin, including the nail-bed



FIG. 103.—Squamous-celled carcinoma. The patient was a chemical worker.

(fig. 104), the nipple, the anal margin, the choroid of the eye, and they are occasionally submucous, e.g. the colon or the nasal fossa.

Clinical Features.—The clinical history is of great importance. The simple tumours fall into a number of clinical types according to history:

(1) *Congenital* (fig. 105). (a) The common type is a lightly or darkly pigmented lesion, often hairy and not infrequently papillary. If removed in childhood these lesions always show some junctional activity, but this subsides later. This mole never becomes malignant. (b) Giant lesions sometimes occur which may cover a quarter or more of the body surface, and often show irregular pigmentation. Malignant change can occur.



FIG. 104.—Melanoma of the nail bed.

(2) Pigmented *nævi* may appear at any age after birth, but their peak period is during the years around *puberty* when some boys or girls produce hundreds or even thousands of small pigmented *nævi* and lentigos. On section these *nævi* show junctional activity. The lesions grow for some years, and then become quiescent, but some may continue to grow and later become malignant.

(3) *Multiple nævi of middle age* commonly appear on the face, particularly in women. These lesions are often non-pigmented and appear as rounded swellings. They may become quite large and ugly. Microscopically the cells are all in the dermis and no cellular activity is seen. Malignant change does not occur.

Treatment is necessary (a) if the lesion by reason of its size and position is a nuisance, e.g. if cut when shaving, (b) for cosmetic reasons, or (c) because the history suggests that malignant change has occurred (see below). Surgical excision is the only treatment which should be considered, and all lesions should be sent for histological examination.



FIG. 105.—Congenital melanoma.

MALIGNANT MELANOMA¹

It is now generally agreed that a malignant melanoma should be regarded as a carcinoma—an epithelial melanocarcinoma of the skin, or a neuroectodermal carcinoma in the case of the eye. A malignant melanoma arises (a) most commonly in a previously existing mole—often a small insignificant one, or (b) *de novo*.

Clinical Recognition.—A malignant melanoma does not occur before puberty. It should be suspected after puberty when: (1) A previously existing mole continues to enlarge, bleeds, becomes ulcerated, becomes more deeply pigmented, or produces a halo of pigment in the surrounding skin. (2) A pigmented lesion appears in an adult and grows progressively. (3) A

¹ We are indebted for much of the material on this subject to Sir Stanford Cade, Consulting Surgeon, Westminster Hospital, London.

rapidly growing fleshy ulcerated skin tumour appears which looks as though it may be malignant. Some malignant melanomas are amelanotic, so that in these circumstances the tumour is more likely to be an *amelanotic melanoma* than anything else.

Unless adequately treated, a malignant melanoma carries a high mortality as these growths give rise to early and widespread dissemination. Exceptional cases have been reported in which spontaneous regression has occurred. Tumours arising in the skin permeate the lymphatics and intermediate deposits commonly occur between the primary growth and the regional nodes (fig. 106). At a later stage, bone, breast, and viscera are likely to

FIG. 106.—Melanoma malignum of the foot, with secondary deposits in the lymphatics of the thigh.



be involved. Secondary deposits are typically black, but sometimes contain little or no pigment (see *amelanotic melanoma*, above). Melanomata occurring in the choroid cause blurring of vision, and frequently give rise to visceral deposits, which may be enormous in size, particularly in the liver, and which may cause melanuria. After removal of an eye for melanoma a latent period of many years may elapse, but secondary deposits almost inevitably appear (p. 483).

It has been wrongly alleged that pregnancy encourages malignant change in a naevus, or stimulates malignant melanoma to increase activity. Pregnancy certainly increases pigmentation, but has *no* effect on melanocytic neoplasms. Neither is there any evidence that trauma or friction, e.g. on the sole of the foot, causes malignant change in a naevus.

Differential diagnosis is from: (a) Sclerosing angioma (p. 102); (b) Pigmented seborrhœic wart (p. 102); (c) Pigmented basal cell carcinoma; (d) Squamous celled papilloma.

Treatment.—Surgical excision is the only treatment which carries any hope of cure.

(1) If the clinical diagnosis is certain, treatment is by excision of a wide area of skin and subcutaneous fat with a minimum of 5 cm. clearance distal to the growth and 10 cm. clearance in the direction of the lymphatic flow.

(2) In lesions where the diagnosis is in doubt the tumour must be excised with 1 cm. of surrounding skin and submitted to histological examination (frozen section and routine paraffin section). *Immediately* the presence of a malignant melanoma is confirmed, the wide clearance is performed as above.

The excision of this magnitude creates a defect which must be closed by skin grafting (p. 114).

Subungual melanoma (fig. 104) require amputation, including the relevant metacarpal or metatarsal.

Small inadequate excisions in the casualty department must not be allowed. The application of caustics, or scraping the tumour, is almost certain to spread this terrible disease.

Excision of Lymph Nodes.—If the tumour is sufficiently adjacent to the lymphatic field, i.e. on the head or neck, or near the axilla or groin, a block dissection is performed in continuity. Excision of lymph nodes must be radical, e.g. the axilla is cleared in the same meticulous manner as for breast carcinoma, and a groin dissection not only includes the inguinal nodes, but also those in relation to the three iliac vessels.

If the primary tumour is remote from the lymphatic field it is dealt with as already described. Block dissection of regional nodes is not carried out as prophylaxis, and is only undertaken if, or when, the nodes become involved (fig. 107).

The administration of cytotoxic drugs (p. 631), has in some cases resulted in regression, or even disappearance of growths, and is well worthy of consideration.

Malignant melanoma arising in the choroid is treated by enucleation of the eye, and those occurring on the conjunctiva may be successfully treated by a radioactive strontium plate.

Prognosis.—The prognosis of malignant melanoma has for generations been gloomy, mainly on account of inadequate or delayed surgical treatment. Radical measures, combined with early diagnosis and immediate operation,

have so improved results that with vigorous treatment an overall five-year cure rate of fifty per cent. can be achieved and with early treatment the results are even better.

Kaposi's pigmented sarcoma occurs mainly in middle-aged Hebrews from Eastern Europe and Italy. Cases are also prevalent in the Burkitt Tumour zone of Africa (p. 158). Multiple bluish nodules occur, especially on the limbs. In due course the nodules ulcerate as sarcomatous changes develop. Fortunately these tumours are sensitive to radiotherapy and chemotherapy.

BURNS AND SCALDS

Burns are due to dry heat and scalds to moist heat. The management of a patient with such injuries depends upon the consideration of:

1. **Shock.**—The area of body surface involved is important in relationship to the development of shock. The surface area is estimated by the 'Rule of Nine' (fig. 108).

2. **Healing.**—The depth of burns determines the manner of healing. Only two grades need to be considered: (a) partial skin thickness burns (fig. 110) and (b) a full thickness burn.

(a) A partial thickness burn will heal from the epithelial elements in the deeper part of the skin (hair follicles, sebaceous glands, sweat glands).

(b) In full thickness burns all the epithelial elements have been destroyed

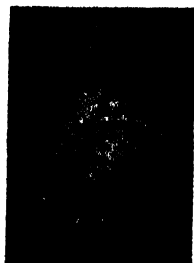


FIG. 107.—a
Malignant melanoma. Block dissection of lymph nodes in the groin.

and the dead skin will separate in two to three weeks revealing a red granulating surface. This can only heal from the edge, very slowly, and with the risk of contraction (fig. 109). This type of burn requires treatment by skin grafting (p. 114).

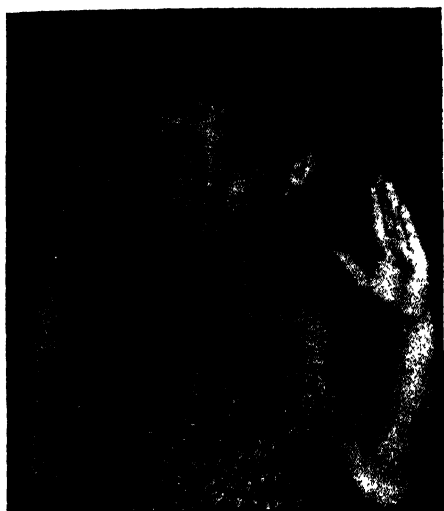


FIG. 109.—Contractures following burns (Children's Burns Unit, Guy's Hospital, London). (Patrick Clarkson, F.R.C.S.)

3. **Scarring.**—Partial thickness burns usually heal without much scarring. Alteration in pigmentation is quite commonly found. Maltreated and neglected full thickness burns always lead to unsightly, often incapacitating, scars. This may be minimised with properly timed skin grafting. In late contractures it may be necessary to excise the scar completely and replace this with a skin graft.

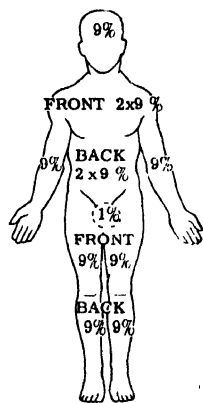


FIG. 108.—'Rule of nines' for estimating the extent of a burn. By adding the affected areas together the percentage of the total body surface burnt can be calculated quickly.

Treatment of Burns

The sections that follow deal with the treatment programme for burns shock, the local treatment of the wound, and the general treatment. The section on skin grafting follows thereafter.

Treatment of Burns Shock (A treatment programme)

(1) *Estimate percentage of surface burns.*—This is done using the 'Rule of Nine' (fig. 108).

(2) *Estimate weight (in kgms) from (a) known weight or (b) measured weight or (c) age and height tables.*

(3) *Transfusion with plasma.*—(a) All adults with burns over 15 per cent. (b) All children with burns over 10 per cent. (Some children with 5 to 10 per cent. surface burns need careful watching.)

(4) *Method of intravenous administration.*—In a shocked patient with collapsed veins, do not waste time with trying to put up a drip with needles—do a cut-down immediately. Avoid the use of veins in the legs if at all possible (use forearm, arm, or external jugular veins) (p. 90).

(5) *Volume of plasma transfused.*—The plan is to give six equal 'rations' of plasma in six periods. The volume is calculated by the following formula (after Muir and Barclay) (*Dextran* may be used in place of plasma). One

$$\text{'ration' of plasma (in ml.)} = \frac{\text{Total per cent. of burn} \times \text{weight (kgm.)}}{2}$$

Three rations are given in the first twelve hours, two rations are given in the second twelve hours, leaving the last ration for the third twelve hours.

An alternative method of calculating the plasma and fluid requirement is to use the Evans formula. In the first twenty-four hours 1 ml. of plasma and 1 ml. of normal saline is given for every kilo body weight and for each 1 per cent. of body burned, plus 2,000 ml. 5 per cent. dextrose in water. In the second twenty-four hours half the previous amount of plasma and normal saline is given, plus 2,000 ml. 5 per cent. dextrose in water. For burns of over 50 per cent. the need is calculated as for a 50 per cent. burn.

(6) *Progress and control*.—The need for transfusing and the rate of infusion are adjusted according to clinical and laboratory observations. These are: (i) Blood pressure and pulse rate; (ii) Restlessness; (iii) Hæmatocrit; (iv) Colour; (v) Urine volume; (vi) Nausea, vomiting, or gastric aspirate.

(7) *Blood transfusion*.—In deep burns (as opposed to superficial burns) red cells are destroyed. Blood is given instead of the plasma 'ration' in patients with deep burns of over 10 per cent. of the surface of the body. The second ration is replaced by blood in burns of 10 to 25 per cent., and the second and sixth ration in burns of 25 to 50 per cent.

(8) *Morphia*.—10 to 20 mg. is given intravenously or into the drip as indicated.

(9) *Urine*.—(a) Urine is collected hourly, measured and tested; (b) For burns over 35 per cent. all patients are catheterised.

(10) *Oral fluids*.—Water is required for metabolic fluid requirements. Start with 60 ml./hour and proceed to 100 ml./hour if there is no nausea. A nasogastric tube is usually passed in burns of 35 per cent. or over, and the stomach aspirated hourly before giving the oral fluid.

Local Treatment

The aims of local treatment are: 1. To prevent infection. 2. To obtain healing as quickly as possible so as to minimise deformity and loss of function.

In minor burns local treatment can be started immediately. If the patient has to be transfused, local treatment should be delayed until it is certain that the circulation is stable (the burn is covered with a sterile towel).

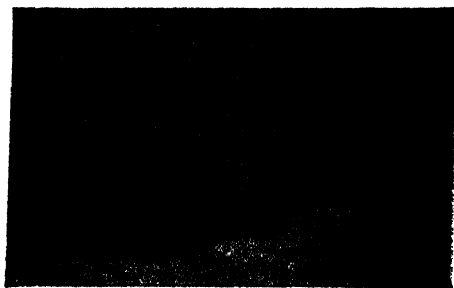


FIG. 110.—Partial thickness burns.
(I. F. K. Muir, F.R.C.S., London.)

(a) *Exposure treatment*: The burn is cleaned with cetramide-chlorhexidine solution, loose skin is removed, and blisters are pricked. The area is then exposed to the air (fig. 110) and a crust is allowed to form which protects the burn and permits healing to take place. This method is suitable for burns of the head and neck, and burns of a single surface of trunk or limbs. As the burn heals the crust lifts up at the edge and can then be clipped away.

(b) *Dressings*: The burn is cleaned as above and then dressed. The dressings are applied in three layers:

(i) **First layer.**—The layer next to the skin should be non-adherent and 'antiseptic'. The reagents suitable are gentamycin, bacitracin, framycetin, dinitrofurazone, and chlorhexidine. These may be used alone or in combination. They may be applied in a wide mesh gauze already impregnated with the reagent, e.g. sofratulle, or the reagent may be sprayed on, e.g. polybactrin spray, and a layer of paraffin tulle gauze applied.

(ii) The second layer of cotton gauze.

(iii) An outer layer of absorbent wool is applied and held in place with gauze or crepe. This should be sufficiently thick to absorb all exudate without penetration to the surface and yet be porous. Should the wool become saturated, a further layer must then be applied without removing the original dressing. This dressing should be changed on the third and tenth days.

Specific Local Treatment.—(1) *Partial thickness burns:* The treatment should be continued as above until the burn is healed.

(2) *Full thickness burn:* If a full thickness burn is present, the area of dead skin will be obvious as a black slough in two to three weeks. Treatment should therefore be changed to a method which will help separation of the slough. This is achieved by baths and frequent dressings with hypochlorite solutions, e.g. Eusol.¹ As soon as the slough has separated and clean granulations are present, thin split skin grafts cut from the undamaged areas should be applied to expedite healing.

(3) *Special types of burns:* Some well localised deep burns are best treated by early excision and grafting. Electrical burns (fig. 111) are localised and often very deep, and may need complicated methods of repair.

General Treatment

The medical and nursing staff should take every precaution to prevent infection of a burn. It is desirable that the patient should be isolated (in a cubicle if possible), and barrier nursing instituted to prevent cross-infection.

(a) Respiratory damage may occur due to inhalation of smoke and hot gases and this may require oxygen and/or a tracheostomy.

(b) Sedation to reduce pain and restlessness—morphia 10 to 20 mg. may be given intravenously, usually via the drip. In children—Nepenthe 1 minim (0.06 ml.), or morphia 0.5 mg. for each year of age.

(c) Diet: Large amounts of protein are necessary to stimulate healing and make good protein losses due to catabolism.

(d) Antibiotics: Systemic antibiotics should be given as the clinical condition demands and in accordance with bacterial cultures.

(e) Late blood transfusion: Anæmia commonly develops and blood transfusion must be given if the Hb. falls below 80 per cent.

The Cold Water Treatment of Burns.—In some countries this is a recognised first-aid treatment, and it is specially useful for burns of the hands, arms, and legs.



FIG. 111.—A post-mortem photograph of an electric burn. The deceased touched a live terminal.

¹ Eusol = Edinburgh University SOLUTION. It must be freshly prepared.

The part is immediately immersed in cold tap water (about 15° C.). Pain and exudation are reduced to a remarkable extent, and mortality is lessened. The chief disadvantage may be the increased risk of bacterial contamination, but, intelligently applied, this method is of special value in areas where medical and special burns-treatment facilities are sparse.

SKIN GRAFTING

Skin grafting, by free grafts or pedicled flap, is necessary to obtain healing and limit deformity and disability when a large area has been lost by injury (e.g. burn), by disease (e.g. varicose ulcer), or by surgical excision of a neoplasm (e.g. malignant melanoma).

Free Skin Grafts.—These grafts provide the simplest method of restoring skin cover. They can be used when the raw surface is composed of healthy vascularised tissue (example: subcutaneous tissue, deep fascia, paratenon), or healthy granulation. Ischæmia and infection prevent grafts from surviving.

1. *Split thickness grafts* are the most generally useful grafts. Though often suitable for permanent repair they have a tendency to shrink. The grafts originally popularised by Thiersch were very thin, but the grafts now commonly used consist of one-third to two-thirds of the thickness of the skin. They are usually taken from the thigh by means of a special knife (Blair or Humby pattern, fig. 112 and fig. 113). If a large area has to be covered

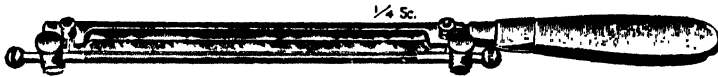


FIG. 112.—Humby's dermatome.

(e.g. an extensive burn), the electric dermatome is useful. The Padgett dermatome, which necessitates the use of an adhesive, can be used to cut grafts from the abdomen or back. The graft may be affixed by sutures.

Alternatively pieces of 'postage stamp' size are applied—the epithelium grows out from them to cover the intervening bare areas. The graft may be spread on paraffin tulle gras and laid or sutured on the area to be grafted. Light pressure is then exerted by either wool soaked in flavine emulsion or a piece of sterilised sponge covered by a crepe bandage. The donor site of the split skin graft is covered with a single layer of tulle gras and

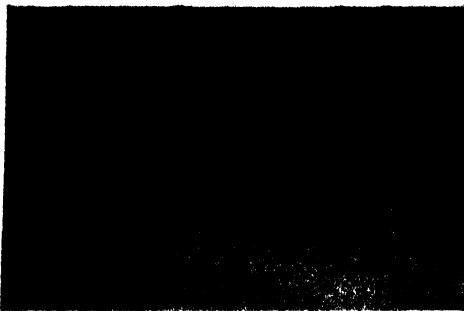


FIG. 113.—A split skin graft, cut with a Blair knife. (I. F. K. Muir, F.R.C.S. London.)

heals in two weeks by epithelialisation from the remaining deeper parts of the skin.

2. *Full Thickness Skin Graft* (Wolfe).—This graft consists of the full thickness of the skin to which *no* fat should be adherent. The criteria for the graft to

Karl Thiersch, 1882–1895. Professor of Surgery, Leipzig.
John Reisberg Wolfe, 1824–1904. An Austrian ophthalmologist who came to Scotland. In 1873 he described how to repair an eyelid by a post-auricular graft.

take are more strict than in the case of the split skin graft but the Wolfe graft shows no tendency to contract, and is of better colour and texture than the split skin graft. It is therefore useful for defects of the face (example: when excising a rodent ulcer from a lower eyelid), when the graft may be taken from the posterior surface of the ear.

Pedicle Flaps.—Free grafts will not ‘take’ on (i) cartilage, open joints, bare cortical bone, or bare tendon, (ii) if the vitality of the recipient area has

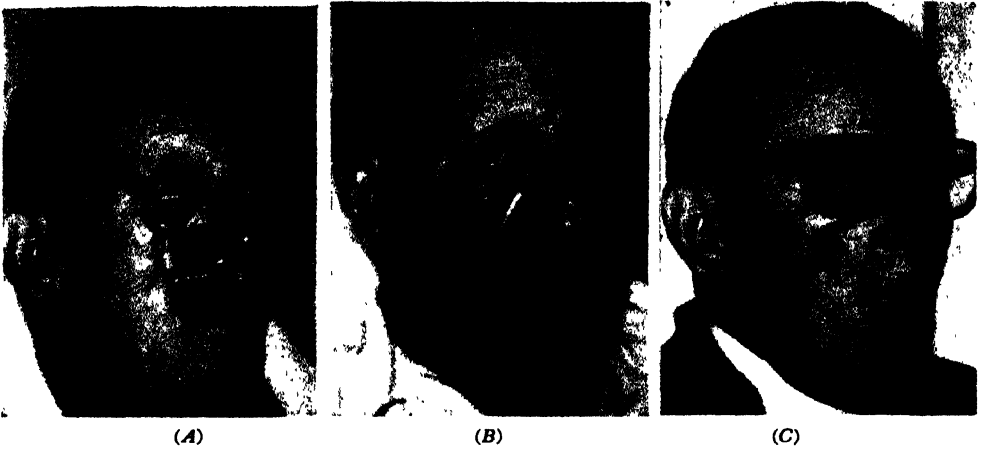


FIG. 114.—A large rodent ulcer involving the upper lip, the side of the nose, and the antrum has been excised leaving the facial deficiency (A), in which post-operative biopsies show no residual growth. The upper lip and the side of the face are reconstructed by a flap graft (B), and the residual deficiency made good by a prosthesis, (C), which is held in place by a pair of spectacles. (J. F. K. Muir, F.R.C.S., London.)

been depressed by scarring or irradiation, (iii) if the defect involves a cavity (example : full thickness excision of lip or cheek (fig. 114)). Under any of these circumstances the repair must be by means of a flap of skin with some subcutaneous tissue, which remains attached to the body by one of its margins through which it retains a blood supply (i.e. by a pedicle or stalk) until it has united in its new position. Once it has attained its own blood supply the original attachment can be severed and the flap adjusted.

(a) *Local flaps*: These are turned on their own blood supply (fig. 114).

(b) *Direct flaps*: If the donor site can be brought into approximation with the recipient area, the flap can be transferred at one stage and the division completed at a second stage. Examples of this are from one finger to another (cross finger flap), from the calf of one leg to cover a defect on the opposite shin (cross leg flap, fig. 115), or from abdomen to hand.

(c) *Indirect flap*: In some circumstances it may be impossible to bring the donor area and the recipient area



FIG. 115.—A cross leg flap. The legs are held together by a plaster cast.

into proximity and the flap must first be attached to an 'intermediate carrier site', thus tissue can be moved from the abdomen to the face or to the lower leg, using the wrist as the 'carrier'. In order to eliminate raw areas it is convenient to stitch the flap into the form of a tube, i.e. a tube pedicle flap (fig. 116).



FIG. 116.—A tube pedicle flap. The skin tube was raised on the lateral abdominal wall. In the course of months the wrist was used as the intermediate source of its blood supply before final separation from the abdomen and application to the scarred and ulcerated leg.

continues to grow for many months and even years. In appearance a keloid is smooth, sometimes lobulated, or even claw-like, and it may invade the subcutaneous tissues. It has a predilection for the skin over the sternum and the shoulder. A keloid never becomes malignant and though it may persist for years the tendency is for it to flatten and disappear gradually. *Treatment* is difficult, as simple excision is invariably followed by recurrence. Excision with pre- and post-operative radiotherapy may be successful, but the result is unpredictable.

Keloid - like a claw.

Keloid¹ (fig. 117).—This condition occurs in scars, and although the cause is unknown it behaves in some ways like a tumour. The condition is common in black-skinned people and relatively uncommon in white people, though it may be seen in the nordic type and the red-heads. Instead of settling down after a few months, as most scars do, the keloid scar con-



FIG. 117.—Extensive keloid in a West African. (C. Bowesman, F.R.C.S., Kumasi, Ghana.)

CHAPTER 8

ARTERIES AND VEINS

ARTERIAL SURGERY—BASIC CONSIDERATIONS AND PRINCIPLES

THE first section of this chapter deals with those aspects which can be regarded as the essentials of arterial surgery.

COLLATERAL CIRCULATION

When the arterial blood supply to any part of the body is interrupted, necrosis or gangrene will occur unless there is an alternative pathway to by-pass the obstruction, that is, a collateral circulation (fig. 118). The formation of a collateral circulation depends primarily upon the anatomical arrangement of the arteries concerned. If an 'end artery' is obstructed (e.g. a coronary artery), no collateral circulation is possible and necrosis occurs. In obstruction of the aorta (e.g. coarctation, Chap. 30) or the limb arteries, good collaterals are possible. The factors which aid the establishment of an adequate collateral blood flow include freedom from atherosclerosis, tissues containing a rich vascular network, subjacent muscles, and normal cardiac function. Adverse factors include atherosclerosis, embolism with extension-thrombosis (p. 130), joints (e.g. knee joint) which are not covered by muscle, and heart failure.

Time is necessary for collateral vessels to open up fully, and while some degree of by-pass is effected immediately, it takes many days and even weeks to restore a blood supply comparable to that which originally entered the part via the main artery.¹

The measures which are taken to aid the establishment of a collateral circulation are: (i) The prevention of thrombosis occurring or extending throughout the arterial network (by using anticoagulants, p. 131), and (ii) the use of 'vasodilator methods' (p. 122). (iii) The treatment of heart failure or atrial fibrillation, if present.



FIG. 118.—Collateral circulation.

ISCHAEMIC PAIN

Intermittent Claudication.—Ischaemia of muscles causes a cramp or vice-like pain, which occurs during exercise. While the patient with coronary

¹ John Hunter (1728–1793), Surgeon, St. George's Hospital, and founder of the Hunterian Museum of the Royal College of Surgeons, was the first to demonstrate the formation of collateral circulation. He tied the external carotid artery of a stag from Richmond Park. The stag's antler fell off, but later another one grew. Hunter reopened the original wound, and found that while his ligature was intact, a collateral circulation had been established.

artery disease may experience the vice-like pain of angina pectoris on exertion, the person with an obstructed femoral or popliteal artery suffers from intermittent claudication.¹ The pain is felt in the calf after walking a limited distance (claudication distance), and it becomes so severe as to bring the patient (usually a man) to a halt. But in a minute or two, perhaps while he looks in a shop window, it fades away, and he is able to proceed for a similar distance before being forced to stop again. The claudication distance varies according to the site and extent of the arterial obstruction, the efficiency of the collateral circulation, the general condition of the patient (heart failure, anæmia), the speed of walking, the presence of an incline or stairs, and a head-on wind. In some patients the collateral circulation is sufficient to enable the sufferer to walk slowly for an indefinite distance.

Claudication affecting the thigh (*angina cruris*) and the buttocks, implies that either the aorta or the iliac arteries are occluded. *The differential diagnosis between arterial and orthopædic disease* in such a case is sometimes difficult, and frequently the two conditions are found to coexist.

Rest Pain.—In arterial occlusion affecting the lower limb, the pain is severe, affecting the forefoot and toes, and is first noticed at night. It interrupts or prevents sleep, though some relief may be obtained by exposing the foot to the cold air and by hanging it over the side of the bed. When more severe, the patient spends the night sitting in a chair or walking about. Later still (when the foot is in the pregangrenous stage or there is overt gangrene), the pain is present all day. It is relentless, and, since it cannot be completely relieved by any kind of analgesic, the patient becomes distracted by it.

In acute ischæmia a severe cramp affects the whole limb. In embolism and acute arterial thrombosis this is an important presenting symptom which must not be ignored (fig. 36).

GANGRENE AND ULCERATION

The pathological and clinical features, and the management of gangrene are discussed in Chapter 3. The ischæmic nature of some ulcers is also referred to (p. 36).

INVESTIGATIONS

Clinical Examination (General).—The general examination of a case of arterial disease needs to be comprehensive, and it should be supported by the appropriate investigations (X-ray, E.C.G., blood count, etc.). Patients with atherosclerosis may have general disease, and previously have suffered other cardiovascular episodes (e.g. coronary thrombosis, cerebral thrombosis). Many suffer from bronchitis, and are in the age-group likely to have carcinoma of the bronchus. Others may have peptic ulceration, prostatic enlargement, and depressed renal function. Diabetes and anæmia should always be excluded.

Clinical Examination (Local).—The local examination of the part affected includes taking note of the following:

¹ Claudius I (10 B.C.—A.D. 54), Emperor of Rome during the life of Christ, had a permanent limp (? due to poliomyelitis). His courtiers, walking behind him, used to imitate his limp. Claudus (L) = Lame.

(i) *Appearance and Temperature*.—The colour of an acutely ischæmic limb varies from a dead white to a mottled purple. In the pregangrenous state of atherosclerotic obstruction of the lower limb, the foot and toes may be red and swollen, and the skin peppered with tiny red areas of extravasated erythrocytes. Alternatively, the foot will appear withered. Gangrene is usually obvious, but it is necessary to look carefully for other sites ('skip' areas, p. 40), especially under the heel.

The acutely ischæmic limb feels quite cold. In other types where there is unilateral disease, the affected limb feels colder than the sound one. In the reddened pregangrenous state described above, the foot, paradoxically, may feel warm, even without superadded infection.

(ii) *Sensation and Movement*.—An ischæmic limb is examined for loss of sensation to 'touch', and the level to which this loss extends upwards. In acute ischæmia and pregangrene, gentle handling is essential, as those areas of skin on the borderline of ischæmia are hyperæsthetic (p. 40). Paresis of muscle groups is a special feature of the acutely ischæmic limb, those commonly affected being the dorsiflexors of the foot, and the flexors of the wrist and fingers (p. 186).

(iii) *Arterial Pulsation and Auscultation*.—Normal, diminished, absent, or abnormal expansile pulsation is to be elicited along the course of the main artery to the limb. For disease affecting the leg, this means palpating the aorta in the abdomen; the femoral artery in the groin; the popliteal artery behind the knee; the posterior tibial artery behind the medial malleolus; and the dorsalis pedis artery on the dorsum of the tarsus. An abnormal expansile pulsation of a mass in the line of an artery is an intrinsic sign of an aneurysm (p. 125). A pulsation which imparts a thrill may be due to (a) a partial obstruction, (b) an aneurysm, or (c) an arteriovenous fistula.

Auscultation over the course of an artery is another aid in diagnosis. A soft systolic murmur is heard over a stenosis. It is usual to listen over the carotid arteries in all cases of atherosclerosis to exclude carotid stenosis. A bruit heard over the renal arteries may point to renal artery stenosis (fig. 119) in cases of hypertension (Rob). A systolic murmur may be heard over an aneurysm, but in an arteriovenous fistula a continuous machinery murmur is characteristic.



FIG. 119.—Renal artery stenosis. Arteriogram by retrograde femoral catheterisation.

(iv) *Venous Refilling*.—At first sight, the veins of an ischæmic limb may appear to be adequately filled. The limb should be elevated for thirty seconds and then laid flat on the bed. In this way the speed of the venous flow will then be apparent. Normal refilling occurs within a few seconds. Harvey's

sign¹: If the two index fingers are placed firmly side by side on a vein, and the finger nearer the heart is moved so as to empty a short length of vein, the release of the distal finger will allow the speed of venous refilling to be observed.

Increased venous return is observed in veins leading from an arterio-venous fistula (p. 128). The veins in such a case may be varicose.

Plain X-ray exposures will show the presence and extent of arterial calcification. Flecks of calcium may outline an aneurysm. Plain X-ray films, *per se*, are generally of limited value in helping the surgeon to formulate his opinion and treatment policy. Arteriography is usually necessary.

Arteriography.—This procedure furnishes information concerning the course and size of arteries, constrictions, obstructions, or dilatations, and the state of a collateral circulation (if present). The vascular pattern of tumours

may also be studied. Hypaque 45 to 65 per cent. solution (sodium diatrizoate or diatrizoate compounds) is a satisfactory contrast medium. The following methods are employed:

(i) *Retrograde Percutaneous Catheterisation.*—Under a local or general anæsthetic, and with operating theatre aseptic ritual, a special needle and cannula (fig. 120) are inserted into the artery. A suitable length of flexible guide wire is threaded through the cannula, which is withdrawn so that a polythene catheter can be passed over the guide wire and into the vessel.

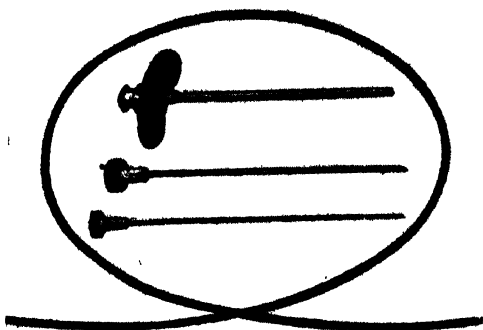


FIG. 120.—Seldinger needle and guide wire for introducing an arterial catheter.

The common femoral artery is used for aortic, renal, mesenteric, iliac, and femoropopliteal arteriograms, while the brachial artery is employed for subclavian, innominate, thoracic aorta, and vertebral arteriograms.

(ii) *Direct Percutaneous Arterial Puncture.*—This may be used for carotid arteriograms, and for demonstrating the abdominal aorta (translumbar route) when the femoral arteries are occluded, or if the retrograde method cannot produce the necessary information (fig. 121).

A trial or test injection of 5 to 10 ml. of 45 per cent. Hypaque must be made in order (a) to exclude iodine sensitivity (Chap. 35) (cf. intravenous cholangiography) and (b) to ascertain that the tip of the catheter or the point of the needle is in the correct place. A dissecting aneurysm can occur if the bevel of the needle is partly in the wall of the artery. To prevent this hazard a special needle without a bevel but with a side hole may be employed.



FIG. 121.—Translumbar aortography. Atherosclerotic stenosis of both common iliac arteries prevented proper visualisation by retrograde catheterisation.

¹ William Harvey (1578–1657), Surgeon anatomist to the College of Physicians; Physician, St. Bartholomew's Hospital, London, discovered the circulation of the blood. The sign of venous refilling should be eponymous, as it was the observation on the direction of blood flow in the veins that clinched Harvey's discovery.

If the result of the test injection is satisfactory, the whole volume (30 ml.) of the contrast medium is injected rapidly as a 'bolus', and serial X-ray exposures are made to obtain radiographs of the length of the arterial tree under examination. In the case of the leg, the examination must extend from the lower part of the abdominal aorta to the ankle, as a second obstruction may be present.

OPERATIONS ON ARTERIES

Direct arterial surgery is used either to restore the continuity of blood flow, or to provide an alternative channel (by-pass).

Arterial Repair.—For closure of a simple wound or arteriotomy for embolectomy, a continuous suture is employed, using 0000 silk or polypropylene

on a 16 to 20 mm. atraumatic needle. A patch of vein is frequently used for repairs in order to prevent narrowing the lumen of the vessel (fig. 122).

Embolectomy and thrombectomy are described on p. 131.

Thrombo-endarterectomy ('disobliteration' or 'rebore') is described on p. 136.

Artery Grafting (figs. 123 and 124).—*Veins* are commonly used for by-pass grafting peripheral vessels. The internal saphenous vein is usefully placed for by-passing a femoro-popliteal obstruction. *Synthetic grafts* of knitted or woven Dacron or Teflon are used mainly for aorto-iliac replacement (fig. 136). They are less suitable for peripheral work, as those of small calibre tend to thrombose more easily than vein grafts. As they are crimped in concertina fashion, they require to be stretched before insertion. Very little blood oozes

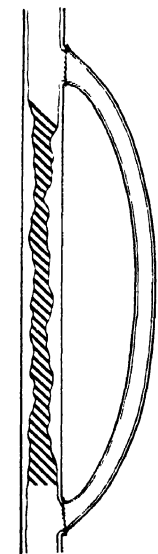


FIG. 123. — By-pass grafting.

through the interstices of the woven variety. After insertion, these grafts are incorporated by an invasion of fibroblasts, and within the lumen they become lined with endothelial cells which are believed to originate from the cellular component of the blood.

Homografts, which are arteries taken from cadavers, may also be used, but they are not as popular as formerly, owing to the technical difficulties in collection, sterilisation, and preservation (deep freeze or freeze-drying) as well as their propensity to undergo aneurysmal dilation after a few years.

Indirect arterial surgery, namely **sympathectomy**, is employed (a) for the treatment of vasospastic conditions (p. 139), (b) for Buerger's disease and other cases of peripheral arteritis (p. 137), and (c) for 'want of better' in severe degrees of atherosclerosis where direct measures are contraindicated (p. 136). In (b), and particularly in (c), *sympathectomy* is *capricious* in its effects. The techniques are described on p. 140.

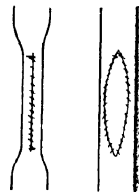


FIG. 122. — Where arterial suturing may reduce the lumen, a vein patch (right) is useful.



FIG. 124. — End of graft widened and sutured as a 'sucker foot' to the side of the parent artery.

CONSERVATIVE MEASURES

No Smoking.—Cigarette smoking should be strongly discouraged in patients with intermittent claudication due to atherosclerosis, Buerger's disease, and peripheral arteritis. Buerger's disease has yet to be reported in a non-smoker (Eastcott). The mechanism, whereby smoking causes small vessel contraction and thrombosis, is not clearly understood.

Vasodilator Methods.—While it is doubtful if the collateral arteries in muscle groups can be influenced by vasodilator drugs, it is certain that a dilatation of the 'run-off' or 'outflow' of the peripheral arterial bed distal to an obstruction can be beneficial, particularly in preserving the trophic state of the skin, and so minimising or preventing gangrene. The measures which are adopted include:

(i) *Buerger's Position* (fig. 125).—The head of the bed is raised six inches (15 cm.) (all day, or for periods of one to two hours). The hydrostatic congestion should cause some vasodilatation.

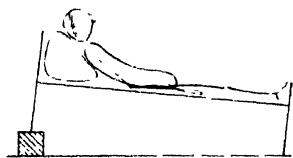


FIG. 125.—Buerger's position.

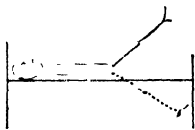


FIG. 126.—Buerger's exercises.

(ii) *Buerger's Exercises* (fig. 126).—The leg is elevated for two minutes, and lowered beside the bed for two minutes. Elevation and lowering are repeated

for séances of about thirty minutes, twice or thrice daily. Elevation can be readily obtained by putting a chair on the bed (fig. 164).

(iii) *Reflex Heating.*—The hands and trunk are warmed by an electrically heated pad. This causes reflex vasodilatation of the leg and foot in certain patients.

(iv) *Vasodilator Drugs.*—Alcohol by mouth (such as sherry, whisky, or brandy) is the most effective. A quick-acting barbiturate (e.g. quinal barbitone 100 to 200 mg.) which normally has an associated vasodilator effect, is useful to induce sleep. The legion of proprietary vasodilator drugs is useful in only a few cases.

(v) *Paravertebral Injections of the Sympathetic Chain.*—For the leg this implies the injection of 20–30 ml., 1 per cent. lignocaine beside the bodies of the second, third, and fourth lumbar verte-

bræ. Fig. 127 shows how to position the patient (a lateral lumbar-puncture position is used for patients who cannot sit up). Fig. 128 shows how a long (15 cm.) needle is inserted, firstly to seek the side of the vertebral body and,

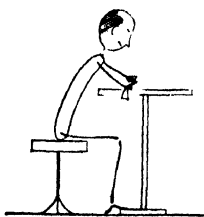


FIG. 127.—Sitting position for a paravertebral injection. The patient rests his head and arms on a pillow.

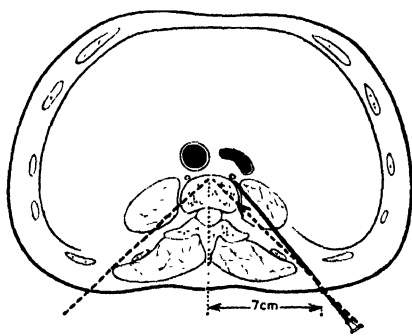


FIG. 128.—Lumbar paravertebral injection.

secondly, to pass alongside it. The effect of the injection (warmer feet and improved venous filling) may last for two or three days. Further injections may be given, or a permanent effect will more easily accrue if 5 to 10 ml. of 5 per cent. phenol solution in water is injected (chemical sympathectomy). Care is needed to avoid penetrating the aorta or the vena cava. Paravertebral injections are not given if anticoagulants are being prescribed.

For the arm a shorter needle is introduced vertically to the skin, 4 cm. lateral to the spines of the second and third and fourth thoracic vertebræ. The needle seeks the back ends of the ribs and is then passed over or under them for another 1 to 2 cm. while the bolus of lignocaine is injected. Special care is needed to avoid puncturing the lung and thereby causing a pneumothorax.

(vi) *Intra-arterial injection* of proprietary vasodilator drugs, papaverine, and oxygen have been fashionable—a painful procedure of temporary and doubtful value.¹

Protection of Ischæmic Parts.—In the severely ischæmic limb, steps must be taken to prevent gangrene occurring in the areas likely to be subject to pressure (e.g. the heel, p. 41). All cases of peripheral vascular disease need careful chiropody, so often does gangrene follow the trauma of a badly cut toe nail.

Exposure of Ischæmic Parts to the ambient temperature of the ward reduces the tissue metabolism, the demand for oxygen, and may therefore reduce pain. Heating or freezing an ischæmic limb should never be employed.

Reduction of Muscular Activity.—Many patients who have intermittent claudication are able to live with their disability by restricting their muscular activity. Their walking distance is kept to within their claudication distance. However, by walking at a slower pace than usual, this distance may be greatly increased. Many find that by gradually increasing their distance they obtain improvement. Raising the heels of the shoes by a quarter to half an inch (6–12 mm.) may also be effective, since it diminishes the amount of work done by the calf muscles. Division of the tendo calcaneus (Achilles tenotomy) is a minor operative procedure which has the same effect, converting a painful walk into a painless shuffle.

Relief of Pain.—Ischæmic pain² which is unrelieved by surgical and conservative vasodilator measures is liable to be intractable. Reliance should first be placed on drugs such as dihydrocodeine (DF 118) 30 mg. Except for short-term use, morphine and pethidine should be avoided. Pethidine is notoriously addictive in these cases.

Anticoagulant therapy (p. 131.) may be employed in this conservative regimen in order to prevent subsequent thrombotic episodes which will worsen the condition of the limb.

DISEASES OF ARTERIES

INJURY

Injuries to arteries usually take the form of division, rupture, or contusion.

Division occurs in penetrating wounds. A classical example is 'butcher's thigh', which is a penetrating wound in the femoral triangle, due to the knife slipping while boning meat. Any of the femoral arteries may be severed, sometimes with an immediate fatal result. *Rupture* of an artery can be

¹ The reader is referred to the book *Annapurna*, by Maurice Herzog (*Jonathan Cape, Ltd.*), who experienced intra-arterial and paravertebral injections for the treatment of frostbite.

² Quinine bisulphate, 200 mg. may help some patients with claudication.

A *true aneurysm* is due to dilatation of an artery, whereas a *false aneurysm* is a sac lined by condensed cellular tissue which communicates with the artery through an opening in its wall.

A *fusiform aneurysm* (the commonest type), is one in which the lumen is more or less equally expanded. The artery wall is usually weakened by severe atheroma and hypertension. In syphilis, the media of the arch of the aorta may be weakened by endarteritis of the vasa vasorum (fig. 131). In a *mycotic¹ aneurysm*, the artery wall is weakened by infection, either in the form of a local abscess, or because an infected embolus has come to rest at that point. Such an embolus originates from the vegetations on the valves of the heart in acute bacterial endocarditis, in which a pyogenic staphylococcus infection is superadded to the commensal streptococcus viridans of subacute endocarditis.

A *saccular aneurysm* is due to stretching of part of the arterial wall and commonly follows injury to superficial arteries (figs. 130 and 132).

Clinical Features.—(a) *Intrinsic*.—

A swelling exhibiting expansile pulsation is present in the course of an artery. The pulsation diminishes if proximal pressure can be applied, and the sac itself

is compressible, filling again in two or three beats if proximal pressure is released. A thrill may be palpable, and auscultation sometimes reveals a bruit.

(b) *Extrinsic*.—Neighbouring or distal structures are affected. Thus pressure on veins

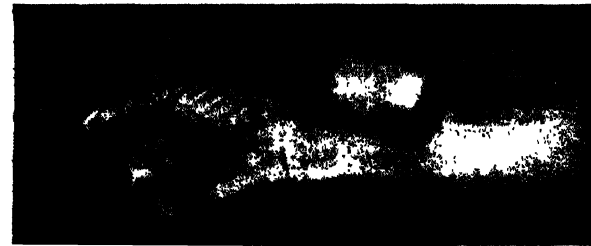


FIG. 132.—Saccular aneurysm of the radial artery.
(Professor A. K. Toufeeq, F.R.C.S., Lahore, Pakistan.)

or nerves causes distal œdema or altered sensation, and the pulse is delayed or smaller in volume. Bones, joints, or tubes (such as the trachea or œsophagus), are sometimes affected, but structures which are resilient, such as the intervertebral discs, often withstand prolonged pressure.

Differential Diagnosis.—1. *Swelling Under an Artery*.—An artery may be pushed forwards, e.g. the subclavian by a cervical rib, and thus rendered prominent. Careful palpation distinguishes this condition.



FIG. 131.—Aortic aneurysm which has eroded the sternum.

¹ 'Mycotic' is a misnomer, as the disease is not due to a fungus but to bacterial infection.

2. *Swelling Over an Artery*.—In this case transmitted pulsation is liable to be mistaken for that caused by expansion. However, proximal pressure does not reduce the size of the swelling, and posture may diminish pulsation; thus a pancreatic cyst examined in the genupectoral position falls away from the aorta, and consequently pulsation is less definite.

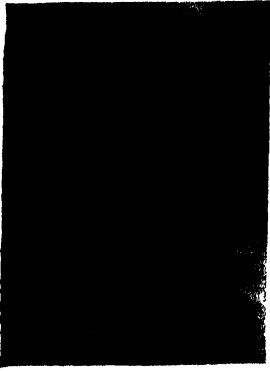


FIG. 133.—Serpentine abdominal aorta.

3. *Pulsating tumours*, such as bone sarcoma, osteoclastoma, and a metastasis, especially from a hypernephroma.

4. *An Abscess*.—Before making an incision into a swelling believed to be an abscess, especially of the chest wall or popliteal fossa, it is essential to make sure that it does not pulsate! This is a veritable *pons asinorum*.

5. *A Serpentine Artery* (fig. 133).—In addition to being a diagnostic problem in the abdomen, this condition is often confused with aneurysm of the innominate artery.

6. *Other Causes of Deep-seated Pain*.—Cases of persistent and severe back-ache are occasionally due to aneurysm of the aorta.

Natural Terminations.—1. *Spontaneous Cure* (Consolidation).—This sometimes occurs in cases of saccular aneurysm, due to gradual clotting in the sac (fig. 134).

2. *Extension and Rupture*.—This occurs either slowly as a leakage, or suddenly, in which case death may follow in a few moments if a large vessel is involved.

3. *Infection*.—Usually arises from organisms in the blood-stream. Signs of inflammation supervene and, if untreated, suppuration and rupture follow.

Treatment.—This is surgical. The scope of the operation required may be indicated by arteriography, which will show the state of the arteries above and below the lesion. Due to the presence of mural laminated blood-clot, the aneurysmal dilatation is seldom visualised. Fig. 135 is designed to itemise the procedures which are available:—

Ligation and Excision.—Aneurysms of secondary arteries are treated by ligation of the proximal and distal vessels. Excision is not obligatory, save in special anatomical or physiological circumstances (fig. 130).

Hunterian Ligation (fig. 135A).—This operation was designed by Hunter for the treatment of popliteal aneurysm, but the principle can be applied to the treatment of other limb aneurysms, when a simple and quick measure is needed.

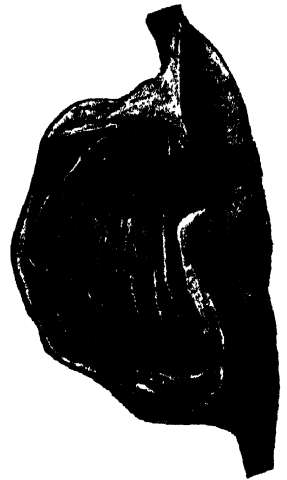


FIG. 134.—Aneurysm of the aorta. Extensive clotting in the sac has almost resulted in spontaneous healing. (R.G.S. Museum.)

The ligature must be placed above the main collateral nearest the aneurysm (e.g. above the descending geniculate artery). In this way the blood flow through the aneurysm is not immediately cut off. The collateral circulation is given time to open up before consolidation occurs.

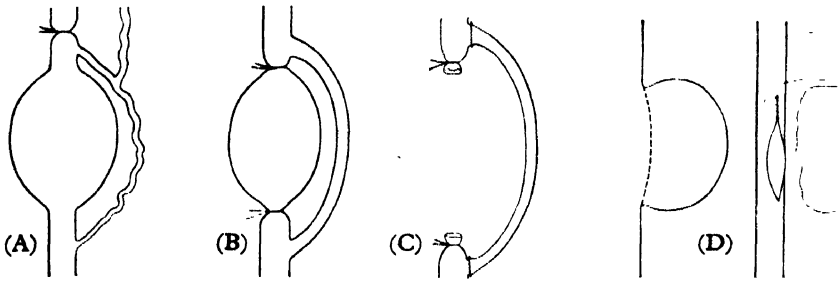


FIG. 135.—Operations for aneurysm.

- (A) Hunterian ligation. (C) Excision and by-pass graft.
(B) Exclusion and by-pass graft. (D) Reconstruction (Matas).

Exclusion and By-pass Grafting (fig. 135B).—Excision is not always necessary, in fact it is usually undesirable, as neighbouring vital structures may be damaged (e.g. the vena cava in abdominal aneurysm). The excluded aneurysm thromboses, and rapidly shrinks into a fibrous mass, containing porridge-like atheroma and blood-clot in the centre.

Excision and artery grafting (fig. 135C) is the alternative if excision should be desirable. The graft can be inserted 'end to end' or 'end to side'.

Excision of the sac and arterial reconstruction (Matas' reconstructive aneurysmorrhaphy) (fig. 135D), is a very useful procedure for small saccular aneurysms of important arteries (e.g. femoral or popliteal).

Abdominal aortic aneurysms are often amenable to surgery. The patient complains of abdominal or back pains, and may be aware of the pulsatile abdominal mass. Left alone, the average expectation of life before rupture is about three years. When the aneurysm ruptures, the patient presents with abdominal pain and hæmorrhagic shock. The diagnosis may be difficult, as the clinical features mimic some acute abdominal emergency (e.g. perforated diverticulitis, acute pancreatitis, etc.). Emergency surgery has a mortality of 80 per cent., due usually to post-operative renal and hepatic failure. Modern intensive care wards have improved the prospects. Elective surgery, with a mortality of 10 per cent., is therefore advised in otherwise fit patients, and when the aneurysm is believed to lie below the level of the renal arteries (usually the case).

Exposure is through a long paramedian, a curved transverse, or a flank incision, the latter using an extraperitoneal approach. The aorta is curiously amenable to encirclement with the finger and the application of a clamp just below the renal arteries. The aneurysm may simply be excluded or evacuated and obliterated by suturing. A Dacron finely knitted graft is

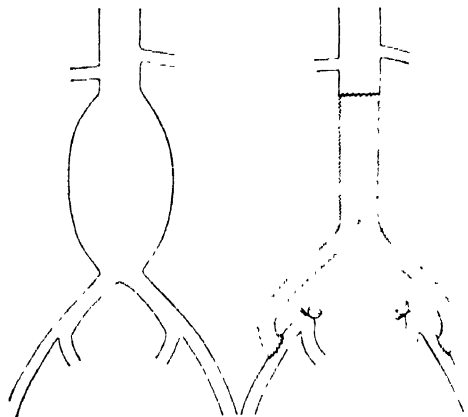


FIG. 136.—Grafting for an abdominal aneurysm. The excluded or obliterated aneurysm is omitted for the sake of clarity.

inserted (fig. 136). Sometimes the aneurysm is opened and evacuated, and the walls are wrapped round the graft.

Dissecting aneurysms (fig. 137A) occur in the aorta and its large branches, and are due to the separation of an atheromatous plaque, which allows blood to insinuate

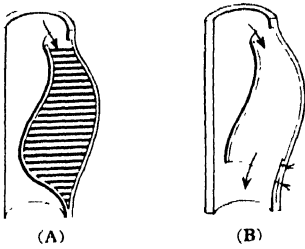


FIG. 137.—Dissecting aneurysm. (A) The dissection may obstruct the lumen. (B) The re-entry operation.

itself between the inner and outer layers of the media. Rupture, or obstruction of the true arterial lumen, may occur. Thus, when extending, say, into the iliac vessels, it mimics an embolus or thrombosis. Resection and grafting is suitable for a localised aneurysm, otherwise the artery is incised, and an opening is constructed so that blood can flow freely from the false into the true lumen (fig. 137B).

False aneurysms are traumatic in origin, and the extravasated blood is enclosed in a sac of condensed cellular tissue.

Fig. 138 is a typical example, and the caption recounts the method of repair.

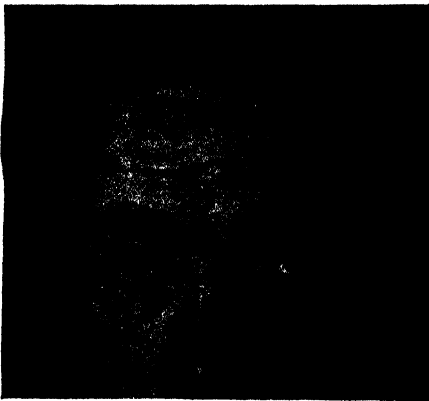


FIG. 139.—Arteriovenous fistula of inferior orbital vessels, caused by a cricket-ball.

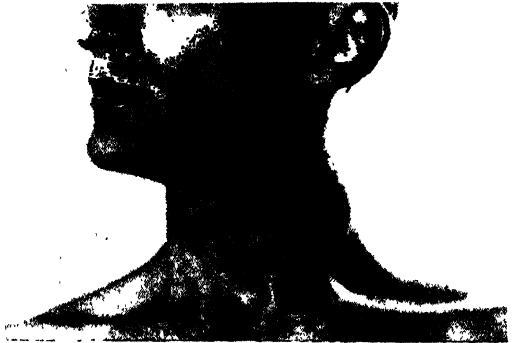


FIG. 138.—A false, and rapidly extending aneurysm of the common carotid caused by a stab wound. Note (above) the ptosis, myosis, and enophthalmos of a Horner's syndrome, due to concomitant severance of the cervical sympathetic nerve.

The scales of justice hung in the balance while the carotid artery was repaired and the sac evacuated (not excised), with a good result.

Arteriovenous Fistula.—Communication between an artery and a vein (or veins) may be either a congenital malformation, or acquired by the trauma of a penetrating wound or a sharp blow (fig. 139). Such a communication has both a structural and a physiological effect.

The *structural effect* of the arterial blood flow on the veins is characteristic, as they become dilated, tortuous, and thick walled (arterialised), and they also make the lesions diffuse and so render surgical procedures difficult. This is specially the case with the congenital variety.

Physiological Effect.—There is an arteriovenous shunt, which requires an increased pulse-rate and cardiac output. The pulse pressure is high if there is a large and persistent shunt. Left ventricular enlargement and, later, cardiac failure will occur. The congenital fistula in the young may cause overgrowth of a limb. In the leg, indolent ulcers are the result of relative ischæmia below the short circuit.

Clinically, a pulsatile swelling may be present if the lesion is relatively superficial. On palpation, a thrill is detected and auscultation reveals a buzzing bruit. Dilated veins may be seen, in which there is a rapid blood flow. Pressure on the artery proximal to the fistula causes the swelling to diminish in size, the thrill and bruit to cease, the pulse-rate to fall, and the pulse pressure to return to normal.

Arteriography confirms the lesion, which is noteworthy for the speed with which venous filling occurs. It is often difficult to pinpoint the actual site of the fistula.

Treatment.—*Congenital* lesions are usually stationary. Excision is advocated only for severe deformity or recurrent hæmorrhage. It is often wise to enlist the aid of a plastic surgeon in order that proper ablation and reconstruction can be effected. Ligation of a 'feeding' artery is of no lasting value.

The *acquired* lesions tend to be progressive, and operation is indicated. The vessels are separated, and, if possible, repaired by suturing, any intervening sac being excised. Failing this, ligation of the involved artery and vein is required above and below the lesion. Quadruple ligation is sometimes necessary.

In the case of arteriovenous aneurysm of the internal carotid artery and cavernous sinus, ligation and division of the common carotid artery may diminish the pulsating exophthalmos and relieve the continuous buzzing, provided that these features are improved by a trial compression of the common carotid artery against Chassaignac's tubercle, which is situated on the transverse process of the sixth cervical vertebra. Ligation and division of the common carotid artery is a safer procedure than ligation of the internal carotid, which is likely to be followed by cerebral effects.

EMBOLISM AND THROMBOSIS

An embolus is a foreign body which circulates in the blood-stream, and which may become lodged in a vessel and so cause obstruction.

Simple emboli are due to blood-clot, vegetations from cardiac valves, or fragments from an atheromatous plaque. In the heart, mitral stenosis or auricular fibrillation predisposes to their formation, which is also encouraged if fibrillation is treated with quinidine. An endo-cardial mural thrombus, following a myocardial infarct, which may be 'silent', accounts for about one third of cases.

In some situations the results of embolism are characteristic, e.g. :

BRAIN.—The middle cerebral artery is most commonly affected, resulting in hemiplegia, temporary or permanent.

RETINA.—Occlusion of the central artery causes a momentary flash of light, followed by total and permanent blindness.

Marie Chassaignac, 1805–1879. Professor of Anatomy and Surgery, Paris.

MESENTERIC VESSELS.—Causing engorgement and possibly gangrene of the corresponding loop of intestine.

SPLEEN.—Commonly affected, with local pain and enlargement.

KIDNEYS.—Resulting in pain in the loin and hæmaturia.

LUNG.—Pulmonary embolism (p. 680) is a catastrophe which may fatally interrupt convalescence after operation.

LIMBS.—An embolus is arrested at the bifurcation of a main vessel (fig. 140), causing *pain, pallor, paresis, loss of pulsation, and anæsthesia*, followed in most cases by the appearance of gangrene; occasionally spontaneous recovery ensues. More than half the cases involving the legs are due to obstruction of the common femoral artery, after which, in probable order, the aorta, popliteal, and common iliac are affected. In the arm an embolus is usually arrested at the bifurcation of the brachial artery.

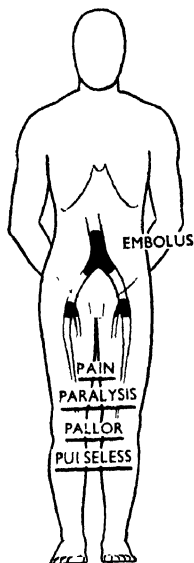


FIG. 140.—The symptoms and signs of embolism.

The danger of extension-thrombosis (consecutive or secondary thrombosis) is present immediately after an embolus becomes arrested. The presence of the embolus and the consequent stasis of blood favours the formation of blood-clot which, in a matter of hours, extends proximally as far as the nearest branch and distally down the main vessels and its branches (fig. 141). Should a collateral circulation not open up, the peripheral stasis, and therefore the thrombosis, extends still further, and gangrene occurs.

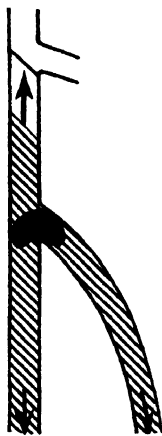


FIG. 141.—Extension - thrombosis subsequent to the arrest of an embolus.

Other types of arterial thrombosis are conveniently mentioned here,

because clinically they mimic an embolus, and they are equally complicated by extension-thrombosis. Trauma causes arterial contusion (p. 123), which results in a local thrombus. The popliteal, external iliac, and axillary arteries are specially susceptible. In axillary artery thrombosis, extension-thrombosis rapidly descends to obstruct the bifurcation of the brachial artery, a fact of great practical importance (see embolectomy). Arterial thrombosis, and this dangerous sequel may also occur in an artery affected by atheroma (e.g. coronary thrombosis, cerebral thrombosis, and femoral thrombosis, p. 133).

Treatment.—*Conservative treatment* is employed (a) if the embolism is mild and signs of recovery of the circulation are evident (loss of pain, return of movement), (b) if the patient is unfit for operation (heart failure), or (c) in cases where the limb is showing early gangrene—too late for embolectomy. *Embolectomy* is advised if a main artery is involved, the patient is fit for operation, and, generally speaking, within ten hours of the onset of the emergency.

Conservative treatment consists of (1) anticoagulant therapy, (2) treating

any cardiac failure or arrhythmia, and (3) employing those vasodilator and other conservative measures which are described in the section on basic principles (pp. 122-123).

Anticoagulant Therapy

Heparin.—This substance is present in body tissues, especially the liver and lung. It is produced by the mast cells, and prolongs the clotting time of blood. Heparin has to be given intravenously, usually in four-hourly doses of 5,000-10,000 units (50-100 mg.), via an indwelling needle or polythene cannula.

Overdosage leads to hæmorrhage, but the effect can immediately be neutralised by an intravenous injection of protamine sulphate. Each ml. of a 1 per cent. solution neutralises 1,000 units of heparin.

Synthetic Anticoagulants¹.—Phenindione B.P. (e.g. Dindevan) and dicoumarol derivatives (e.g. Warfarin or Marevan) depress the blood level of the prothrombin complex. They exert a full effect thirty-six to forty-eight hours after starting therapy, and for twenty-four hours after stopping. A loading dose is given in divided doses (loading dose of phenindione is 200 to 300 mg., Warfarin 30 to 50 mg.). The prothrombin level is estimated on the third day, and the amount given is then revised if necessary. Control is effected by estimation of the prothrombin time, which should be prolonged to two to three times the normal. The interval between estimation may be lengthened when a stable dose is found.

Overdosage is shown by hæmorrhage, especially hæmaturia. Vitamin K₁ (Phytomenadione B.P., *not* the analogue) very quickly reverses the effects of synthetic anticoagulants. It is usually enough to give 10 mg. by mouth, but if bleeding persists, it should be administered by intravenous injection.

Contraindications to the use of anticoagulants include pregnancy, hepatic or renal deficiency, hypertension, or when there is a risk of hæmorrhage, e.g. in cases of peptic ulceration.

Combined Anticoagulant Therapy.—Heparin is injected for its rapid effect, and at the same time Dindevan is started. When it is judged that the synthetic compound has exerted a full effect, the heparin is discontinued and clotting is controlled by prothrombin estimations (which, incidentally, are not valid within six hours of giving heparin).

Many surgeons prefer to rely upon heparin only, for four to six days before using the synthetic compounds, especially in cases of embolism.

Embolectomy and Thrombectomy.—Local or regional anæsthesia is used, dependant upon the patient's general condition and the scope of the proposed operation. The artery, bulging with clot, is exposed and held up by slings of fine rubber tubing. Through a longitudinal or transverse incision the clot begins to extrude and is removed, together with the embolus. Arterial clamps are applied as soon as bleeding occurs, special note being made of the degree of retrograde bleeding (back bleeding). *Special measures* may be required to extract the extension thrombus:— (a) milking the artery, (b) applying a spiral rubber bandage from below upwards, (c) retrograde flushing with heparin-saline solution (1,000 units to 150 ml. saline), or (d) best of all, by a *balloon catheter* (Fogarty). This catheter is akin to a ureteric catheter, with a balloon tip, and is introduced until it is deemed to have passed the limit of the thrombus. The balloon is inflated and the catheter withdrawn slowly, together with the clot. The procedure is repeated until bleeding occurs. The method is valuable in patients with an aortic bifurcation embolus, since the clot and embolus can be extracted by insertion of balloon catheters via the common femoral arteries in the groin and the patient is saved from a laparotomy. Postoperatively, anti-coagulant therapy is commenced.

In *axillary artery thrombosis*, it is often necessary to expose and even open the brachial artery at the elbow to be sure that it is free of clot.

¹ Dicoumarol was originally discovered in spoiled clover.

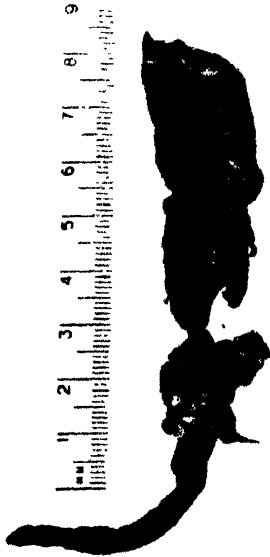


FIG. 142.—Aortic embolectomy. The extension-thrombosis extended proximally up to the renal, and distally into the iliac arteries.

Papaverine sulphate (1 per cent. solution) is painted on the arteries to reduce spasm, which can be so troublesome as to warrant a slow local extra-arterial perfusion of this substance via a polythene catheter for forty-eight hours.

Fibrinolysins (e.g. Actase) may be perfused intra-arterially for some days with the idea of dissolving any thrombus which still threatens the limb. While good results have been claimed, some patients are hypersensitive to fibrinolysins.

Air Embolism

Venous.—Air may be injected into the venous circulation (e.g. pneumothorax), or accidentally sucked into an open vein. Thus venous air embolism occasionally complicates operations on the neck or axilla if a large vein is inadvertently opened, or it may be an accessory cause of death following a cut throat. The risks associated with intravenous infusion are reduced by the use of a drip chamber containing a spherical plastic float which plugs the exit when the fluid falls to a dangerous level.

When air enters the right auricle it is churned up, and the foam enters the right ventricle and causes an air-lock in the pulmonary artery, which may end in right-sided heart failure.

There is also a risk of air embolism during air insufflation of the Fallopian tubes, and it is a special hazard of illegal abortion: air may be carried to the brain by the paravertebral veins and so causes sudden death.

Treatment.—Trendelenburg's position encourages air to pass into the veins of the lower half of the body, and the patient is placed on the left side so that air will float into the apex of the ventricle, away from the pulmonary artery. Oxygen is administered to counteract anoxæmia, and to assist in the excretion of nitrogen. In serious cases the right ventricle should be aspirated by a needle passed upwards and backwards from below the left costal margin. If this fails, the heart is rapidly exposed so that aspiration can be performed under direct vision.

Arterial.—During artificial pneumothorax air may be injected into a pulmonary vein, and so gain entrance to the left side of the heart. This complication occasionally follows operation on the lungs. *Paradoxical embolism* is due to a patent foramen ovale, as no appreciable amount of air from the right side of the heart can pass through the capillaries to the lungs.

Air in the left side of the heart may enter the coronary arteries and cause acute myocardial failure. Cerebral embolism results in dizziness, visual disturbances, and unconsciousness. Mottling of the skin of the head and shoulders, due to emboli in the cutaneous vessels, may be noticed in cases of arterial air embolism, and pallor of part of the tongue occurs if a branch of the lingual artery is occluded.

Treatment.—The foot of the bed is raised in order to hinder bubbles of air from reaching the cerebral vessels, and the patient is turned on to his left side, as owing to the site of origin of the coronary arteries, air is then less likely to enter these vessels.

Oxygen is administered, and artificial respiration instituted if necessary. As in venous embolism, aspiration of the appropriate ventricle may be a life-saving measure.

Fat Embolism

This condition, which is more common than generally supposed, follows severe injury to bone marrow or adipose tissue. It is especially liable to occur after fracture of atrophic bones, as these bones contain more than the normal amount of fat. Cases have been recorded following convulsive therapy. Symptoms are evident a day or so after injury, and two more or less distinct types, cerebral and pulmonary, are recognised. In the cerebral type, the patient becomes drowsy, restless, and disorientated (delirium tremens may be suspected). Subsequently he is comatose, the pupils become small and pyrexia ensues. The pulmonary type is ushered in with cyanosis, which increases in intensity, and signs of right heart failure. White froth may occur at the mouth and nostrils. It may be mistaken for broncho-pneumonia.

One of the earliest signs may be emboli in the retinal arteries, which cause striate hæmorrhages and 'fluffy' patches of exudate. The sputum should be examined for fat droplets, and fat may be excreted in the urine. A fall in the hæmoglobin value of the blood is a constant sign. Petechial hæmorrhages sometimes occur. Treatment consists of the use of oxygen, early heparinisation, and intravenous low molecular-weight dextran (Rheomacrodex, p. 77). Intravenous 5 per cent. dextrose solution with 5 per cent. alcohol, increases the emulsifying power of the blood and reduces the size of fat globules.

Infective emboli consist of masses of bacteria or infected clot, and may cause mycotic aneurysms (p. 125), pyæmia, or infected infarcts.

Malignant emboli are more commonly sarcomatous than carcinomatous, and give rise to secondary deposits, unless sufficient blood is extravasated to isolate the malignant cells from normal tissue, a phenomenon which sometimes occurs with chorion-epithelioma.

Parasitic emboli are due to the ova of *Tænia echinococcus*—and *Filaria sanguinis hominis* (p. 34).

Caisson and Decompression Disease

These similar conditions may affect divers, those who work in compressed air chambers, or who ascend in open aeroplanes to above 25,000 feet. If decompression is too rapid, bubbles of nitrogen are set free in the tissues and blood stream, and occlude small vessels. Symptoms include pain in the muscles or joints, which may be excruciating (the 'bends'), and neurological disturbances; if the spinal cord is affected the patient suffers from weakness of the legs and sphincters. In severe cases, the lungs may be affected, and the patient complains of tightness of the chest and a dry cough (the 'chokes'). Caisson disease requires recompression and gradual decompression. The high altitude flyer is relieved by gradual descent. Inhalation of oxygen assists the excretion of nitrogen. If the spinal cord is not permanently damaged the prognosis is good, but hypertrophic changes may persist in the ends of long bones.

ATHEROSCLEROTIC OBSTRUCTION

The reader is advised to refer to the section on basic considerations and principles of arterial surgery (pp. 117-121) in conjunction with the following.

The Legs.—Patients with atherosclerotic obstruction of the arteries to the legs comprise the majority of cases of peripheral vascular disease. It is salutary to remember that the condition is part of a generalised disease, so that while the patient may present with symptoms in the legs, there may be equally severe manifestations in the coronary, carotid, or cerebral vessels. The age of the patient is usually fifty years and over, but sometimes a 'presenile'

variety is encountered in the twenties and thirties, which must be distinguished from Buerger's disease (p. 137). The number of male patients far outweighs that of female patients in whom, curiously enough, the disease is less amenable to surgery. Non-smokers are much less commonly affected.

The symptoms (intermittent claudication and rest pain) are caused by ischæmia, so they occur when the lumen of the artery (aorta, iliac, femoral, or popliteal) becomes severely diminished, or finally obstructed. Complete obstruction is due to thrombosis of a length of diseased artery, and the patient may become aware of this by an episode of rest pain and pallor which mimics embolism. As stenosis and occlusion occur, the collateral circulation usually has time to develop, and any episode of worsening of symptoms may be followed by a phase of improvement. Eventually, even the ostia of the collateral branches become obstructed, and should further thrombosis occur, rest pain and gangrene will follow (about 10 per cent. of patients). Sixty per cent. of the patients die from coronary thrombosis, 12 per cent. from congestive cardiac failure, and 12 per cent. from cerebral thrombosis or hæmorrhage.



FIG. 143. — Aortogram showing thrombosis of the right common iliac artery. (Alan Small, F.R.C.S., London.)

The signs as recounted on p. 119 are an altered appearance and temperature; diminished sensation; diminished or absent pulsation; the presence of bruits; slow venous filling; or gangrene. It is to be remembered that while claudication pain commonly occurs in the calf, its appearance in the thigh and buttocks is indicative of obstruction to the aorta or iliac vessels (fig. 143), and examination of the pulses should confirm this deduction¹. Osteoarthritis of the hip and a prolapsed intervertebral disc are distinguished by the occurrence of pain on occasions other than walking. However, the conditions may coexist.

Arteriography (p. 120) is essential to show the exact site and the extent of the obstruction, as well as the presence of multiple lesions.

The treatment policy therefore depends upon (1) the degree of disability, (2) the general condition of the patient, and (3) the feasibility of an operation as shown by arteriography.

(1) *The Degree of Disability*.—Rest pain and early gangrene are severe degrees of disability, and demand an attempt to salvage the leg. The disability of claudication is, however, very variable. Some patients with a walking distance of fifty yards may be content to restrict their activities within such a limit. Others, such as business executives, keen walkers, golfers, and gardeners complain that a distance of 300 to 400 yards is intolerable, and they are reluctant to sacrifice either position or enjoyment.

(2) *The General Condition of the Patient.*—The absolute contraindications to operation are heart failure, severe bronchitis and emphysema, open tuberculosis, and advanced malignant disease. A recent coronary thrombosis (within three months), or a cerebral thrombosis (within six months), are temporary contraindications. Special care is required if a patient has an active peptic ulcer, since the stress occasioned by the operation (or even the arteriogram) may cause it to bleed. The ulcer requires priority treatment.



FIG. 144.—Thrombo-endarterectomy ('disobliteration' or 'rebore') from the common femoral to the popliteal artery.

(3) *The feasibility of direct arterial surgery* to restore an adequate circulation depends upon the evidence given by arteriography of the site and extent of the obstructive disease. Providing that a short length of popliteal artery is patent and there are no other obstructions, an operation is feasible (fig. 144). Obstruction to the iliac and the femoro-popliteal sections may require a two-stage procedure, the first being to restore the circulation through the iliac arteries into the deep femoral artery (*profunda femoris*).

Conservative Treatment.—If the disability is mild, and/or the general condition of the patient is poor, or an operation is not technically feasible, conservative treatment (p. 122) is advised.

Operation.—Either *by-pass artery grafting* (p. 121) or *thrombo-endarterectomy (rebore)* is advised. The latter procedure is favoured by some because, should thrombosis recur, the patient is usually no worse than before. Should a graft thrombose, the chance of gangrene is high. By-pass grafting is usually restricted to patients with severe rest pain and gangrene who are otherwise faced with amputation. If direct arterial surgery is immediately successful, 80 per cent. of the patients continue to be satisfactory for the succeeding three years. A week after operation, long-term anticoagulant therapy (e.g. Dindevan, p. 131) may be commenced.

Sympathectomy (p. 140) is *capricious in its effects*. Most patients with claudication are not helped by sympathectomy, because any resulting vasodilation affects the skin and not the muscle vessels. In threatened gangrene, a sympathectomy may tip the scales one way or the other. It is worth observing the effect of a paravertebral injection (p. 122) before deciding to perform sympathectomy.

Gangrene is treated by conservative surgery if an operation to improve the circulation is successful, otherwise a radical amputation is necessary (p. 42).

Thrombo-endarterectomy (disobliteration or rebore), with vein patch-grafts.

Femoro-popliteal Obstruction.—The patient is positioned and the incision made according to fig. 145. The artery is exposed either along the whole of the obstructed length or only in the region of the upper and lower limits.

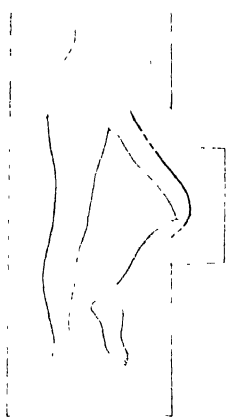


FIG. 145.—Position and incision for thrombo-endarterectomy.

The artery is exposed either along the whole of the obstructed length or only in the region of the upper and lower limits. The disobliteration is accomplished through arteriotomy incisions by inserting a steel loop (figs. 146 and 147) around a 'core' of atheroma and degenerate media, and pushing this upwards along the diseased segment. The core is divided at the upper limit and then pulled out of the artery (fig. 144). The portion of atheroma remaining at each end is dissected out. A good lumen with a free 'bleed-back' must be obtained from the popliteal artery, while above the dissection is taken into the common femoral artery. All collaterals are preserved. The rebored segment and the popliteal artery are flushed with heparin saline solution (see embolectomy, p. 131). The patient is given 2,500 units of heparin I.V. Arteriotomy wounds are closed by using vein patches (p. 121).

Aorto-iliac disobliteration usually requires extensive direct arteriotomy. The abdomen is opened by a paramedian incision, the intestines are eviscerated into a polythene bag, the aorta and iliac arteries are exposed and lifted up by slings of fine rubber tubing so that the clamps can be applied. The aorta is incised, and after any red jelly-like mural thrombus has been extracted, the inner atheromatous layer is peeled out, leaving an outer layer of media and adventitia. This procedure is carried down the iliac vessels until a satisfactory lumen and 'bleed-back' is obtained. Should an intraluminal thrombus extend up to the level of the renal arteries (as in Leriche's syndrome), it is milked or scraped out while pressure from a thumb occludes

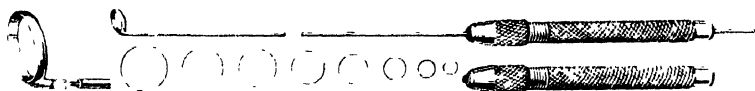


FIG. 146.—Shucksmith type of Cannon's endarterectomy loops.

the aorta and protects the renal vessels. Heparin saline is used (p. 131) for flushing out and filling the distal vessels.

Alternatively, a Dacron bifurcation graft is inserted (fig. 136) as an alternative procedure; knitted grafts are considered to be preferable to the woven variety.

Other Sites of Atherosclerotic Obstruction.—The principles of arterial surgery can be applied to other arteries which are stenosed by the disease.

Carotid stenosis causes transient, recurrent, and progressive 'strokes' (hemiplegia). On the contralateral side to the stroke, a carotid arteriogram will show either a local stenosis at the origin of the internal carotid artery, or a complete obstruction of that artery up into the skull, to where the ophthalmic artery enters as a collateral. Only a local stenosis is suitable for the disobliteration and vein-patch operation. Special measures may be taken to prevent cerebral damage occurring while the clamps are in place. Postural hypertension (operating with the patient tilted head down) can be used. A temporary polythene by-pass may, however, be required.

Vertebral artery stenosis, causing basilar artery 'strokes' of giddiness and impairment of vision, may be demonstrated by retrograde brachial arteriography (p. 120). The stenosis, at its origin from the subclavian artery, is removed and a vein patch sutured into place.

Subclavian artery stenosis (p. 139)

causes pain and a feeling of coldness in the arm and hand. Small patches of thrombus and atheroma break off as emboli, and occlude the arteries of the forearm, hand, and fingers. Resection and grafting, or disobliteration, may be performed.

Subclavian Steal Syndrome.—If the first part of the subclavian artery is obstructed, the vertebral artery provides collateral circulation to the arm, and its flow is reversed, as is demonstrated by arteriography. The cerebral circulation is diminished, hence the term 'steal'. Clinical features include syncopal attacks, visual disturbances, facial paraesthesia and diminished pulse on the affected side. A Dacron bypass of the affected part of the subclavian artery, or thrombo-endarterectomy (rebore), relieves the symptoms.

Mesenteric Artery Stenosis.—Post-cibal central abdominal pain in a patient with atherosclerosis elsewhere may be shown by an arteriogram (lateral view) to be due to stenosis of the origin of the mesenteric artery. Usually the collateral circulation via the inferior mesenteric artery has already been obstructed. The pain occurs so soon after food that the patient is afraid to eat. A by-pass graft is led from the aorta to the mesenteric artery, distal to the stenosis.

Renal artery stenosis may be responsible for some cases of hypertension (fig. 119). If the function of the kidney is satisfactory, the stenosis may either be disobliterated or by-passed (fig. 148).

Coronary Artery Stenosis.—The technique of disobliteration of a local stenosis is being developed. For many years surgeons have also developed procedures which bring a collateral circulation to the heart, e.g. cardio-omentopexy, cardio-pneumopexy, or the implantation of the internal mammary artery into the wall of the ventricle.

Thrombo-angiitis Obliterans.—Juvenile obliterative arteritis (*syn. Buerger's disease*) is a progressive condition (erroneously stated to be more common in Russian Jews), occurring in patients before the age of thirty. It affects mainly the lower limbs, usually terminates in gangrene, and is associated with severe pain and sometimes with intermittent claudication. Thickening and inflammatory changes in the arterial wall and veins with local thrombosis

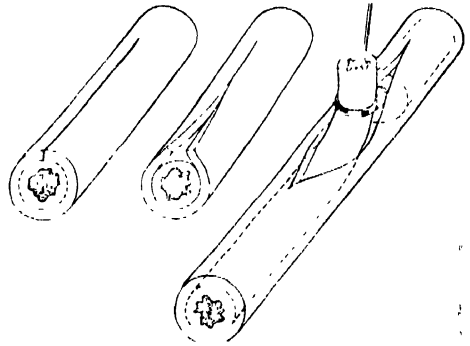


FIG. 147.—Commencement of disobliteration. An incision through the outer layer of media discloses a plane of cleavage and the core of atheroma and thrombus.

may result in complete obstruction. There appears to be a direct relationship to tobacco consumption.

In severe cases, gangrene of the toes and foot is followed by gangrene of the fingers. Even gangrene of the intestine has been recorded.

Examination will reveal reduction of peripheral pulses. The leg blanches and the veins empty on elevation of the limb owing to the deficiency of arterial blood flow. Differential diagnosis is important in distinguishing femoropopliteal thrombosis attributable to presenile atherosclerosis. Femoral arteriography serves to distinguish the two conditions.

Treatment.—In early cases, conservative measures as described on p. 122, are taken to preserve nutrition of the limb and to keep the patient ambulatory. Warm stockings, avoidance of trauma to the feet, and gentle exercise within the limit of tolerance are important. Lumbar sympathectomy should be performed early, in order to ease or relieve pain and promote the healing of ulcers. Prognosis must be guarded. Many cases, after a remarkable initial improvement in skin nutrition, are apt to relapse, although pain is usually relieved for the time. In young subjects ulceration and gangrene eventually necessitate amputation (p. 42). The prognosis in the young patient is always worse than in the older subject in whom atherosclerosis runs a less progressive course. Total abstinence from smoking is imperative, i.e. 'You can have your cigarettes or your legs, but you can't have both.'

Bilateral adrenalectomy and steroid therapy has been popular on the continent of Europe for several years. A few striking successes are claimed, and there is some improvement in half of the cases.

Other types of arteritis of the distal vessels of the leg are encountered in rheumatoid arthritis, diffuse lupus erythematosus, and polyarteritis. The principles of treatment are similar.

Diabetes is discussed on p. 43.

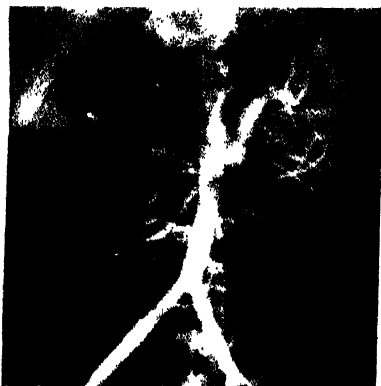


FIG. 148.—A by-pass vein graft from the aorta to the left renal artery in a little girl with bilateral renal artery stenosis, causing hypertension. The subclavian and other arteries were also occluded by Takayasu's arteriopathy (see text).

Temporal arteritis or occipital arteritis, which is a local collagen disease causing headache and sometimes retinal vessel changes, may be relieved when the tender thrombosed artery is excised for biopsy purposes.

Takayasu's arteriopathy (obliterative arteritis of females, pulseless disease) causes narrowing and obstruction of major arteries and usually pursues a relentless course (fig. 148).

Myxomatous Degeneration.—Instances of an accumulation of clear jelly (like a synovial ganglion) in the outer layers of a main artery have been reported, mainly in the popliteal artery. The lesion so stiffens the artery that pulsation disappears, and claudication occurs when the limb is flexed (as on walking up stairs). Arteriography shows a smooth narrowing of an otherwise normal artery, and a sharp kink, or buckling, when the knee is flexed. Decompression, by removal of the myxomatous material, is all that is required, but the ganglion may recur and require excision of part of the arterial wall and a vein-patch repair.

VASOSPASTIC CONDITIONS

Raynaud's disease is a spasmodic condition occurring in women, which affects the upper extremities more than the lower. The peripheral pulses are normal. The condition is attributable to abnormal sensitivity in the direct response of the arterioles to cold. When cooled, these vessels go into spasm, and as a result the part becomes blanched and incapable of finer movement. The decreased blood flow leads to an accumulation of metabolites in the capillary circulation. The capillaries dilate and become filled with slowly flowing deoxygenated blood, the part therefore becomes swollen and dusky. As the attack passes off, the arterioles relax, oxygenated blood returns into the dilated capillaries, the hands become red and a burning sensation or pain is produced by increase in tissue tension. Thus the condition is recognised by the characteristic sequence of blanching, dusky anoxia, and red engorgement. Eventually obliterative changes may occur in the peripheral vessels, superficial necrosis occurs, the tips of the fingers undergo dry gangrene, and the distal parts of the terminal phalanges are absorbed. Early cases of Raynaud's disease must be distinguished from chilblains and vascular disturbances which are sometimes associated with the costoclavicular (cervical rib and the scalene) syndrome (p. 522).

Scleroderma occurs locally in a certain number of severe cases (acro-sclerosis), or it may be associated with generalised or focal scleroderma. In the Tieberg-Wiesenbach syndrome, telangiectases of the face and scleroderma of the œsophagus are present. Œsophageal carcinoma may occur.

Treatment.—*Conservative.*—Protection from cold, avoidance of pulp and nail-bed infections, and the use of vasodilator drugs are part of the conservative regimen that is advised for mild cases. Triiodothyronine (5 micrograms thrice daily) may be efficacious (Peacock). Walder reports alleviation in hospital by the intravenous use of low-molecular weight dextran (Rheomacrodex (p. 77) combined with Heparin).

Sympathectomy.—The immediate results of sympathectomy are good, but after a few months the susceptibility to cold returns, although cyanosis is not so severe as before the operation, and subjective symptoms are less marked. This partial relapse is an indication that the underlying cause of Raynaud's disease is not in the sympathetic system, but is due to some abnormality in the smaller arteries and arterioles; sympathectomy apparently raises the threshold at which spasm occurs. From the practical point of view preganglionic section of the thoracic sympathetic chain can be recommended as a palliative procedure in Raynaud's disease, especially if performed before the onset of ulceration and absorption of the terminal phalanges.

Raynaud's Phenomenon.—Peripheral vasospasm also occurs secondarily to organic disease involving the main artery to a limb. Thus in the arm, atherosclerotic stenosis of the subclavian artery (p. 137), or cervical rib and the scalene syndrome (p. 522) may be responsible. Treatment is directed primarily to these causal lesions, though sympathectomy is sometimes required.

Acrocyanosis may be confused with Raynaud's disease, but this condition is painless and is not paroxysmal. It occurs in young females, and the cyanosis of the fingers may be accompanied with paræsthesia and chilblains. In severe cases sympathectomy may be necessary, and gives good results.

Poliomyelitis.—Old cases of poliomyelitis suffer from vasospasm. The limb, in which the muscles have been replaced by cuffs of subcutaneous fat, becomes cyanosed and painful in cold weather and chilblains and ulcers make their appearance. Sympathectomy is of value in these cases.

Operations and Odd Injuries.—Arthrodesis of a peripheral joint and some injuries, e.g. crushing and frostbite, may be followed by local cold sensitivity and vasospasm. The mechanism is obscure. Sympathectomy gives relief.

Severe chilblains (perniosis) may sometimes warrant sympathectomy.

Sympathectomy

Preganglionic Cervico-dorsal Sympathectomy.—(a) *Supraclavicular Method*—Through a supraclavicular incision, the clavicular part of the sternomastoid, the posterior belly of the omo-hyoid, and the scalenus anterior muscles are divided, the phrenic nerve being displaced inwards. The subclavian artery is exposed, and the thyro-cervical trunk is divided. The subclavian artery is depressed and the supra-pleural fascia is divided, so that the dome of the pleura can be displaced downwards. The stellate ganglion is identified as it lies on the neck of the first rib (fig. 149). The sympathetic trunk is traced downwards and divided below the third thoracic ganglion.

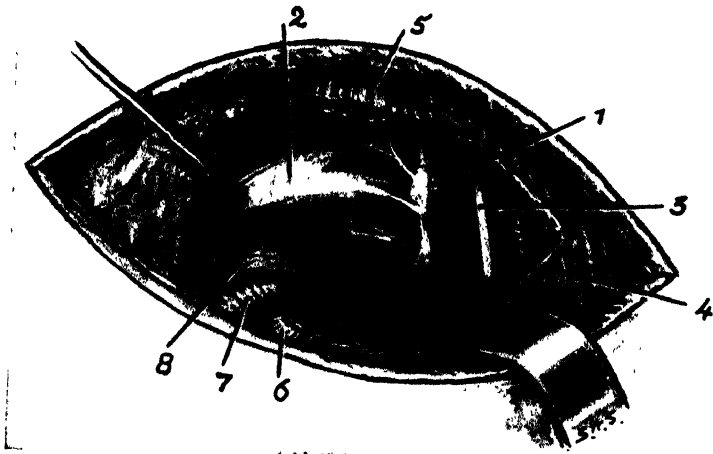


FIG. 149.—Exposure of the right cervicodorsal sympathetic chain from the front.

- | | |
|--|--|
| 1. Stellate ganglion. | 5. Divided scalenus anticus muscle. |
| 2. Lower trunk of brachial plexus. | 6. Divided posterior belly of the omo-hyoid. |
| 3. Phrenic nerve displaced inwards. | 7. Subclavian artery displaced downwards. |
| 4. Partially divided sternomastoid muscle. | 8. Dome of the pleura. |

All rami communicantes associated with the second and third ganglia and the nerve of Kuntz (a grey ramus running upwards from the second thoracic ganglion to the first thoracic nerve, are meticulously divided.

(b) *Transthoracic Method.*—This gives a greater exposure and facilitates the removal of the sympathetic chain from the fifth ganglion up to the lower fringe of the stellate ganglion. It tends to give better results than (a), and can be employed when (a) has failed. Through a periscapular incision, the posterior half of the fourth or fifth rib is removed, the pleura opened, and the lung retracted. The sympathetic chain is easily seen and after dividing the pleura, it is dissected out, care being taken to avoid damage to the intercostal vessels, which may cause tedious hæmorrhage.

Lumbar Sympathectomy.—Using a transverse loin incision, an extraperitoneal

approach is used in which the colon and peritoneum, to which the ureter clings, are stripped inwards so as to expose the inner border of the psoas muscle (fig. 150).

The sympathetic trunk lies on the sides of the bodies of the lumbar vertebræ, and on the right side is overlapped by the vena cava. Lumbar veins are apt to cross the trunk superficially. The sympathetic trunk is divided on the side of the body of the fourth lumbar vertebra, and is traced upwards to be divided above the large second lumbar ganglion, which is easily recognized by the number of white rami which join it.

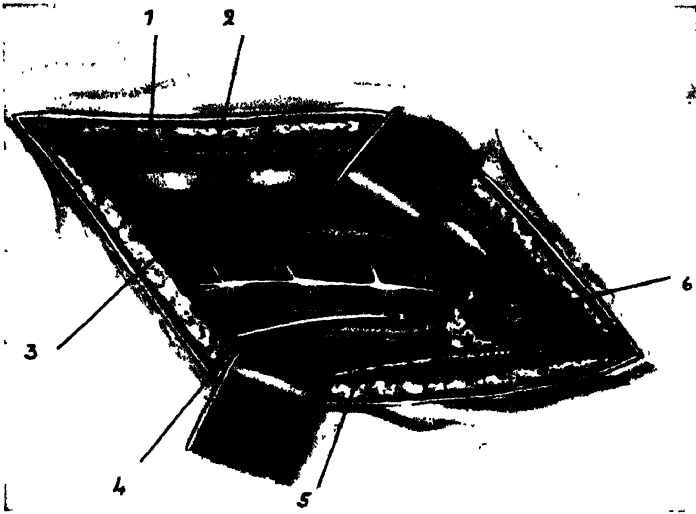


FIG. 150.—Left lumbar ganglionectomy.

- | | |
|--|--------------------------|
| 1. Peritoneum stripped forwards, with ureter (2) adherent to it. | 4. Genito-crural nerve. |
| 3. Aorta. | 5. Psoas. |
| | 6. Lower pole of kidney. |

Occasionally both sides are done at one sitting. Should a *transperitoneal* route be preferred, the colon on each side is mobilised medially by incising the parietal peritoneum in the paracolic gutter.

Bazin's disease (*syn.* erythema induratum) is due to localised areas of fat necrosis and particularly affects adolescent girls who have fat legs and a tendency to chilblains. Symmetrical purplish nodules appear, especially on the calves, and gradually break down to form indolent ulcers, which leave in their wake pigmented scars (fig. 151). The condition slowly responds to rest, general treatment, and firm elastic bandages. Vitamin K and nicotinic acid may improve the condition by causing vasodilatation, and sympathectomy is usually curative.

VEINS

Thrombosis of veins is predisposed to by:

(1) Change in the vessel wall with damage of the endothelium, e.g. inflammation or injury.

(2) Diminished rate of blood flow, as occurs during and after operations, and in debilitating conditions, such as typhoid fever.

(3) Increased coagulability of the blood, such as occurs in infective conditions, or after hæmorrhage. Some such blood change may be the



FIG. 151.—Bazin's disease.

explanation of Trousseau's sign—thrombosis of veins in association with visceral carcinoma.

The results of thrombosis are as follows:

(1) *Locally*.—The clot may organise into fibrous tissue, or the vein later becomes recanalised. Calcification sometimes follows, resulting in the formation of a phlebolith, so commonly seen in pelvic veins. Suppuration can occur, forming a localised abscess, or giving rise to pyæmia.

(2) *Distally*.—Œdema may occur, the degree depending on the size of the vessel affected. The venous collateral circulation is soon established, as evinced by widespread varicosity of the superficial veins.

(3) *Proximally*.—Thrombosis may extend upwards to larger veins, and portions of clot are liable to become detached. The resultant emboli may cause pulmonary infarcts (p. 681). If the portal drainage area is affected with infected clot, foci of infection will riddle the liver (pylephlebitis, Chap. 34).

Superficial Vein Thrombosis (Thrombophlebitis).—Commonly varicose veins are affected, or a vein which is cannulated for transfusion (p. 90). There is pain, and the vein is a tender hard cord. The skin may appear dusky and inflamed. Spontaneous thrombophlebitis, which may be sporadic and migratory (thrombophlebitis migrans), brings to mind such diseases as Buerger's disease in a young man, a visceral carcinoma (Trousseau's sign), polycythaemia, and polyarteritis. Some cases are idiopathic. The symptomatic treatment is by compression with a supportive crêpe bandage or as described on p. 149. A short sharp course of penicillin (1 mega unit twice daily for four days) is often of value.

As the thrombus is adherent to the wall of the vein, an embolus is unusual. However, a thrombus which spreads rapidly up the long saphenous vein to the groin may require emergency saphenous ligation.

Deep-vein thrombosis (phlebothrombosis) follows childbirth, operations, muscular violence, local trauma of any kind, immobility, and any debilitating illness. The thrombus may commence in a venous tributary of a main vein, where there are eddying currents around a valve. It extends in a serpentine fashion into the main deep vein where the relatively faster stream may cause a portion to break off and so cause a pulmonary embolus (p. 680). The pelvic and calf veins are more commonly implicated than others. Occlusion of a length of the deep femoral vein will cause painful congestion and œdema of the leg. If in addition there is an associated lymphangitis, the swelling will increase and is likely to be protracted (e.g. 'white leg'—phlegmasia alba dolens). Extensive deep vein thrombosis of the iliac and pelvic veins may cause a 'blue leg' (phlegmasia cærulea dolens) in which either venous gangrene or areas of infarction may threaten part or the whole of the limb (fig. 152).



FIG. 152.—Phlegmasia cærulea dolens.

Prophylaxis.—*Before operation.* When possible, grossly over-weight patients should reduce weight (Allison). Those over forty, rendered immobile during a period of in-patient investigation

or other treatment, are less at risk if they have a spell of 2-3 weeks activity at home before readmission for operation.

During operation it is essential that the venous return from the lower limbs is not impeded in any way. Pressure of the calf on the operating table *must* be prevented by elevating the heel on a sandbag or sorbo-rubber pad. At the end of an operation it is a good plan to elevate and massage the legs.

After operation conditions which predispose to a sluggish circulation such as immobility of the lower limbs, dehydration, and delayed venous return should be avoided by massage, leg movements, adequate hydration, and early ambulation. Patients should not be allowed to sit out of bed with their legs dependant. Prophylactic anticoagulant therapy is sometimes of value, e.g. in fracture femur cases affecting the elderly.

Detection of Thrombosis.—Phlebothrombosis is often symptomless, but there may be a complaint of pain in the affected calf. Any unexplained elevation of temperature or pulse-rate should arouse suspicion of thrombosis, especially when occurring towards the end of the first post-operative week. In all cases where thrombosis is anticipated, the calves should be examined daily for tender areas of induration. Tenderness may be elicited along the course of the posterior tibial and peroneal veins. There may be pain in the calf on dorsiflexion (Homan's sign). Slight swelling of the calf muscles is another important sign.

Treatment of Deep-vein Thrombosis.—(a) *Anticoagulants, Bandage and Rest.*—Opinion is divided as to the best method. At one extreme, the whole limb is bandaged with a supportive crêpe, 'blue-line' or other elastic bandage, heparin is given for eight days, and the patient is kept mobile. At the other extreme the limb is also bandaged, but the patient remains in bed until the elevated temperature and the local signs abate, and anticoagulants are not used unless a small pulmonary embolus has occurred. A middle course is to employ anticoagulants, combining heparin and phenindione (p. 131), and to continue with them for three to six weeks, taking care to tail-off slowly at the end of the course to prevent a 'rebound' of thrombosis.

(b) *Venous thrombectomy*, e.g. with a Fogarty balloon venous catheter (cf. p. 131), is being practised in the acute stage of a deep venous thrombosis, and it may give immediate relief of pain and swelling. Anticoagulant therapy and exercises are commenced post-operatively.

(c) *Ligation and 'Sieve' ('Lattice') operations.*—The repeated passage of emboli up the inferior vena cava may be barred in two ways. Ligation of the cava does not always succeed, and in the case of a woman with pelvic vein thrombosis it is necessary to ligate the ovarian veins, since they undergo compensatory dilatation and provide an alternative pathway for emboli. Ligation may also be followed by persistent œdema of the legs. The other method is to construct a 'sieve' within the lumen of the cava by a criss-cross darn of a monofilament suture material. This is a success in about 75 per cent. of cases of recurrent emboli.

(2) **Iliac Vein Compression and Obstruction** (Cockett) (fig. 153 (a), (b) and (c)). Good venograms of the iliac and pelvic veins, and the cava, may be obtained by injection of Hypaque (diatrizoate) into the femoral vein, or into the bonemarrow via the greater trochanter (per-trochanteric venogram). In some patients with swelling of a leg (see lymphœdema, p. 151), the left common iliac vein or the cava is compressed by the over-riding aortic bifurcation. In addition thrombosis in, or a stricture of, the vein may be present. Operations involving the mobilisation of the aortic bifurcation, or direct venous disobliteration, are being tried.

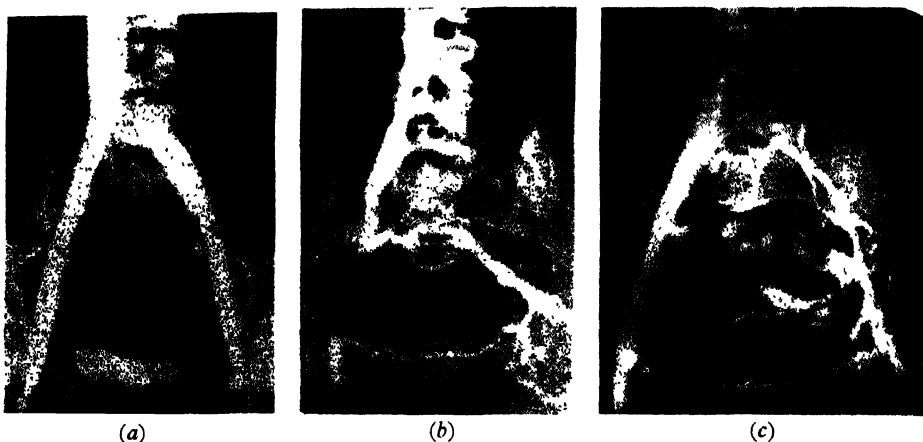


FIG. 153.—Iliac vein obstruction. (a) The defect which is often seen on iliac venograms even without symptoms is representative of an average degree of iliac compression.

(b) The venogram in the acute stage of an iliac thrombosis following on iliac compression. This patient had an embolus a few days prior to this X-ray. The whole of the left common iliac vein has disappeared and there is a large bulge in the vena caval bifurcation which is probably the smooth edge of the residual clot.

(c) Iliac obstruction in the chronic phase, with a block at the upper end of the left common iliac vein, and the typical, quite profuse, collateral circulation across to the right side. (F. B. Cockett, F.R.C.S., London.)

Axillary Vein Thrombosis.—Thrombosis of the axillary vein is not uncommon, and can occur after unaccustomed use of the corresponding arm (e.g. distemping a ceiling). The vein is damaged by the excessive movement that occurs between the clavicle and the first rib.¹ ‘Spontaneous’ thrombosis may be due to a pyjama sleeve becoming twisted around the axilla during heavy slumber, with consequent compression of the vein. The thrombosis results in painful congestion and œdema of the arm (fig. 154). As a collateral venous circulation develops, the symptoms subside; this takes about three months. The extent of the thrombosis can be limited if heparin and phenindione therapy is given in the early stages.



FIG. 154.—Infra-red photograph showing collateral venous circulation following thrombosis of the axillary vein. (Max Pemberton, F.R.C.S., Enfield, London.)

veins are affected. The three last conditions are dealt with elsewhere.

Ætiology.—Varicose veins are part of the penalty we pay for the adoption of the erect posture. Animals do not suffer from this condition.

VARICOSE VEINS

A vein is stated to be varicose when it is dilated, lengthened, and tortuous. The condition commonly occurs in connection with the veins of the leg, and also the spermatic, œsophageal, and hæmorrhoidal

¹ Henry the Fourth of Navarre (1553–1610) led his army into the battle of Ivry, shouting ‘rally round the white plume of Navarre’. He used his sword to such good effect that he could not use his arm for six weeks, and probably had an axillary vein thrombosis.

The Venous Pump.—In the human, the return of venous blood from the lower limb to the heart requires a pump equipped with non-return valves. The pumping action is provided by the muscles. Their tone and contractions, acting within the strict confines of the encircling deep fascia, squeeze or milk the blood in the direction insisted upon by the valves, i.e. towards the heart. Therefore a congenital paucity of valves, muscle weakness and wasting, or deep fascia stretching, will impair the function of the pump. Consequently, the whole weight of the column of blood from the legs to the heart is exerted on the valves (up to pressures of 90 mm. Hg), particularly on those guarding the communications between the superficial and deep venous systems of the leg (blood in the superficial system normally flows into the deep veins). The main valves affected are the sapheno-femoral lying at the junction of the long saphenous and common femoral veins (leading to dilatation of the long saphenous system), and the sapheno-popliteal (leading to dilatation of the short saphenous system). However, varices may commence at the site of any incompetent communicating vein, either alone or in conjunction with one of the above (fig. 155).

When these valves become incompetent, as they eventually must, there is a high pressure leakage of blood into the superficial system (fig. 156). The main superficial veins possess muscular walls which in some cases prevent the varicosity which affects mainly the intercommunicating network of veins.

Varicosity is also predisposed to by any obstruction which hampers

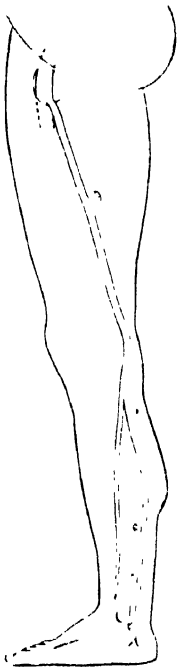


FIG. 155.—The sites of communicating veins between the deep and superficial (long saphenous) veins. The short saphenous vein has similar communications on the lateral side of the tendo achilles.

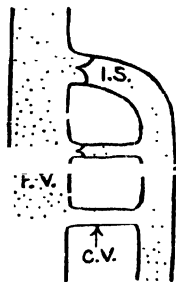


FIG. 156.—An incompetent communicating vein (C.V.), femoral vein (F.V.), internal saphenous vein (I.S.).



FIG. 157.—Varicose veins of the legs and trunk in a giant cavernous hæmangioma. Arteriography did not reveal an arteriovenous fistula.

venous return, e.g. tumours and pregnancy, and by thrombosis of the deep veins.

Varicose veins occurring below the age of twenty may be due to congenital arteriovenous fistula, or an extensive cavernous hæmangioma (fig. 157).

Symptoms occur in connection with varicose veins only when there is a retrograde flow, and they depend on the extent of the back pressure. The commonest is a tired and aching sensation, felt in the whole of the lower leg, and especially in the calf, towards the end of the day. Sharp pains, when present, are localised to the site of the varices, and are especially noticeable in grossly dilated thigh veins. The ankle may swell towards evening, or the skin of the leg may itch. Some patients suffer from cramp in the calf shortly after retiring to bed; this is due to a sudden change in the calibre of the communicating veins which stimulates the muscles between which they pass.¹

Examination.—The condition may be widespread in both legs, or a single varix is sometimes present. If this is situated close to the saphenous opening, it is called a 'saphena-varix',² and it is readily distinguished from a femoral hernia on account of the characteristic thrill when the patient coughs, or if the vein below is tapped with a finger.

The examination of the varices is most important, for upon it depends the success, or failure, of treatment. The aim is to locate the site of the incompetent superficial-deep valves (fig. 155), remembering that if an incompetent valve is present the venous flow is retrograde, so that veins when emptied fill from above; normally they fill from below. The examination, based upon the test described by Brodie (1846) and Trendelenburg (1890), involves what has aptly been described as 'the intelligent use of the tourniquet'.

Brodie-Trendelenburg Test.—Briefly, the patient lies upon his back and raises his leg to empty the veins (fig. 158A). A venous tourniquet is applied just below the saphenous opening (fig. 158B), and he stands up (158C). The constriction is then released (158D). If the sapheno-femoral valve is incompetent, the veins fill immediately from

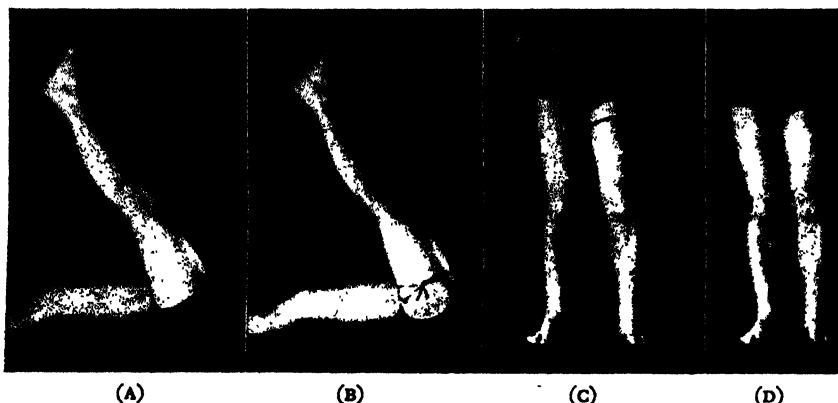


FIG. 158.—The Brodie-Trendelenburg test (see text). (Dr. S. M. Rivlin, London.)

¹ Quinine dihydrochloride (200 mg.), taken on retiring, is often efficacious in relieving night cramps.

² The word 'saphenous' is of Arabic derivation and means 'seen easily'.

Sir Benjamin Collins Brodie, 1783–1862. Surgeon, St. George's Hospital, London.
Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Leipzig.

above; if not, the veins fill slowly from below. The veins may fill rapidly from above, even though the tourniquet has not been released; this means that the varices are in communication with a perforating vein (one is commonly present in the lower third of the thigh), or the sapheno-popliteal junction is incompetent. In this case, the tourniquet test must be repeated, with application at successively lower sites on the thigh and leg, until the point of origin is shown by prevention of the abnormal direction of flow.

Treatment.—(i) **Palliative treatment** is required for varicose veins in pregnancy, and for patients who do not wish for, who are unfit for, or who are waiting for, operation. The veins are supported by elastic stockings or crêpe or elastic bandages.

Rowden Foote describes how a patient with an aching leg used to obtain relief by employing the Brodie-Trendelenburg manœuvre. If he laid down, elevated his leg, and then pressed hard in his groin, the veins remained empty when he stood up, and the aching was relieved.

(ii) **Injection.**¹—The injection of chemical sclerosants, such as monoethanolamine oleate² 5 per cent. (Ethamolin), or sodium tetradecylsulphate 3 per cent. (Sotradecol or Trombovar) acts by damaging the intima of the vein so that a thrombosis, and later sclerosis, develops. Intimal damage will take place only whilst the sclerosant is able to act in sufficient concentration; as soon as it is diluted by the blood in the deep veins the effect is lost. If an excess of the sclerosant is injected at one site it may reach the deep veins in sufficient concentration to initiate a thrombosis before it becomes sufficiently diluted to render it harmless. *The minimum fully effective dose* for a sclerosant should always be known, and should not be exceeded at any one site. *Indications.*—Unless careful attention can be paid to the detail of giving a course of injections (see below), injection treatment is limited to: (a) As a 'follow-up' for small veins which persist after an interval of two months following stripping, and (b) possibly for cosmetic reasons.

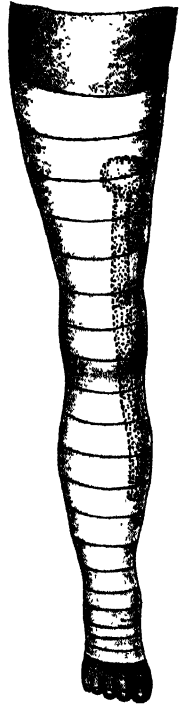


FIG. 159.—Bandage and pressure in the injection treatment of varicose veins (see text), and the ambulatory treatment of thrombophlebitis (p. 142).

Technique.—In order to make the venous occlusion permanent, injections must be given into an empty vein, so that wall adheres to wall with no intervening blood clot and thrombosis which will certainly recanalise. The needle is inserted into the vein with the patient standing, but the injection is given with the patient lying down with the leg elevated. A sorbo pad is then put over the site of the injection, and if necessary along the length of vein (fig. 159), and it is maintained by pressure bandage and a stocking overall for six weeks (Fegan). Injections are also given at the site of the perforating veins as delineated by the Brodie-Trendelenburg test (above) and at the maximum site of tenderness.

The maximum dose at any one time and any one point is 2 ml. It is usual to commence treatment with a test dose of 1 ml. so that the extent of the local reaction may be gauged and the dose modified as necessary.

¹ A word of warning is necessary in this era of the contraceptive pill. While it is still not proven that the incidence of spontaneous deep vein thrombosis is related to the taking of 'the pill', caution should be exercised before giving sclerosant injections to a woman practising this form of family planning. Fatal thrombo-embolism has been reported.

² Strange to relate, Mother Nature has, from the days of the primeval swamp, provided this substance from platelets in the actual chemistry of thrombosis.

(iii) Operations for Varicose Veins

Ligation Procedures.—The basic principle of the operative treatment of varicose veins is the ligation and division of those veins into which the high-pressure leak from the deep venous system has primarily occurred. Almost always this means ligation of the long saphenous vein where it enters the femoral vein (sapheno-femoral flush ligation). A flush ligation proximal to any tributaries is essential, otherwise a recurrence is inevitable (fig. 160).

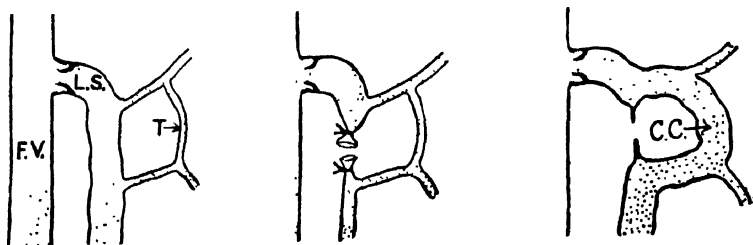


FIG. 160.—Tributaries (T.) must be ligated as well as the long saphenous vein (L.S.) flush with the femoral vein, otherwise a collateral circulation (C.C.) develops.

Should the short saphenous vein be the site of a high-pressure leak, it must be exposed in the popliteal fossa and ligated, proximal to any superficial tributaries, close to or flush with the popliteal vein.

In other instances of incompetent valves (or 'blow outs') of the communicating veins (fig. 155), they must be ligated under the deep fascia (p. 150).

Technique.—An oblique incision is made just below the groin, commencing over the pulsation of the femoral artery and extending some 6 to 7 cm. medially. The proximal portion of the long saphenous vein is exposed and traced to the femoral junction, which may lie 1.25 cm. deep to the fossa ovalis, *dividing and ligating all tributaries* encountered on the way. It is then tied flush with the femoral vein.

Stripping.—This procedure is ancillary to ligation, and is a method whereby the main veins of the superficial system (long or short saphenous) can be removed, in order that the remaining varicosities of the superficial network will thrombose and be permanently occluded.

After juxta-femoral ligation of the long saphenous vein and ligation of its tributaries a metal stripper is introduced into the vein near the internal malleolus (fig. 161). The stripper is threaded upwards to emerge from the proximal end of the vein, the leg is elevated, and the vein avulsed by traction. Bleeding is controlled by pressure bandages applied from below upwards as the stripper is withdrawn. The wounds are sutured, and the patient is ambulatory the following day. If advisable, an incision over the popliteal fossa permits stripping of the short saphenous vein, after it has been ligated at its entry into the popliteal vein.

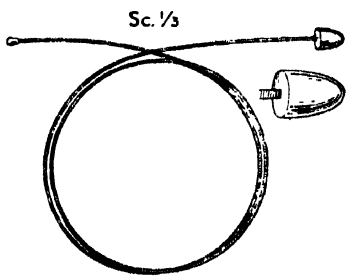


FIG. 161.—Myer's vein stripper.

Contraindications to Injection and Operative Treatment

(a) *Acute Infective Thrombophlebitis.*—At least three months should be allowed to pass, after this has completely subsided, before injecting.

(b) *Deep Thrombosis.*—Due to any cause and revealed by a history of prolonged confinement to bed with a painful swollen leg. Perthes' test is informative if doubt exists regarding the patency of the femoral vein. The saphenous vein is occluded by a tourniquet applied immediately below the saphenous opening, and the patient

walks 15 to 20 yards (13·5 to 18 metres). Normally, the veins below the constriction become less obvious, but if the communicating veins or the femoral vein are obliterated, these subcutaneous veins become engorged.

(c) *Pregnancy and pelvic tumours.*

COMPLICATIONS OF VARICOSE VEINS

Thrombophlebitis of superficial veins reveals itself as a reddened, tender cord in the subcutaneous tissues. Ambulatory treatment is safe and convenient. Strips of foam rubber or P.V.C., the edges of which are bevelled, are laid over the inflamed vein, and an inch (2·5 cm.) above it a double thickness is placed transversely. The leg is then bandaged with an elastic bandage (fig. 159). The strapping is removed after a fortnight. This procedure gives immediate relief, and it need be renewed only if tenderness persists.

Eczema (chronic dermatitis) may follow minor trauma or to the patient scratching his itching skin due to the extravasation and break-down of red-cells in the affected area. It may be an allergic manifestation resulting from ointment or strapping applied for treatment. The condition is treated before dealing with the varices, by the application, twice daily, of an ointment containing zinc oxide and coal tar. As soon as the skin is healthy, the varices should be treated, otherwise a recurrence of the itching predisposes to a further exacerbation.

Venous ulcers¹ occur either in connection with varicose veins (fig. 162), or follow deep-vein thrombosis in which recanalisation of the deep vein has occurred, but the valves are either no longer apparent or competent. Venous stasis is the underlying cause of both types. It is important, firstly, to be sure that the ulcer is not an ischæmic ulcer due to atherosclerotic arterial obstruction, arteritis (p. 138), or syphilis (p. 27). Secondly, it is important to differentiate between these venous ulcers, for whereas a varicose ulcer responds promptly to ambulatory treatment or operation (see below), post-thrombotic ulcers are refractory to treatment, and may require bed-rest with possibly excision and skin grafting. Carcinomatous changes may occur in a chronic ulcer (fig. 49).

A *deep venogram* is particularly useful in difficult cases, as it will show the patency and the size of the lumen of the deep veins, the presence of valves, and the existence of high-pressure leaks in the calf:—

A fine rubber tourniquet is applied just above the malleoli to occlude the superficial veins. An injection of 20 to 30 ml. of 45 to 65 per cent. Hypaque (sodium diatrizoate) is made into a superficial vein of the foot, via a fine polythene cannula. (A test injection is, of course, given first, p. 120). The contrast medium is thus forced into the deep veins, and serial X-ray exposures are made of the whole leg. High-pressure conditions are simulated by tilting the patient into the semi-vertical position or pinching the nostrils and endeavouring to expire forcibly.

Treatment.

(a) **Bisgaard method.** Almost any venous ulcer can be healed by elevation, bandaging, exercises, and massage. A physiotherapy department can be most helpful in applying the Bisgaard treatment, which consists of:

(1) Massage in elevation to the whole leg and particularly to soften the indurated area round the ulcer.



FIG. 162.—Venous ulcers with pigmentation of skin and talipes equinus deformity.

(2) Passive movements to maintain the mobility of the foot and ankle.

(3) Active movements to the calf muscles in elevation, and in standing (with bandages on).

(4) Teaching correct walking, placing heel down first and using the calf muscles to lift the heel of the back foot, giving 'spring' to the walk and therefore improving the venous pump (p. 145).

(5) A firm elastic (e.g. 'blue line') bandage is applied spirally from the base of the toes to the knee, so that movements in walking alternatively stretch and relax the bandage and produce an added pumping effect.

(b) **Bandage and elevation.** Without facilities for Bisgaard treatment, reliance is placed upon the use of compression bandaging (e.g. wet 'Viscopaste' type, or a firm spiral of elastic bandage over a suitable dressing). Proper bandaging reduces the local œdema and aids the muscular component of the venous pump

(p. 145). The patient should sleep with the foot of the bed raised (fig. 163) and periods of high elevation of the leg are to be strongly encouraged (fig. 164). A 'wet' bandage cannot be wound spirally round the leg without the bias of the weave cutting into the skin. It must be applied in many encircling and overlapping strips. Often a piece of felt, with bevelled edges and cut to a size larger than the ulcer, is interposed in the bandage, over the ulcer, to reduced the local œdema which so often retards healing. A crêpe bandage is put on over the 'wet' bandage. The patient is instructed to continue his usual work, and to wash off any discharge which may percolate through the bandage.

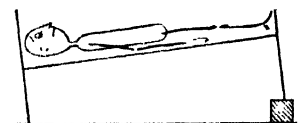


FIG. 163.—Sleeping with the foot of the bed raised.

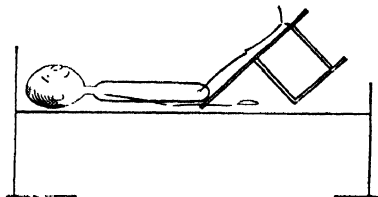


FIG. 164.—High elevation of the leg, using a chair.

bandage cannot be wound spirally round the leg without the bias of the weave cutting into the skin. It must be applied in many encircling and overlapping strips. Often a piece of felt, with bevelled edges and cut to a size larger than the ulcer, is interposed in the bandage, over the ulcer, to reduced the local œdema which so often retards healing. A crêpe bandage is put on over the 'wet' bandage. The patient is instructed to continue his usual work, and to wash off any discharge which may percolate through the bandage.

The first application is removed after one week, and thereafter the bandage is renewed at fortnightly intervals until the ulcer is healed. The area of the ulcer should be measured at each visit, so that the decrease in size can be recorded.

(c) **Subfascial Ligation** (Cockett & Dodd Operation).—Most venous ulcers are due to incompetence of perforating veins on the inner side of the ankle (fig. 155). Therefore, when the ulcer has healed, the perforating veins should be identified and divided through an incision in the lower half of the leg 2.5 cm. behind the posterior border of the tibia. The veins, which may be enormously dilated, are best secured by exposing them beneath the deep fascia. If necessary the saphenous veins are also dealt with, as already described.



FIG. 165.—Calcified varicose veins, with periostitis of the tibia.

An indolent ulcer is sometimes best treated by excision and skin grafting (p. 114). In order to avoid œdema, pressure bandages must be worn for a few weeks after the operation.

Hæmorrhage from a ruptured varicose vein is usually profuse. Elevation of the leg and the application of a firm pad and bandage easily control the bleeding. *On no account* should a tourniquet be used (p. 70).

Calcification occasionally occurs in veins which have been varicose for many years (fig. 165).

Periostitis occurs in long-standing cases if the ulcer is situated over the tibia.

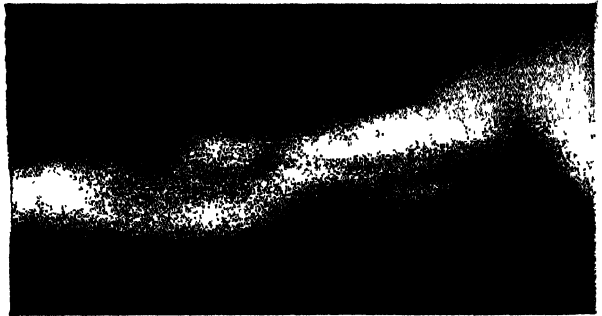
Talipes equinus may result from a long-standing ulcer. The patient finds that walking on the toes relieves the pain, and after some years the tendo Achillis becomes contracted. Cases usually respond to remedial exercises. The Bisgaard treatment should prevent this complication.

CHAPTER 9

LYMPHATICS AND LYMPH NODES

Acute lymphangitis is caused by infection spreading from a wound in the drainage area of the involved lymphatic vessel. The infection is usually limited by the lymph nodes immediately proximal to the site of infection, where an abscess may form, but occasionally infection by-passes a group of lymph nodes and affects those at a higher level. Thus, in the case of an infected foot, the abscess may form in the external iliac lymph nodes instead of the groin, and because the wound itself has sometimes healed and been forgotten by the time the mass appears, it may be mistaken for an appendix abscess. Lymphangitis from hand infections is described on p. 160.

FIG. 166.—Acute lymphangitis of the arm. Red streaks and blushes external from the infection on the forearm up to the axilla.



Acute lymphangitis is characterised by the appearance of red blushes and streaks in the skin, corresponding to the inflamed lymphatics (fig. 166). The streptococcus is the common infecting organism. Toxæmia is often severe, and is the greater the more proximally the lesion has extended. Permanent lymphatic occlusion may follow acute lymphangitis, leading to persistent œdema.

Treatment consists of bed rest, with the affected limb comfortably elevated, and giving penicillin, which usually causes rapid resolution. Only when suppuration is present should an incision be made.

Chronic lymphangitis may follow one attack, or more commonly, repeated attacks of acute lymphangitis, usually due to the hæmolytic streptococcus or staphylococcus pyogenes. Tuberculous lymphangitis also occurs and may be seen in the neck, when the lymph nodes are matted together, and in the groins when it may produce chronic lymphatic obstruction and elephantiasis.

LYMPHŒDEMA (and ELEPHANTIASIS)

Lymphangiography has solved many of the problems concerning lymphatic œdema. Patent blue dye is injected into the web between the toes, thus showing up

Olof Rudbeck, 1680–1702, Anatomist, Uppsala, Sweden, first described the lymphatic system.

the lymphatics on the dorsum of the foot, which are exposed, cannulated, and then injected with a radio-opaque solution (ultra-fluid lipiodol) in order that the deeper main trunks in the leg may be visualised on X-ray (Kinmonth and Taylor). The method requires patience and practice.

Lymphoedema is caused by the accumulation of fluid in the lymphatics. In the limbs it affects mainly the subcutaneous tissues where there is an increase both in the stagnant lymphatic channels and in the supporting fat.

The cause of the lymph stasis is either congenital or acquired.

(a) **Congenital malformations (primary lymphoedema, fig. 167)**, which may be of three varieties:

(i) **Aplasia**—the main subcutaneous trunks fail to develop. The dermal lymphatic plexus is dilated.

(ii) **Hypoplasia**—the main subcutaneous trunks are few and underdeveloped.

(iii) **Dilated and varicose lymphatic trunks**—this may be associated with congenital arterio-venous fistula, and also with 'chylous reflux' (p. 153).

(b) **Acquired obstruction (secondary lymphoedema)**. The cause of this may be:

(i) **Trauma**, e.g. the removal of axillary lymphatics in radical mastectomy.

(ii) **Repeated acute infection**, as in those who go about barefoot.

(iii) **Chronic infection**, e.g. tuberculosis, filariasis¹ (p. 34), and fungus infection.

(iv) **Obstruction by malignant disease**.

Whatever the cause, the stagnation in the lymph stream is usually followed by recurrent infection. Each such attack makes the obstruction worse until the leg becomes grotesquely swollen, the skin folded and wrinkled and coarsened (like that of an elephant) with brown areas of dry undesquamated keratin. Ulceration eventually follows.

The congenital variety is either hereditary or familial (Milroy's disease). The oedema may be in one or both legs. It may start at birth (Lymphoedema congenita), or at puberty (L. praecox), or in adult life (L. tarda). It continues throughout life and does not of itself produce any constitutional disturbance.

Differential Diagnosis.—*Bilateral* oedema of the legs always requires an examination to exclude cardiac, renal and metabolic (hypoproteinaemia) causes. *Unilateral* lymphoedema may be confused with that caused by deep vein thrombosis or compression, particularly of the iliac veins on the left side (figs. 152, 153 and p. 142).

¹ Sir Patrick Manson, 1844–1922, discovered the filaria in China in 1877. The organisms may be discovered microscopically in a nocturnal blood smear.

John Bernard Kinmonth, *Contemporary*. Professor of Surgery, University of London (St. Thomas's Hospital).
Gerald William Taylor, *Contemporary*. Professor of Surgery, University of London (St. Bartholomew's Hospital).
William F. Milroy, 1855–1942, Professor of Clinical Medicine, University of Nebraska, described the condition in 1892.



FIG. 167.—Primary lymphoedema of the right leg.

Treatment.—1. *Palliative.*—For the attacks of inflammation, prolonged bed-rest, elevation, and the appropriate antibiotic are important. Even in between attacks, the patient should sleep with the foot of the bed raised and may require to use elastic pressure—preferably of the spiral type (e.g. ‘blue line’ elastic bandage). Intermittent diuresis, induced by modern diuretic drugs, also is helpful.

2. *Surgery* is reserved for those with severe disability. The most useful of many surgical procedures is the removal of all the abnormal subcutaneous tissues and the covering of the exposed deeper tissues with a split skin graft (flaying operation). Operations seeking to divert the lymph flow (*a*) through the deep fascia by removing strips of it (Kondoleon’s operation) or (*b*) across the obstructed zone by a skin pedicle bridge, have failed. This is partly because the great mass of abnormally thickened subcutaneous tissue still remains.

Chylous Reflux.—Lymphangiography has shown that in some cases of lymphœdema there is a backflow of milky chyle from the cisterna chyli, there being dilated lymphatics, resulting in cutaneous chylous vesicles and fistulæ on the limbs. Ligation and excision of the lymphatics on the posterior abdominal wall may cure the reflux, though lymphœdema persists. A few cases have hypoplastic lymphatics and hypoproteinæmia.

LYMPHANGIOMA

There are two types:

Capillary Lymphangioma.—Localised congenital anomalies may be composed of capillary-like lesions in the skin. They are brownish papules or wart-like excrescences. On examination with a lens, small vesicles can be seen, which are lymphatic nævi.

Cavernous lymphangioma is often associated with the preceding variety, and consists of masses of lymphatic cysts, particularly in the neck or axilla, the condition being termed a *cystic hygroma* (fig. 168 and p. 520).

A similar lymphangiectatic condition may affect the lips (macrocheilia) or the tongue (macroglossia) producing sometimes gross soft tissue enlargement.



FIG. 168.—A cystic hygroma.
(Harold Rogers, F.R.C.S., Belfast.)

LYMPH NODES

Acute Inflammation.—Lymph nodes, draining any area where there is acute infection, will also become inflamed. This has been discussed at the beginning of the chapter.

Chronic inflammation is either simple (pyogenic) or specific.

1. *Simple Lymphadenitis.*—Chronicity is present because of persistence of infection, such as occurs in recurrent tonsillitis or pediculosis capitis; or in inefficient treatment with antibiotics. The treatment is to remove the cause and attend to the general health of the patient.

2. *Specific Lymphadenitis*

(a) **Tuberculous lymphadenitis** is common in children and young adults, particularly those who have been in contact with open human tuberculosis or who drink infected milk.¹ It also occurs in the aged. The cervical lymph nodes are most often seen to be enlarged, but the remnants of disease may be observed in the mediastinal and mesenteric lymph nodes as the speckled calcification on routine X-rays (fig. 169). Axillary lymph nodes may be involved by spread from the mediastinum.



FIG. 169.—Extensive calcification of cervical lymph nodes.

Tubercle bacilli most commonly reach a lymph node by lymphatics, when tubercles first form in the cortex. Blood-borne infection sometimes occurs, in which case the medulla of the lymph node is the first part to be affected. Microscopically, endothelial cells are in evidence, and giant cells are commonly seen with many nuclei arranged round the periphery like a horseshoe.

The stages of infection show most clearly in the neck. From the tonsillar portal of entry the infection spreads by the lymphatics to the nearest lymph node. If resolution fails and the disease spreads, several lymph nodes are involved. They coalesce and break down to form caseous tuberculous pus, which may perforate the deep fascia and present as a fluctuant swelling on the surface (collar-stud abscess) (fig. 170). The skin is at first cold or white,

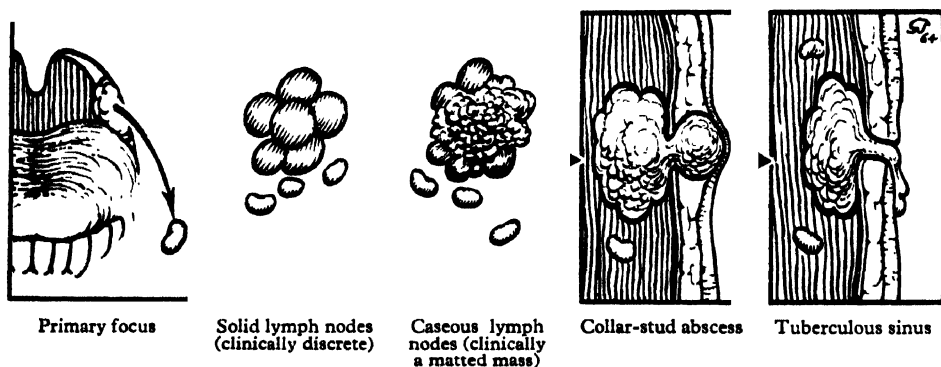


FIG. 170.—A summary of the natural history of tuberculous lymphadenitis.

but later becomes red (fig. 171), breaks down, and forms a sinus which if ignored will remain unhealed for years. From each of these stages resolution may take place with calcification (if caseation has occurred), and with much scarring (if sinuses have formed).

Treatment.—(i) Attention to nutrition and general health.

(ii) Tuberculous material is aspirated for culture and drug sensitivity tests. (A specimen must be obtained before the anti-tuberculous drugs are started.)

¹ Tuberculosis has now been eradicated from cows in Great Britain.

(iii) Anti-tuberculous drugs are given immediately after the aspiration.

(iv) When the patient's condition begins to improve, breaking down tuberculous lymph nodes must be removed, because the drugs will not reach the organisms in the avascular caseous material.

(b) **Syphilitic adenitis** can occur in any stage of the acquired infection. 'Shotty' lymph nodes in the groin associated with a genital chancre are characteristic. During the secondary stage a generalised enlargement of lymph nodes occurs. Especially noticeable are those above the internal epicondyles and along the posterior border of the sternomastoid. In the tertiary stage a gumma may occur in a lymph node, but is a curiosity. More commonly the lymph nodes enlarge as a result of secondary infection from a broken-down gumma.

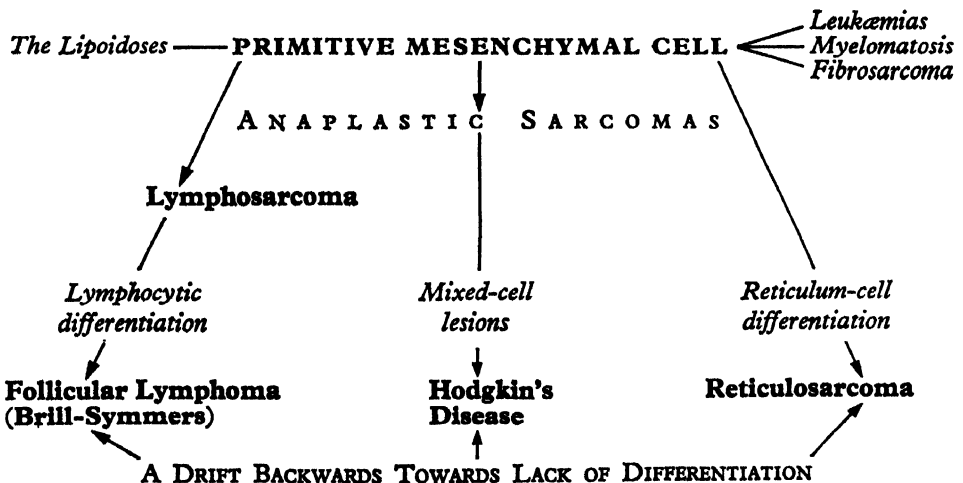
Glandular Fever (*syn. infective mononucleosis*).—This is an acute virus infection, and variable in its manifestations. The incubation period is from five to fourteen days. The usual clinical manifestations are enlargement of lymph nodes, which are elastic and slightly tender, associated with an irregular fever. A sore throat, splenic enlargement and a rash are not uncommon. Examination of the blood reveals an absolute and relative lymphocytosis, and an unusual concentration of sheep-cell agglutins (the heterophile antibody of the Paul-Bunnell reaction). The condition may be mistaken for acute leukaemia or lymphadenoma, but blood examination is diagnostic. Treatment is symptomatic, and recovery is the rule, although it may be some months before the lymph nodes return to normal. The patient should be isolated while symptoms persist.



FIG. 171.—A cold abscess of the neck.

THE RETICULOSES

Numerous progressive diseases arise from neoplastic change in the various elements of the reticulo-endothelial system. In general, the less they are histologically differentiated, the more rapidly do they grow. In addition, they may start as slowly growing well-differentiated lesions and then become rapidly growing and more primitive as time goes on ('drift-back'). There is a great deal of variation in the histological types and no rigid classification is entirely satisfactory. The following diagram gives a possible classification, and summarises this paragraph.



These diseases may either present as lymph node enlargement, or in the systemic form when virtually any structure can be affected. The lymph nodes are usually enlarged as one group, though other groups (e.g. Hodgkin's disease) may be affected. The nodes are discrete and elastic, being more rubbery in Hodgkin's disease and follicular lymphoma, but larger and softer in lymphosarcoma and reticulosarcoma. Tuberculous nodes classically are matted together by peradenitis, but may be discrete in early cases and in the senile form.

Diagnosis.—The clinical examination includes palpation of other groups of lymph nodes, and the abdomen for the presence of an enlarged spleen or liver. Investigations include X-ray of the chest (? mediastinal lymph nodes) and a full cytological examination of the blood (? leukæmia). A biopsy of an easily accessible lymph node should not be delayed. It is best to perform this under a general anæsthetic. The groin is avoided, if possible, as histology of the nodes is often complicated by inflammatory changes.

Hodgkin's disease is now described as an example:

Hodgkin's Disease (Lymphadenoma)

Pathology.—On macroscopical section the involved lymph nodes are fleshy, pinkish grey in colour, and as there is no peradenitis they are discrete.

The spleen is the seat of 'hard-bake' infiltration, by which is meant that its cut surface resembles toffee containing almonds. In nearly all cases coming to necropsy, metastases are present in the long bones. Histologically, the picture varies with the stage of the disease. At first there is a proliferation of leucocytes, which is followed by the appearance of pale round endothelial cells. Characteristic giant cells (Sternberg-Reed) are often in evidence; these contain two or more pale nuclei which overlap each other ('pennies on a plate'). Plasma and eosinophil cells are usually to be seen. There is a fine fibrosis throughout, and the structure of the normal lymph node is lost.

Clinical Features.—It is commoner in males, and usually affects young adults, but cases vary widely as regards age incidence and virulence. Occasionally, and especially in children, the expectation of life is merely a matter of weeks, the associated irregular and often high temperature leading to errors of diagnosis.

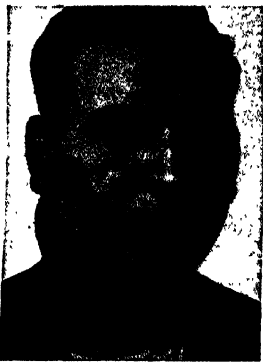


FIG. 172.—Hodgkin's disease. Diagnosis confirmed by histological examination of a lymph node.

More commonly the patient first notices a painless swelling, e.g. in the supraclavicular region (fig. 172), associated with malaise and an irregular temperature. Pressure effects, due to deep nodes, especially mediastinal, e.g. obstruction to the superior vena cava, may follow, or occasionally cause the first symptoms. On examination the nodes are discrete, painless, and rubbery in consistency. The spleen is enlarged, but rarely enough to be easily palpable. Fig. 173 shows the various sites in which the disease presents. In the later stages most organs in the body become affected, and irregular rises of temperature occur

Thomas Hodgkin, 1798–1866, described seven cases of this affliction while he was Curator to the Museum, but he failed to obtain the post of Physician to Guy's Hospital, London.

Karl Sternberg, 1872–1935. Pathologist, Germany.

Dorothy Reed, Contemporary. Pathologist, Johns Hopkins Hospital, Baltimore, U.S.A.

during exacerbations. The *periodic* bouts of temperature (Pel-Ebstein), traditionally and erroneously associated with Hodgkin's disease, were due to undulant fever, but *irregular* elevations of temperature do occur. Osseous involvement and pathological fractures are not uncommon, and an X-ray reveals areas of rarefaction in affected bones which resemble multiple myelomas. Death is usually due to the resultant anæmia.

Blood examination reveals a secondary anæmia, with occasionally slight eosinophilia, and serves to distinguish other conditions, e.g. lymphatic leukæmia. Excision of an appropriate node usually clinches the diagnosis.

Lesser-known Symptoms.—Pruritus, unassociated with eruption, is a frequent early symptom. It occurs some time in the course of the disease in 25 per cent. of cases. Pain induced by imbibing alcoholic beverages is a firmly established, but unexplained, symptom that is not infrequent, and is sometimes the first symptom of this fell disease. The pain, which is acute and lasts about twenty minutes, is located most often in the thorax; at other times it is experienced in the site of obvious lymphadenopathy. Late in the course of the disease intraosseous metastases give rise to bone pain in 20 per cent. of cases.

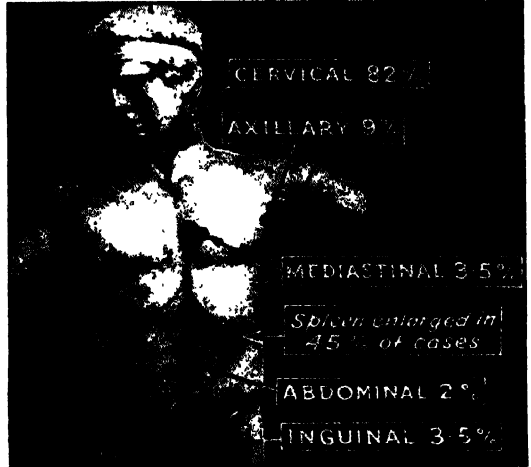


FIG. 173.—Showing the position of the lesion when the patient was seen first. In 14 per cent. of cases more than one region was involved. (Baker and Mann's statistics.)

Treatment of the Reticuloses.—Nowadays this usually rests with radiotherapy and chemotherapy. Other than for biopsy, surgery is not often employed, though if Hodgkin's disease is confined to one group, a successful ablation may be possible. Lymphosarcoma is often dramatically radio-sensitive and the five-year cure rate for localised disease in the neck is 70 per cent. Up to 50 per cent.



FIG. 174.—Reticulosarcoma of the parotid, which responded to radiotherapy.

Pieter K. Pel, 1852–1919. Professor of Medicine, Amsterdam, Holland.
Wilhelm Ebstein, 1836–1912. Professor of Medicine, Göttingen, Germany.

five-year cures have been obtained in Hodgkin's disease and reticulosarcoma (fig. 174).

Chemotherapy is used for the palliation of generalised disease as it may produce some remissions. Nitrogen mustard, which gave severe reactions, has been replaced in turn by chlorambucil and methyl-hydrazine.

Steroid therapy in the form

of prednisolone 10 mg. q.d.s. is often very helpful to the patient's morale, and improves the blood picture. It may also relieve itching. Blood transfusion to 100 per cent. is desirable in any degree of anæmia.

Malignant Lymphoma of Africa (Burkitt Tumour) (fig. 175).—*The ætiology* is of special interest. This rapidly growing tumour is the commonest tumour in



FIG. 175.—Burkitt tumour. (Dr. Denis Burkitt London.)

children aged between 3–9 years living in the equatorial regions of Africa, in low-lying country and where the annual rainfall is not less than 20 inches and the mean temperature of the coldest month of the year is not less than 60° F. This ætiological environment corresponds with that of trypanosomiasis and yellow fever and it is believed that a mosquito is the vector of a cancer-producing virus of the herpes type. A similar tumour has been encountered in the equatorial regions of S. America. Other evidence to support the viral theory is that babies under eighteen months are immune (natural immunity from the mother), that the disease is uncommon after puberty (active immunity), and that immigrants to the endemic area may contract the disease.

Clinical and Pathological Features.—The rapidly growing, soft and relatively painless tumour is of multifocal origin with a predilection for the jaws and abdominal viscera (mainly kidneys and ovaries). Other parts of the skeleton and other viscera

may be affected, but peripheral lymphadenopathy and involvement of the spleen is unusual. The jaw tumour, which is osteolytic, invades the orbit and the mouth; the eye is displaced and the teeth drop out. Paraplegia is caused by compression of the spinal cord by the growth invading the vertebral canal. On microscopic section dense masses of darkly staining primitive lymphoid cells interspersed with large clear histiocytes give an appearance, under low power magnification, of a 'starry night'.

Treatment.—Remissions, some prolonged and a few permanent, are achieved by cytotoxic chemotherapy, e.g. cyclo-phosphamide or methotrexate. The best results are obtained if treatment is commenced as early as possible and if the dose of the drug is not such as will suppress the immunological response of the patient to the causal virus. The tumours are also radiosensitive, but facilities are rarely available. Surgery is restricted to dealing with tumour masses easily removable, e.g. ovaries.



FIG. 176.—Chloroma. (A. A. McConnell, F.R.C.S.I., Dublin.)

Chloroma (syn. myeloblastic reticulosarcoma) is a rare condition in which the bones of the face, and especially those of the orbits, are involved (fig. 176). It is a leukæmic condition, and at post-mortem the tumours present a bright green colour.

Boeck's Sarcoidosis.—This is *not* a variety of reticulosis. It is a local tissue response to an antigen, which, in Britain, is probably the tubercle bacillus. There is some evidence that in pine forests, e.g. Scandinavia and some parts of America, pine pollen may be the causative agent. A variety of tissues become infiltrated with epithelioid and giant cells. The lesion looks like a tubercle, but there is no caseation. Healing is by fibrosis.



FIG. 177.—Bone cysts in sarcoidosis.

The main surgical manifestations are enlargement of lymph nodes, lacrimal and parotid glands (p. 515). Bone cysts occur in the hands and feet in 6 per cent. of cases (fig. 177), but only if skin lesions are present (Geraint James). The stomach or bowel may be involved, and confused with leather-bottle stomach or Crohn's disease. Hypercalcaemia predisposes to renal calculi, and the neurologist will be interested in space-occupying and cranial nerve lesions.

Other manifestations include uveitis, pulmonary infiltration which may progress to fibrosis, right heart failure, and, most commonly, cutaneous nodules, papules, and keloid formation.

Kveim's test consists of an intradermal injection of sarcoid tissue, and if positive a dusky nodule of sarcoid tissue appears within a month. A biopsy of a skin nodule, lymph node, or any accessible tissue is diagnostic.

Cortisone or ACTH results in temporary improvement, and favourable results have been reported following the administration of mepacrine or chloroquine.

D. Geraint James, Contemporary. Physician, The Royal Northern Hospital, London.

Burrill B. Crohn, Contemporary. Gastroenterologist, Mount Sinai Hospital, New York City, U.S.A.

Morten Ansgar Kveim, Contemporary. Pathologist, Copenhagen, Denmark.

CHAPTER 10

INFECTIONS OF THE HAND AND THE FOOT

INFECTIONS OF THE HAND

IN 30 per cent. of cases the infection commences without known injury; it is then surmised that an epithelial crack from chapping or a forgotten prick is the portal of entry of the causative bacteria.

Pathology.—The initial lesion, a cluster of organisms surrounded by a zone of inflammation, may resolve—how often is not known. At least 80 per cent. of cases are caused by *Staph. aureus* whose toxin causes early death of tissue. A few cases are caused by *Strep. pyogenes*. The extent of the necrosis is governed by the toxigenicity of the organism and the ability of the tissues to swell without hindrance. Thus, sloughing is conspicuous in the fibro-fatty subcutaneous tissue of the palm and the pulp spaces of the digits, because in these situations the dermis is tethered.

Clinical Features.—Infections of the hand, which are encountered most commonly in manual workers and housewives, commence frequently as cellulitis, and unless the infection can be aborted, suppuration will follow. The early detection of the presence of pus and its accurate localisation are of cardinal importance. If the patient complains of throbbing pain, worse when the hand is dependent, and if *this pain interferes with sleep*, it is highly indicative of pus somewhere in the zone under suspicion. The inflamed area is pressed with a blunt-pointed probe or a matchstick, rather than with the finger; by this means the site of maximum tenderness can be determined accurately.



FIG. 178.—Pitting oedema of the dorsum is usually due to infection on the palmar side of the hand. (T. J. McNair F.R.C.S., Edinburgh.)

Oedema is an outstanding feature in all infections of the hand. Due to the rich network of lymphatics in the subcutaneous tissues of the dorsum of the hand and to the loose pliable skin covering this area, oedema is most in evidence on the back of the hand (fig. 178), irrespective of the site of the lesion. It is highly important that this oedema should be minimised by elevation of the forearm, as it is a potent cause of subsequent stiffness of the digits.

DIFFUSE SUBCUTANEOUS INFECTIONS

Lymphangitis.—Organisms, nearly always streptococci, gain entrance through an abrasion that may be microscopic. Within a few hours the adjacent portion of the hand becomes swollen and painful, and there is often considerable elevation of the temperature. Because superficial lymphatic

vessels pursue the shortest course to the dorsum, œdema, which comes on early, is most in evidence on the back of the hand. Later, red streaks, so characteristic of lymphangitis (fig. 166), can be seen coursing up the arm. Especially in lesions of the ulnar half of the hand, the first lymph node to become enlarged and tender is the epitrochlear. In a few instances of infection entering the middle finger, the first lymph node to become swollen is *above* the clavicle (fig. 179), in which case infection is liable to enter the general circulation and give rise to severe constitutional symptoms. The lymphatics of the thumb and index finger pass straight to the axillary nodes. Lymphangitis can occur (a) without any other demonstrable manifestation of inflammation, or (b) as an accompaniment of the entities to be described, particularly terminal pulp-space infection and fulminating tenosynovitis.

Deep lymphangitis may, or may not, be associated with red streaks passing up the arm. Indeed, its only evidence may be general malaise and fever with swelling of the whole hand and forearm and complete absence of localising features.

Treatment.—Lymphangitis responds well to the conservative measures detailed on p. 151, and provided the infecting organism is sensitive to the antibiotic employed, residual abscesses (e.g. an axillary abscess, from suppurating lymph nodes) are comparatively infrequent.

Cellulitis is the initial lesion of the fascial-space infections about to be described. In a proportion of cases, higher in loose subcutaneous spaces than in those more confined, the inflammation resolves. In the remainder a localised abscess forms. Incision during the stage of active cellulitis is highly mischievous. On the other hand, it is emphasised that fluctuation must not be awaited in infection of closed and deep spaces. Swelling, induration, and localised tenderness—a triad of signs that in the days of the pre-Listerian Masters was known as the stage of brawny swelling—now indicate that the time is ripe for incision.

Erysipeloid is described on p. 7.

LOCALISED CUTANEOUS AND SUBCUTANEOUS INFECTIONS

Acute paronychia is the most common infection of the hand (30 per cent.). Unlike the others, which occur more frequently in working men, paronychia is encountered in every walk of life, in both sexes, and from infancy to old age. The infection, which is *subcuticular*, arises from a hang-nail, careless nail paring, or a manicurist's unsterile instrument, the original infecting organism being a *staphylococcus aureus* in nearly every case. The inflammation commences beneath the eponychium.¹

Usually suppuration follows. Confined by the adherence

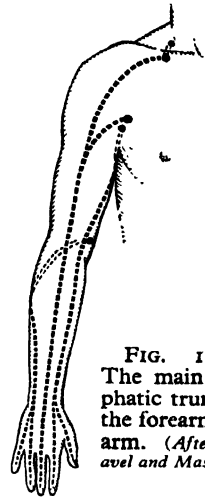


FIG. 179.—The main lymphatic trunks of the forearm and arm. (After Kanavel and Mason.)



¹ Eponychium is called by manicurists the 'nail cuticle'.

of the eponychium to the base of the nail, the pus tends to track around¹ the cutaneous margin (fig. 180) and in about 40 per cent. of cases it extends under the nail, where it can be seen through the nail as a whitish area. In neither situation does the pus extend deeper than the dermis (R. Pilcher). Untreated, spontaneous rupture occurs in about one-third of the cases, but suppuration continues, and not infrequently the abscess cavity becomes secondarily infected with *Esch. coli* and other organisms.



FIG. 180.—Paronychia. Often organisms enter through a 'hang-nail', as in this case.

Treatment.—Penicillin given very early may abort the infection but patients rarely present within the first few hours. Apart from these uncommon instances antibiotics should not be given (except for complications), as they do not alter the course of the infection (D. Bailey). Early operation is the correct treatment.

Operative treatment is conducted in a bloodless field under digital nerve-block anaesthesia (p. 171). It is unwise to use adrenaline (p. 171). By blunt dissection only the eponychium is stripped gently away from the base of the nail with closed forceps. This separation of the eponychium from the base and the sides of the nail must be complete: during this process pus will be evacuated and if there is an extension beneath the nail, this also will gain exit if pressure is exerted over the nail. All pus is wiped away with wisps of gauze; loose cuticle is cut away, and should there be a pocket under the corner of the nail fold, a wedge of overlying skin is removed to ensure healing from the bottom. The loose and ragged eponychium is then excised with delicate, sharp-pointed scissors. When pus has extended beneath the nail, the undermined portion must be excised with fine-pointed, but strong, scissors. 'Floating' nail is dead nail; consequently all unattached nail must be excised. Only when pus has extended beneath half or more of the width of the nail is excision of the proximal third of the nail required.

Chronic Paronychia².—The history is measured by months, rather than days, and the onset is insidious³; seldom does it follow acute paronychia. The infection is a mixed one. The lesions are often multiple. Classically washerwomen were especially prone to this condition; today the housewife who does not wear rubber gloves when 'washing-up' is the usual sufferer. Antibiotic therapy has little or no effect, but operative treatment following the lines detailed above is often successful if the infection is a bacterial one. In many cases, however, the infection is due to a monilia or yeast (p. 12). This can be settled by microscopical examination of scrapings and/or special cultures for fungi.

The best method of treating these indolent infections is by 1:500 Bradosol (domiphen bromide) in spirit, or Penotrane tincture (hydraphen) dropped into the nail-fold twice daily. The solution should not be applied with wisps of gauze or cottonwool, which are liable to be incompletely removed. In the later stages of treatment nystatin ointment (Squibb) cannot be bettered. When the pockets become filled with granulations the treatment is discontinued, and the hands must be kept as dry as possible until epithelialisation occurs. Superficial radiotherapy assists in clearing up resistant cases.

Localised superficial abscesses of the hand, including the digits, can be:

- (a) Intra-epidermal (purulent blister) (fig. 181 a).
- (b) Intradermal (fig. 181 b).

¹ The colloquial American term for paronychia is 'run around'.

² Called by some dermatologists (quite descriptively) chronic *per*ionychia.

³ A sharp look out must be kept for the occasional case resulting from neuropathy, e.g. syringomyelia.

(c) Subcutaneous (fig. 181 c).

(d) The superficial loculus of a collar-stud abscess (fig. 181 d).

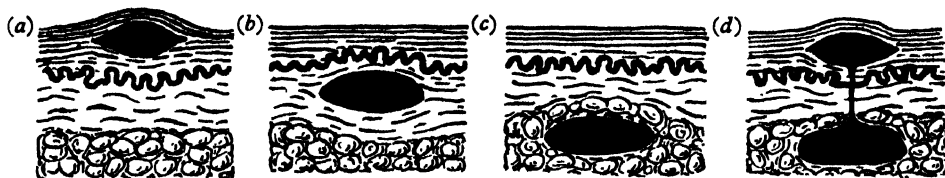


FIG. 181.—Superficial abscesses at various levels. (After R. S. Pilcher, F.R.C.S., London.)

The volar surface of the hands of manual workers is often covered with greatly thickened epithelium. Especially in such individuals, a subcutaneous abscess may burst through the dermis and extend in the layers of the epidermis (fig. 181 d), in which event it is impossible to differentiate it from a purulent blister until the deeper loculus has been discovered at operation.

Treatment.—The abscess is opened by transfixion. The resulting undermined flaps are cut away with scissors. After the unroofed cavity has been swabbed free from pus, it is explored for a sinus leading to a deeper loculus. If such is found, the communicating channel is stretched by inserting and opening the jaws of a small hæmostat.

Infection of the Dorsum and the Dorsal Spaces.—The most frequent cause of dorsal space infection is a boil of the overlying skin, or a penetrating wound. Infection of the *dorsal subcutaneous space* of the hand is common, as also is infection of the corresponding space in a proximal segment of a digit; but infection of the *dorsal subaponeurotic space* is rare. If swelling of the dorsum is accompanied by persistent tenderness, induration, and perhaps redness lasting a few days, fluctuation should not always be awaited.

Operation.—An incision about $\frac{1}{2}$ inch (1.3 cm.) long, which in this instance can be longitudinal, is made over the point of greatest tenderness.

Carbuncle of the Hand—The dorsal aspect of a proximal segment of a digit (fig. 182) and the dorsum of the hand are rather common sites for a carbuncle, infection often being carried thither by wiping the mouth or nose with the back of the hand. The condition is encountered much more often in males than females, because in males these areas are often hairy. A carbuncle in either of these situations is liable to involve an extensor tendon, and is slow to heal. The treatment of carbuncle is discussed on p. 100.



FIG. 182. — Carbuncle situated in the skin of the dorsal aspect of the proximal segment of the index finger.

Barber's Pilonidal Sinus.—Hair clippings have a bevelled extremity like the point of a hypodermic needle. Barber's pilonidal sinus is due to the customers' clippings penetrating the skin, most frequently the web between digits 3 and 4 of

the left hand. In the uninfamed state the lesion is marked by a small black dot with a collarette of epithelial scales around it; a cyst-like nodule can be palpated beneath the visible lesion. Recurrent attacks of subacute or acute inflammation (fig. 183) in the sinus cause the patient to seek relief. (For the common variety of pilonidal sinus see Chapter 41.)



FIG. 183.—Interdigital pilonidal sinus in a barber.

Human Bites.—Because the wound becomes contaminated with so many types of bacteria (including Vincent's organisms from the mouth), a human bite can prove very dangerous to life or limb. Although not strictly a bite, a common type of injury of this kind is an incised wound over the knuckles resulting from a clenched fist of one combatant striking the front teeth of his opponent. The joint is usually penetrated, but the track closes when the fingers are extended. In such a case the wound must be excised and, if the capsule has been penetrated, a portion of the capsule must be included in the debridement. Because of heavy contamination, primary closure of the wound resulting from excision of a human bite is inadvisable.

The chronic undermining ulcer of Meleney is due to a symbiotic infection by *micro-aerophilic non-hæmolytic streptococcus* and *hæmolytic staphylococcus aureus*, which produces a destructive lesion of the skin and subcutaneous tissues, usually of the dorsum. The gross appearance is an outer zone of erythema, an intermediate dark purple zone, and an inner zone of gangrenous skin. The centre of the lesion becomes a granulating ulcer. The lesion continues to spread until the patient dies, or the infection is brought under control by local and systemic bacitracin.

DEEPER LOCALISED INFECTIONS

Infection of the terminal pulp space (*syn.* Felon) is the second most frequent infection of the hand (about 25 per cent. of all cases). The index finger and the thumb are affected most often. The origin of the infection is usually a prick.

Surgical Anatomy.—The deep fascia, which is attached to the thin skin of the distal flexion crease, fuses with the periosteum just distal to the insertion of the deep flexor tendon, thereby closing the terminal pulp compartment at its proximal end.

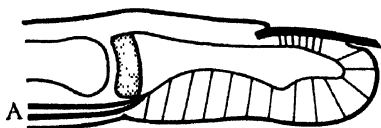


FIG. 184.—Distal pulp space to show how its proximal end is closed by attachment of the deep fascia and by the fibrous septa. The blood-supply to the epiphysis does not traverse this area. A = tendon sheath and deep fascia.

Through the space, which is filled with compact fat, feebly partitioned by fibrous septa, run the terminal branches of the digital artery. Thrombo-arteritis of these vessels accounts for the frequency with which osteomyelitis complicates infection of this closed space. The basal plate of the epiphysis is rarely involved.

Clinical Features.—Dull pain, worse when the hand is dependent, and swelling are the first symptoms. Forty-eight hours later there are severe nocturnal exacerbations of throbbing pain, interfering with sleep. Light

pressure over the affected pulp increases the pain. Frequently the corresponding regional lymph node is enlarged and tender. If the pulp is indurated and has lost its normal resilience, pus is present (fig. 185). Untreated, the abscess tends to point towards the centre of the pulp beneath a patch of devitalised skin. A collar-stud abscess then occurs; if still untreated, the abscess bursts.

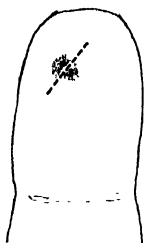


FIG. 186.—Incision for draining a pulp abscess. It is essential to drain the deep loculus of a collar-stud abscess, if such is present.

Treatment.—Operation without delay is the rule except in the early stages, when there is no localisation and cellulitis only is present. Large doses of penicillin must then be given. Only in those rare cases where resolution occurs should operation be withheld.

Operation.—A short incision (fig. 186) following one of Langer's lines (which are well defined in this situation) is made through the skin at the point of greatest tenderness. The beginner is warned not to be beguiled by entering only the superficial loculus of a collar-stud abscess. Removal of slough, which is frequently present, is most desirable, but great care must be taken not to traumatise the periosteum.

Osteomyelitis of the terminal phalanx is a fairly common sequel of terminal pulp-space infection (fig. 184). Occasionally, in a case of some standing, that part of the bone bereft of its blood-supply is found to be loose, and can be lifted out of the abscess cavity at the time of the operation. More often the sequestrum separates some weeks after the abscess has been opened, in which event the wound continues to discharge. Repeated radiographs and probing will indicate when the sequestrum has separated. Only then must it be removed, after which healing will proceed apace. In the case of a child, regeneration of the diaphysis is possible, provided the periosteum is relatively undamaged. In the adult no regeneration occurs, and the patient is left with a shortened terminal phalanx covered by an ugly curved nail.

'Aseptic' subcutaneous felon (fig. 187) is due to infection by the virus of herpes simplex (*Herpesvirus hominis*). Hospital nurses are particularly, indeed almost exclusively, attacked by this infection, and what is even more remarkable is that the nurses so infected at St. George's Hospital, London, had nearly all handled catheters used for aspirating respiratory secretions (H. Stern). The relative absence of constitutional symptoms and of regional adenitis makes a clinical diagnosis of this new entity possible. Complete resistance to all forms of treatment decrees non-intervention, but the digit must be kept covered, otherwise secondary staphylococcal infection is probable.

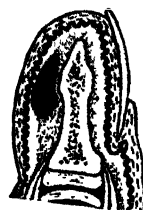


FIG. 185.—The location of pus in terminal pulp-space infection.



Fig. 187.—'Aseptic' subcutaneous felon of the thumb. (Dr. H. Stern, London.)

Apical space infection arises from a prick (including a splinter) beneath the nail, causing infection of the space between the subungual epithelium and

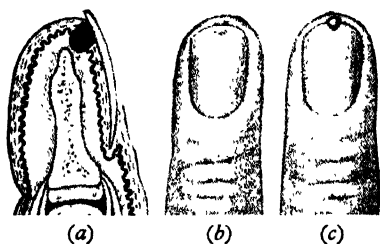


FIG. 188.—(a) Showing the anatomical relations of an apical abscess. (b) Apical abscess; clinical presentation. (c) V-exposure for evacuating the contents of the abscess.

the abscess is excised also (fig. 188 c). The amount of pus and debris evacuated is surprisingly small; commonly the abscess cavity extends down to the bone, but osteitis is unusual. Following the operation, relief of symptoms is immediate and the wound heals in under a week.

Infection of the Middle Volar Space of a Digit.—The fibro-fatty tissue occupying this space is more loosely packed than that of the terminal pulp space. The middle volar space is separated above and below by fibrous partitions while, like the proximal space, it is shut off from the dorsal cellular tissue by fibrous septa extending from the skin to the periosteum.

Clinical Features.—Infection of this space is much less common than that of the terminal pulp space. The finger is held in semi-flexion. In about one-third of cases attempts to straighten it are painful. There is tender induration over the space, while the soft tissues of the terminal and proximal segments, although swollen, are neither tender nor indurated. In late cases frequently a purulent bleb appears in the distal flexion crease (fig. 189). In early cases it is difficult to distinguish infection of the middle volar space from infection of the underlying flexor tendon sheath; however, in the former, extreme tenderness over the proximal end of the tendon sheath is completely lacking.

Infection of the Proximal Volar Space.

—This space is well partitioned from the middle volar space but it communicates freely with the corresponding web spaces. Once localisation has occurred (fig. 190) infection of the proximal volar space is comparatively easy to diagnose, and frequently the swelling is asymmetrical because of concomitant involvement of a web space (see below).

Operation.—After pus has become localised in either of the above spaces, it should be evacuated through a transverse incision made at the site of greatest tenderness. When the diagnosis is uncertain (localised tenosynovitis cannot be excluded) the space should be explored through a lateral longitudinal incision.

Infection of a Web Space

Surgical Anatomy.—The web spaces are the three triangular regions between the dorsal and volar skin filled with loose fat that bulges between the divisions of the palmar fascia (fig. 191). The spaces, when filled with pus, straddle the deep transverse ligament; consequently, although most of the pus is volar, the abscess points dorsally.

the periosteum (fig. 188 a). The lesion (fig. 188 b), which is exquisitely painful, gives rise to comparatively little swelling. Tenderness is greatest just beneath the free edge of the nail, and pus comes to the surface here or beneath the nail. The condition is often confused with a terminal pulp-space infection, or when there is redness around the nail, with paronychia.

Operation.—A small V is removed from the centre of the free edge of the nail, and a little wedge of the full thickness of the skin overlying



FIG. 189.—Abscess of (a) the middle and (b) the proximal volar spaces. Showing direction of spread.



FIG. 190.—An abscess of the proximal volar space. (After M. Iselin.)

Ætiology.—The infection arises (1) from a skin crack, (2) from a purulent blister or from beneath a callosity on the forepart of the hand, or (3) via a lumbrical canal from an abscess in a proximal volar space.

Clinical Features.—As the constitutional symptoms are severe, patients with this condition are often seen before localisation of the infection has occurred. At this stage there is oedema of the back of the hand, and although the condition can be strongly suspected from the location of the tenderness, a precise diagnosis cannot yet be made. The patient should be in bed with the arm splinted and elevated by suspension. Penicillin is administered intramuscularly. Once localisation has occurred, the signs of infection of a web space become manifest.



FIG. 192.—Web-space abscess. Note the separation of the related digits.

Localising Signs.—The base of one finger is swollen, and in severe cases the fingers immediately adjacent to the space are separated. Often there is a fan-shaped blush extending from the web on to the dorsum and a small area of purplish discoloration of the skin over the affected space (fig. 192). The maximum tenderness is found in the web and on the anterior surface of the base of one of the fingers extending a short way into the palm. Untreated, pus can track across the base of the finger into an adjacent web space, and also along the sides of the proximal segments of the digits related to the infected web.

Operation.—A transverse incision is made on the palmar surface over the affected web space (fig. 193). The incision must be deepened very cautiously until the subcutaneous fat is reached. Only a few strands of palmar fascia need to be divided, and if pus does not flow it is sought with a probe or director. If the diagnosis is correct, probing is soon rewarded by a gush of pus. The opening is enlarged to reveal an abscess cavity, often the size of a thimble. The edges of the wound are cut away, so as to leave a diamond-shaped opening (fig. 193). When the abscess communicates with a dorsal pocket, a counter-incision is advisable. If, as sometimes happens, there is a twin web abscess (one on each side of the digit), each space must be opened, but there is no need to slit the communicating channel.

Deep Palmar Abscess

An abscess beneath the palmar fascia is a serious, but rare (about 1 per cent), infection of the hand.

Ætiology.—The infection can arise as (1) a penetrating wound; (2) infection via the blood-stream of a hæmatoma in this situation; (3) as a complication of suppurative tenosynovitis (p. 168).

Clinical Features.—As a rule the patient is a manual worker. At an early stage there is intense throbbing pain in, and deep tenderness of, the palm of the hand. Almost from the commencement this is accompanied by obvious cedematous swelling of the dorsum of the hand, which increases rapidly to become greater than that seen in any other infected lesion of the hand—so great as to give rise to what is known as 'the frog hand'. The fingers are held in a flexed position because the palmar fascia



FIG. 191.—The three web spaces between the divisions of the palmar fascia are floored by the transverse ligaments.

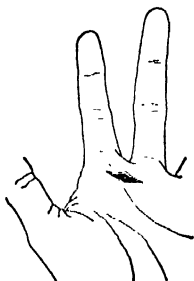


FIG. 193.—Incision for draining a web-space abscess.

is more relaxed in this posture. Extension of the metacarpophalangeal joints is very painful, but extension of the interphalangeal joints is both painless and free. This is a most valuable observation in distinguishing the condition from suppurative tenosynovitis. The temperature is raised, and regional lymphadenitis is commonly present.

As tension mounts, the normal concavity of the palm becomes flattened; a time is reached when the imprisoned pus erodes and bursts through the palmar fascia, then suddenly the intense pain passes off, but the palm becomes slightly convex. If the patient is seen for the first time after this happening, there is no means of distinguishing a deep palmar abscess with a collar-stud extension from an abscess of a subaponeurotic space, except by operation.

Operation.—Under general anaesthesia (or regional block of the median and ulnar nerves at the wrist), a central transverse incision is made in the line of the flexion crease passing across the middle of the palm. Should pus be encountered beneath the aponeurosis, the floor of the abscess (the palmar fascia) must be probed systematically for a sinus leading to the deeper plane. In other circumstances the palmar fascia is divided in a longitudinal direction (to avoid digital nerves and blood-vessels). Pus is mopped up. Continued free drainage is ensured by trimming, with scissors, the skin edges as well as those of the incision in the palmar fascia. This obviates premature healing, which otherwise is prone to occur.

ACUTE SUPPURATIVE TENOSYNOVITIS

Usually infection of the sheath of a flexor tendon is due to bacteria introduced by the point of a needle or other sharp object penetrating the tendon sheath. Exceptionally the sheath is infected by extension from its terminal pulp space, in some cases from the scalpel transgressing the hallowed ground of the septum that closes the proximal end of the space (fig. 184).

Acute fulminating tenosynovitis involves the whole sheath rapidly, and nearly always the infecting organism is the *staphylococcus aureus* or the *streptococcus hæmolyticus*. The classical local signs are:

1. Symmetrical swelling of the entire finger.
2. Flexion of the finger (the 'hook' sign) with exquisite pain on extension. (Unfortunately this sign is not always present, and it occurs also in infection of the middle pulp space (M. Iselin)).
3. Tenderness over the infected sheath, especially over its proximal *cul-de-sac* (fig. 194).



FIG. 194.—The flexor tendon sheaths. This typical arrangement is present in 75 per cent. of cases.

Due to very early administration of penicillin by the patient's doctor, cases of fulminating tenosynovitis have become less common. The infection is often either aborted before the whole sheath has become implicated, indeed, before the diagnosis of tendon-sheath involvement can be made with assurance, or the infection becomes limited by adhesions to a portion of the sheath.

Localised suppurative tenosynovitis.—Swelling and tenderness are limited to one portion of the digit, rendering confident diagnosis prior to exploration difficult. Infection of the ulnar bursa (fig. 194) is characterised by:

1. Oedema of the whole hand, especially the dorsum, due to lymphatic spread.
2. Moderate swelling of the palm.

3. Sometimes a fullness immediately above the flexor retinaculum.

4. The flexed fingers resist extension, the maximum difficulty being experienced in the little, and the least in the index finger.

5. Especially valuable is Kanavel's sign: the area of greatest tenderness is over that part of the ulnar bursa lying between the transverse palmar creases (fig. 195).

It should be noted that the ulnar and radial bursæ intercommunicate in 80 per cent. of cases, and often when an untreated infection of one has persisted for more than forty-eight hours, the other becomes involved also. In no less than 25 per cent. of cases the tendon sheath of the index, or the middle, or the ring finger communicates with the ulnar bursa, which is a fact of great surgical importance.

Infection of the radial bursa (fig. 194) is distinguished by:

1. The distal phalanx of the thumb is held in flexion, with rigidity and inextensibility of the interphalangeal joint. The other digits can be extended fully.
2. Tenderness over the sheath of the flexor pollicis longus.
3. Sometimes swelling just above the flexor retinaculum.

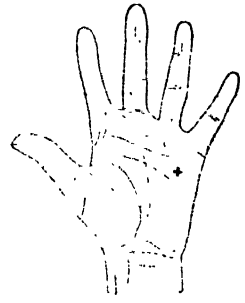


FIG. 195.—Kanavel's sign.

Treatment of Acute Infected Tenosynovitis.—In very early cases one mega-unit of penicillin is given. The forearm and hand, the latter being placed in the position of rest (fig. 198), are splinted and elevated.

Clinical re-examination is made every six hours. Only when the local, as well as the general, response to conservative measures is unquestionable, is non-operative treatment continued, because, in cases where decompression is not carried out within forty-eight hours sloughing of some part of the tendon commonly occurs. Therefore, as soon as the diagnosis can be established with confidence, adequate incision and drainage under antibiotic cover is the best method of treatment.

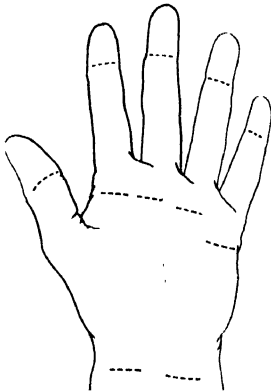


FIG. 196.—Sites of incision for treating acute suppurative tenosynovitis. The sheath is also exposed at the site of the initial trauma when such is discernible.

Operation.—When the site of puncture of the integument is visible, or has been indicated without hesitation by the patient, the first incision is made in a transverse direction directly over the tendon sheath. When there is no certain information as to where the sheath was punctured, the more distal of the two relevant incisions shown in fig. 196 is made, and deepened until the fibrous portion of the sheath is displayed. This is divided and the thin, bulging theca comes into view. Some of the fluid within it is aspirated and sent for bacteriological examination; the theca is then incised in a transverse direction and its edges are pared so as to form a diamond-shaped opening.

Pressure is now exerted over that portion of the sheath proximal to the incision, and if this results in a gush of hazy exudate, a second relevant incision (over the cul-de-sac) is made and similar steps to prevent premature closure of the wound are taken. Employing a fine whistle-tipped ureteric catheter, the length of the sheath is irrigated with a solution of penicillin, 10,000 units per ml. It should be noted that if the original puncture wound is approximately mid-way along the sheath there will be three transverse incisions.

Complications of Suppurative Tenosynovitis

Involvement of the Forearm from the Hand.—When a radial or ulnar bursa, distended with pus, bursts, pus travels up the forearm between the flexor profundus



FIG. 197.—Incisions for draining the space of Parona.

ventrally and the pronator quadratus and interosseous membrane dorsally. It is here, in the space of Parona, that a quantity of pus can collect without giving rise to much swelling. There is, however, brawny induration above the wrist, unless the original lesion has been incised and continues to discharge pus. Therefore, in cases of infection of the radial or ulnar bursa, if pus can be expressed by pressure over the wrist at the time of operation or subsequently, it is essential

that the forearm be drained by making the incisions shown in fig. 197 and deepening them until the periosteum is reached. A hæmostat is then thrust beneath the flexor tendons, and the jaws of the forceps are opened, as a result of which the proximal extremity of the infected bursa (or bursæ) is ruptured thoroughly into the space beneath the flexor tendons.

Continuation of Suppuration.—Provided the principles set out above concerning early decompression of infected tendon sheaths have been followed, continued suppuration is rare. If it occurs, the first point to consider in most situations is the possibility of extension of the infection to a fascial space or another tendon sheath. Should suppuration continue for fourteen days, the hand should be radiographed for evidence of bone necrosis. In relevant cases, the possibility of a non-opaque retained foreign body should also be borne in mind. Sloughing tendon is a potent source of prolonged suppuration and much time will be saved by excising the diseased portion, care being taken to anchor its proximal end by sutures to prevent the cut end being carried into the forearm by muscular contraction, and thereby spreading infection.

Suppurative arthritis in a related joint occurs occasionally as a complication of suppurative tenosynovitis. In these circumstances timely amputation of any digit except the thumb will reduce the period of disability (S. Bunnell).

A Stiff Digit Results.—In the case of a finger, it should be remembered that in many walks of life total amputation of a digit is less of a handicap than a stiff finger, but amputation should seldom be undertaken until the infection has subsided completely. In the case of a thumb, the surgeon's watchword for infection as well as trauma is always 'Save all possible'.

Paralysis of the Median Nerve.—When signs of median nerve palsy develop in a case of infection of the hand, early decompression of the carpal tunnel by severing the flexor retinaculum is recommended (D. Bailey). In these circumstances, involvement of the median nerve is due to compression of the nerve by the distended radial or the ulnar bursa, or (more frequently) by both bursæ, which of course must be drained thoroughly at the same time.

GENERAL PRINCIPLES OF TREATMENT IN ALL CASES OF INFECTIONS OF THE HAND

The five principles in the treatment of infections of the hand can be summarised as follows :

- (i) Antibiotic therapy.
- (ii) Provision of rest and elevation to the affected limb.
- (iii) Early recognition of the presence of pus and its accurate localisation.
- (iv) Evacuation of pus and, in the case of fascial spaces, debridement of the walls of the abscess cavity.
- (v) Adequate after-treatment.

To consider these principles in more detail:

Antibiotic Therapy.—Except in cases of established acute paronychia or cases of well localised superficial infection, antibiotic therapy is given without delay. Because in over 90 per cent. of cases of infections of the hand the original infection is caused by staphylococci or streptococci, or both, penicillin is still the antibiotic of choice.

Should penicillin resistance be suspected, and it should always be suspected in a case of anyone working within the precincts of a hospital (e.g. a nurse), a penicillin

that is effective against penicillinase-producing staphylococci, e.g. methicillin, should always be employed in the first instance. The final choice of antibiotic depends upon the sensitivity tests. It is futile, damaging, and often disastrous to rely on antibiotics when suppuration has occurred. If there is pus in any part of the hand it must be evacuated.

Rest and Elevation of the Hand.—If it is considered possible that resolution will occur, and also following operation, the hand must be placed in the position of rest (fig. 198). When it is anticipated that in all probability some portion of the hand will become stiff, as soon as the ultra-acute



FIG. 198.—The position of rest taken up by an acutely inflamed hand. The index finger is not flexed as much as the others. (After Wood Jones.)

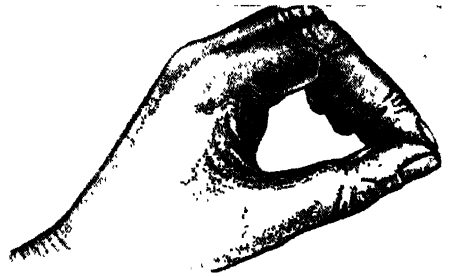


FIG. 199.—The position of function.

stage has passed the digits should be arranged in the position of function (fig. 199). For ambulatory patients a light plaster-of-Paris slab, moulded to fit the volar surface of the hand and forearm, cannot be bettered. In addition, the forearm is supported in a sling as high as possible towards the opposite shoulder, in order to lessen oedema. For in-patients a Cramer wire splint, which is suspended readily, is both efficient and comfortable.

Full elevation in the manner shown (fig. 200) decreases oedema and lessens the throbbing pain.

These remarks concerning elevation do not apply to cases of uncomplicated paronychia and minor superficial abscesses; for these an ordinary sling is all that is required.

In all cases rest for an inflamed hand should be insisted upon. When the acute phase has abated, gentle voluntary movements are encouraged.

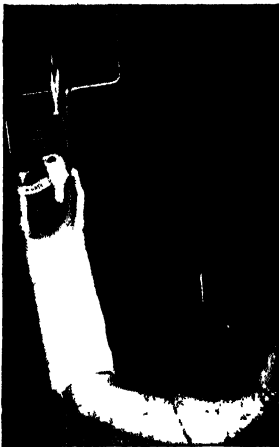


FIG. 200.—Suppurative tenosynovitis being treated by immobilisation and elevation.

Anæsthesia.—For the distal part of the finger regional anæsthesia is employed, using 2 per cent. procaine or xylocaine (*without adrenaline*, as vaso-constriction is harmful). After raising a wheal, the hypodermic needle is introduced at the relevant dual points shown in fig. 201. While injecting the anæsthetic solution the needle is advanced distally and forward until it is judged that the digital nerve has been reached; $\frac{1}{4}$ ml. of the anæsthetic solution is deposited here. The procedure is repeated on the contralateral aspect of the affected finger. By the addition of 300 international units of hyaluronidase to a 5-ml. bottle of either of these solutions (which

thereafter must be stored in a refrigerator to prevent deterioration of the hyaluronidase), the rapidity of action of the anæsthetic solution is enhanced, and oedema, both pre-existing and that caused by the injection of the fluid, is absorbed rapidly. In the case of an abscess of the hand itself, a general anæsthetic is adminis-

tered, or a regional block of the median and ulnar nerves at the wrist is undertaken. On no account should a short general anæsthetic, e.g. nitrous oxide gas, be employed. Complete muscular relaxation and ample time are most desirable when operating in this area.



FIG. 201.—Points of puncture for anæsthetising a digit.

A **bloodless field** is essential. Only in the absence of bleeding can the exact site and extent of the lesion be determined and damage to tendon sheaths and nerves be avoided. The cuff of a sphygmomanometer is applied to the upper arm. The limb is then elevated for two minutes, after which the bag is inflated to a pressure of 40 mm. Hg. above the systolic blood-pressure.

Operation is undertaken at a time when there is a high penicillin level in the blood. With the exception of tendon-sheath infection, it is insufficient merely to evacuate the pus. The operation must be meticulous. Slough is removed unless it is densely adherent and, what is extremely important, granulations are abraded by gauze or scooped away with a curette, avoiding the latter in situations where it might damage the periosteum or a tendon sheath. Only after granulation tissue

has been removed, leaving the walls clean and oozing blood, will the injected antibiotic from the blood enter the cavity freely. Provided every nook and cranny has been attended to in this manner, no drainage material is employed, for no further pus is expected to form; merely a little serum, at first containing blood and dead bacteria is all that oozes from a cavity thus treated. This lessens in amount about the third day, when quick healing is to be expected.

After-treatment of Serious Infections of the Hand.—In all cases dry dressings are employed. The dressings are changed at the end of twenty-four hours after operation. Thereafter often an interval of two days can elapse between re-dressings. The patient must be instructed not to get the dressings wet. These instructions differ only in the case of paronychia; in this instance the patient is instructed to wash the hands frequently, dry them thoroughly on a towel kept for the purpose, and re-apply the dressing himself. Physiotherapy and exercises form an important part of the late after-treatment. In rare cases with persisting deformity, rehabilitation for a new job may be necessary.

INFECTIONS OF THE FOOT

Especially in countries where some of the inhabitants go barefooted, infections of the foot are commonplace; they also occur in the shod. In each and all of the infections of the foot about to be described it will be assumed that the reader, remembering the lymphatic drainage of the foot, will examine the lymph nodes of the groin and, in relevant cases, those of the popliteal space. He should also examine the urine for sugar. Furthermore, he must remember that arterial disease may present as an infection of the foot.

The prelude to the treatment of any of these infections is thorough washing of the foot with soap and water or, preferably, a detergent, applied with sterile gauze or cottonwool. Except in trivial infections, bed-rest with elevation of the foot must be insisted upon until the inflammation has subsided.

Infected blister is one of the most common infections of the foot. When the patient's temperature is normal and the content of the blister appears doubtfully purulent, the blister can be aspirated. If the fluid is opalescent, the patient should be given penicillin and the aspirated fluid sent for bacteriological examination. When a blister is frankly purulent it should be incised.

Paronychia of the great toe is also common. It often occurs as a complication of an ingrowing toe-nail. An abrasion of the eponychium with contaminated scissors is also a frequent cause. The clinical features and

treatment do not differ from those of a paronychia in a finger or thumb, except that when an ingrowing toe-nail is present it is necessary to deal with it radically (p. 175). Washing, as described above, is carried out each time the wound is dressed.

Infected adventitious bursa beneath a corn is usually the result of improper chiropody. There are signs of inflammation around the corn, the slightest pressure on which evokes excruciating pain. Drainage is accomplished by paring the corn with a sterile scalpel until pus exudes.

Terminal pulp space infection is rare as compared with that of the hand. When it occurs in the foot, usually it is the great toe that is affected.

Infected Bursa over a Hallux Valgus (p. 346).

Infection of a Web Space.—The web spaces of the foot are four in number, the space between the great toe and the second toe being the largest. The clinical features of infection of this space are similar to those of the hand, but extension from the plantar to the dorsal aspect of the web occurs earlier, and with great regularity. The treatment also is similar. In a diabetic, orthodox drainage is so disappointing that disarticulation of the relative toe, leaving the flaps unsutured, is recommended (fig. 42).

Infection of a Plantar Interdigital Subcutaneous Space.—There are four interdigital subcutaneous spaces that lie between the five digital slips of the central aponeurosis (fig. 202). Infection of one of these spaces is very common among coolies who work barefooted, especially in urban areas. As a rule the patient states that a sharp stone, a nail, or a thorn penetrated the sole. The patient complains of increasing pain between the shafts of the two metatarsals that bound the infected space. Soon he is unable to walk, and the constitutional symptoms are moderately severe. Exquisite tenderness located over the infected space proclaims the diagnosis. When pus decompresses itself between the two bones into the dorsal subcutaneous space, localisation is more difficult. Drainage must be placed well away from the weight-bearing area, and consequently an incision similar to that used for drainage of a web space is advised, after which a hæmostat is directed into the cavity filled with pus, and its jaws are opened. If the dorsal subcutaneous space is involved, a counter-incision should be made.

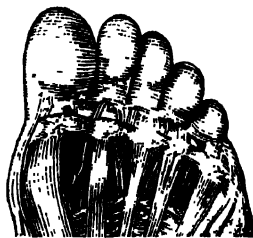


FIG. 202.—The four interdigital subcutaneous spaces, which are separated from the four corresponding web spaces by the transverse plantar ligament.

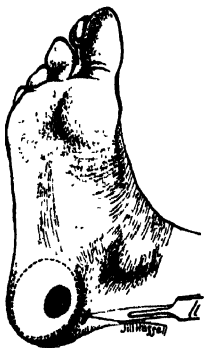


FIG. 203.—The fibrous septa of an abscess of the fat-pad of the heel are so tough as to require division with a scalpel.

Infection of the Heel Space.—The infection is intradermal in one-third of cases, a few of these being a collar-stud extension from a deeper plane. Two-thirds of cases are due to infection of the fat-pad of the heel, which is situated in the subcutaneous portion of the posterior third of the sole. The portal of entry usually is a crack in the overlying calloused skin, and seldom, as one would think, from treading on a thorn or similar object.

Steadily increasing throbbing pain, severe enough to interfere with sleep, is the leading symptom. The patient dare not put his heel to the ground. Swelling of the soft tissues that cover one or both sides of the calcaneus is present, and in severe cases œdema of the ankle becomes manifest. Tenderness over the space leaves no doubt as to the diagnosis. As a rule, by the

time the patient presents, the abscess is ready for incision, which is made through the medial or lateral side of the heel, so that the scar does not come to lie on a weight-bearing area. Fibrous septa within the abscess of the fat-pad need division with a scalpel (fig. 203). The lips of the cutaneous incision should be trimmed elliptically to prevent premature closing of the skin.

Deep Plantar Abscess.—The central plantar space situated deep to the plantar fascia is arranged like an apartment house of four stories, each of which is occupied by the muscles that constitute the flexors of the toes. Infection of the various floors becomes increasingly less common as one proceeds from the ground floor, upwards. For drainage of the central plantar space an incision parallel to, and just above, the medial border of the foot in the neighbourhood of the instep, is made (fig. 204).



FIG. 204.—Incision for draining the central deep plantar space.

Infections of the Dorsum of the Foot

The *dorsal subcutaneous space* usually is infected by extension from a subcutaneous interdigital space or a web space, while the *dorsal subaponeurotic space* is infected either from a direct puncture or from involvement by extension from the deep plantar space. To drain the former space the incision should be placed distal to the dorsal venous arch, but in the line of the digital vessels and nerves, in order to avoid them. To drain the latter space it is best to confirm the presence of pus by attempting aspiration, and if diagnostic aspiration is positive, to make a longitudinal incision alongside the needle.

Leprosy not infrequently attacks the toes, and the initial manifestation may simulate a chronically infected corn (fig. 205). Leprosy is discussed on p. 24.

Madura foot (*syn. mycetoma pedis*) is a chronic granulomatous disease encountered especially in tropical countries, notably in certain parts of India or Africa, but with increasing frequency in territories where hitherto it has been unrecognised, such as the Southern United States, South America, and Cuba. Most cases are caused by a filamentous organism (*Nocardia maduræ*) resembling actinomycetes and abounding in road dust. In nine out of ten cases the organism gains entrance through a prick in those who go about barefooted. The first manifestation is a firm, painless, rather pale nodule. Soon other nodules appear. Later, the nodules become surmounted by vesicles which burst to form discharging sinuses. In the watery discharge granules can be discovered, sometimes only with perseverance. The granules may be yellow, red, or black. In 'black' madura foot, as it is called, spread is mainly in the subcutaneous plane; in the yellow and red varieties the infection burrows deeply, and bone necrosis ensues. As in actinomycosis, there is no lymphadenitis, but unlike actinomycosis, dissemination to other parts of the body does not occur. Gross swelling of the foot (fig. 206) with flattening

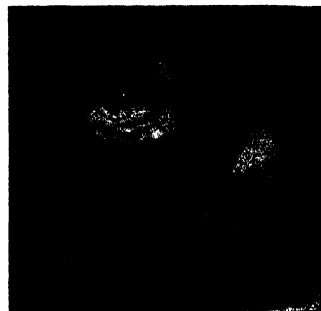


FIG. 205.—Leprosy of the little toe. (G. D. Adhva, F.R.C.S., Bombay.)

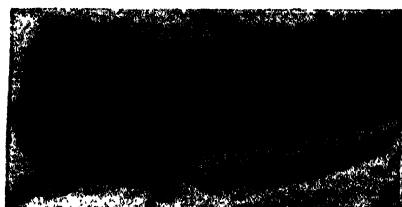


FIG. 206.—Madura foot. (Dr. M. M. Schapiro, Honduras.)

or convexity of the instep is characteristic. Sooner or later secondary infection supervenes, with rapid deterioration of the local condition.

Treatment.—A wide-spectrum antibiotic to deal with secondary infection, fol-

lowed by a prolonged course of dapsone (100 mg. b.d.) improves many patients, and may obviate or postpone the necessity for amputation (Cockshott).

INGROWING TOE-NAIL

Ingrowing toe-nail (*syn.* embedded toe-nail) of the big toe, usually results from encasing sweaty feet in tight shoes, and is encouraged by cutting the nail short and convexly. The side of the nail curls inwards and grows to form a lateral spike, which causes a painful infection of the overhanging nail fold.

Treatment.—Conservative measures can be successful, but much depends upon the patient's willingness to help himself. The overhanging nail fold is pushed away and reduced in size by daily packing of the lateral groove with a wisp of gauze soaked in a mild antiseptic. The centre plate of the nail is thinned slightly by filing; this encourages the nail to become flat. The end of the nail is cut straight across or concavely. The corners must not be cut back; it is necessary for them to extend over the pulp.

Operation.—The nail is like a letter protruding from a shallow envelope. The envelope is the germinal matrix and this must be included in radical removal of the affected side or the whole of the nail. Failure to remove the corners of the germinal envelope (fig. 207) will result in the recurrence of a nail spike. The technique (Fowler) is portrayed in fig. 208. A tourniquet is essential in order that the germinal envelope may be seen and dissected out in its entirety.



FIG. 207.—The relation of the nail to the nail fold and nail walls. Note the corners of the germinal envelope. (Courtesy *British Journal of Surgery*.)



FIG. 208.—Technique of total excision of the germinal matrix. A, Skin incisions. B, Skin-flaps dissected and retracted. The epithelial layer on the deep surface of the flaps is left attached to the matrix. C, Longitudinal section showing the block of tissue to be removed. D, Skin-flaps sutured to cover the raw area. E, Segmental excision of the germinal matrix. The skin-flaps have been raised preparatory to excision of a segment of the germinal matrix.

(Courtesy *British Journal of Surgery*.)

Onychogryphosis is a thickened and crooked overgrowth of a toe-nail, usually that of the big toe. Trauma and fungus infection are implicated as causes. It occurs in elderly people, especially if bed-ridden, and it may become so curled as to resemble a ram's horn. A Gigli saw will cut through the base and the remainder can be controlled by filing. If necessary the nail-bed and nail are removed as above.

CHAPTER II

INJURIES TO BONES

JOHN CHARNLEY

Contusion of bone is encountered where bones are subcutaneous, and is therefore most commonly seen on the shin. A small subperiosteal hæmatoma is produced, which usually subsides rapidly, leaving no residual mark. In a few cases a small bony boss may develop in the hæmatoma, but this will cause no trouble; sometimes the hæmatoma may become infected from an overlying abrasion.

If necessary, resorption is helped by rest and a pressure dressing.

FRACTURES

Fractures are caused by two types of mechanical violence—*direct* violence, as by a local blow; and *indirect* violence, as by a bending or twisting force applied to the limb at a point remote from the site of the fracture.

VARIETIES OF FRACTURE

A large number of descriptive adjectives are used to qualify and describe fractures; the most important of these are:

(1) *Simple* (*syn.* 'Closed')—meaning that the fracture hæmatoma is not exposed to infection. A fracture may still be 'simple' or 'closed', in the presence of a large skin wound, if the wound does not actually communicate with the fracture.

(2) *Compound* (*syn.* 'Open')—meaning that the fracture communicates with the external air. Infection is the obvious danger. Indirect violence extrudes a bone-end through skin which was intact at the moment of fracture; direct violence, as by a missile or machinery, breaches the skin before the fracture is sustained. The former is usually less contaminated by bacteria, clothing, or road-dirt than the latter.

(3) *Comminuted*.—The bone is broken into three or more parts.

(4) *Transverse or Oblique*.—Descriptive of the line of fracture.

(5) *Spiral*.—Sometimes mistaken for oblique. Interesting to recognise in that the violence causing it can be deduced accurately, i.e. torsion.

(6) *Greenstick*.—A type of fracture only occurring in children. The bone bends and stays bent; one cortex remains intact, while the other crumples or cracks (fig. 209).

(7) *Impacted*.—One fragment is driven into the substance of the other fragment, so that no abnormal mobility will be evident. Impacted fractures are usually seen where a shaft of cortical bone joins the cancellous bone forming the expanded extremity of the shaft; the dense cortical bone is thus impacted into soft cancellous bone. Two cancellous fragments can impact one into the other, as in fractures of the vertebræ, but two cortical fragments cannot impact.

(8) *Complicated*.—Associated with nerve or vascular injuries, or injuries of adjacent viscera.

(9) *Pathological*.—Occurs when disease renders the bone abnormally brittle, as in Paget's disease, or has eroded the bone, as in secondary carcinoma.



FIG. 209.—Greenstick fracture of femur.

(10) *Fatigue* fractures are rare fractures where a bone, which is otherwise apparently normal, develops a minute crack in the course of ordinary use, e.g. 'march' fracture of the second or third metatarsal bones (p. 229).

THE SIGNS OF FRACTURE

There are five clinical signs indicating a fracture in addition to the pain, swelling, and bruising which can be present without a fracture:

- (1) Deformity.
- (2) Abnormal mobility.
- (3) Crepitus.
- (4) Loss of function.
- (5) Local bone tenderness.

In a fracture of the midshaft of the femur or the humerus, all of these physical signs will easily be elicited, but there are many other examples where only some of these five signs are represented. Which is the most important sign of the five? Let us examine each sign in detail.

(1) *Deformity*.—Though deformity is usually characteristic of a fracture, a bone can be broken without any deformity. Deformity means that the soft parts, the fascia, intermuscular septa, etc., have been so extensively torn that the fragments are able to separate from each other. The fragments can be *angulated* in relation to each other, *displaced laterally*, or they can even *over-ride*, in which case *shortening* will be present. If the violence has been expended entirely on breaking the bone, the soft parts will be intact and the fracture will often remain *undisplaced*.

(2) *Abnormal Mobility*.—This is characteristic of a fracture; the limb

wobbles about and can be moved in a direction which is anatomically impossible. Abnormal mobility is, however, absent in *greenstick* and *impacted* fractures.

(3) *Crepitus*.—This is the grating sensation imparted to the examining fingers when the bone-ends move against each other. It is an absolute proof of fracture, but it can be elicited in only a minority of cases, because it usually evokes intense pain (though not much pain in the period of slight shock following immediately after the injury). *Crepitus* is never present in *greenstick* and *impacted* fractures.

(4) *Loss of Function*.—This might appear at first sight rather obvious. It is, however, a subtle point when all the other clinical signs of fracture seem to be negative. Thus a patient who has sustained an injury which could have fractured the pelvis, even in the absence of physical signs, probably has such a fracture, *if he cannot walk*. Unlike sprains or bruises of soft parts, fractures involving the extremities render it impossible for the patient to exert any significant force by pushing or pulling against resistance (i.e. loss of function, though not necessarily complete loss of the ability to move).

Impacted fractures may present great difficulty in clinical diagnosis. Not infrequently this injury is missed even by experienced general practitioners, e.g. in resolute old ladies who manage to walk, though with a severe limp, on an impacted fracture of the femoral neck. In the absence of obvious signs suggesting a fracture, the difficulty in walking is erroneously attributed to the effects of a 'bruise,' and the fact that the old lady cannot take weight on the limb is sympathetically attributed merely to her age and frailty. In these cases the fracture is frequently only suspected two or three weeks later, at which time a patient with merely a contusion ought to be able to walk quite easily. Though the clinician will be alarmed for his own reputation in having 'missed' an impacted fracture, he can be consoled with the knowledge that, as far as the welfare of his patient is concerned, only rarely is subsequent drastic treatment required.

(5) *Local Bone Tenderness*.—If bone tenderness can be demonstrated, and is accurately localised to that part of a bone where a classical fracture occurs, it is highly probable that an impacted fracture is present, *even though all other signs of fracture are lacking*. Thus an elderly woman who falls on her outstretched hand and sustains an impacted Colles fracture with trivial displacement may have: (1) no deformity; (2) no abnormal mobility; and (3) no crepitus. The fact that she will have (4) loss of function in that she cannot exert any grip may be attributed to contusion and its significance overlooked. But if she has (5) tenderness localised to a point approximately $\frac{3}{4}$ to 1 inch (2 to 2.5 cm.) above the radial styloid, then this patient has an impacted Colles fracture.

What, then, is the most important sign of a fracture? In most fractures with abnormal mobility and deformity the problem does not arise. In impacted fractures, *local bone tenderness* is the most important clinical sign; loss of function is the most important *symptom*.

Other Signs of Fracture.—Three other clinical signs exist which are strongly suggestive of fracture :

(1) *Shock*.—Though by no means a constant finding, patients who sustain fractures in parts not easily examined clinically, such as the pelvis or spine, are frequently more shocked after the injury than might be expected from a bruise. The old lady with the impacted fracture of the neck of the femur will usually have signs of shock, and therefore not much pain for some hours after the injury, and the doctor can move the limb about without her showing much obvious discomfort.

(2) *Ecchymosis*.—Discoloration of the skin which comes to the surface a few days after an injury and at some distance from the site of injury is strongly suspicious of a fracture. A contusion will cause local discoloration almost immediately at the site of the injury. Dislocation will also be followed by late ecchymosis in parts distal and dependent from the injury.

(3) *Fracture Blisters*.—Undiagnosed and neglected fractures often proclaim themselves to the experienced eye by the appearance of cutaneous vesicles, not unlike those of second-degree burns. The vesicles appear four or five days after the injury, when the skin is unsupported by the pressure of external dressings. They are most commonly seen below the knee in fractures of the tibia, bones forming the ankle joint, and tarsus (particularly the os calcis). It is important to use pressure dressings as first-aid treatment of fractures to prevent these blisters, because if neglected, they may become infected and possibly involve the underlying fracture.

Radiographic Proof of Fracture.—It cannot be too strongly emphasised that even in cases in which there is but the merest possibility that a bone is fractured, an X-ray of the whole bone should be taken in at least two planes. Otherwise the 'reasonable skill and care' of the practitioner may be questioned, and no other branch of surgery is more damaging to a practitioner's reputation. A wise and experienced Irish surgeon taught his pupils that "bones are not filled with red marrow, but with black ingratitude".

UNION OF FRACTURES

The dense bone which composes the cortex of a long bone is very complex in its minute structure, resembling, in fact, a piece of 'plywood' in that it has a large number of layers which are laid down so that the fibres of each layer cross those in the next layer. Great strength is thus obtained with minimum weight. It is obvious that to lay down a specialised tissue like this cannot be done quickly, and thus the complicated stages of fracture healing arise from the fact that the 'callus' which holds the bone-ends together is a primitive tissue comprising a series of transitional tissues, each one of which is stronger and denser than its predecessor. Thus the hæmatoma between the bone-ends is first replaced by a delicate spindle-celled tissue capable of progressing to mature fibrous tissue, cartilage, fibrocartilage, and bone. The texture of callus is unlike the bone of the original shaft in that it has no 'plywood' structure. The later stages of union are con-

cerned with the removal of the temporary callus and its replacement by permanent 'lamellar' bone.

In cases where union is slow, the first primitive tissue passes not to bone but to mature scar tissue, thus giving a 'fibrous' union. In other cases of slow union the first primitive tissue may specialise into cartilage and synovial membrane and a 'pseudarthrosis' results.

The factors responsible for delayed union seem to be local factors in the fracture itself. Diet, systemic disease, and vitamin deficiency have all been incriminated from time to time, but the fact remains that delayed union of the tibia is exceedingly common in healthy men of military age, whereas in old and debilitated subjects the fractures often unite quite well. If the cause of delayed union were systemic, how could one explain the fact that a patient sustaining two fractures at the same accident may have rapid union in one fracture but non-union in the other?

Tests for Union.—In describing the state of union at any given time, the physical state of the fracture is conveniently labelled by the following three terms :

(1) '*Trace of Give*' or '*Sticky*'.—Here the fracture is not freely mobile, but careful examination will suggest a few degrees of movement. The movement here is so slight that it may even be disputed by different examiners. Sometimes this movement is present only in one plane if the fractured bone is splinted by another intact bone; thus the fibula splints and masks movement in the tibia. Union here is only by fibrocartilage.

(2) '*Clinically United*'.—The fracture feels solid when strained vigorously by the examining hands. The fracture will still be clearly visible in the X-ray, but it will be surrounded by a haze of bony callus.

(3) '*Radiological Union* or '*True Bony Union*'.—Here the fragments of the original fracture are no longer clearly visible and, at least in some part of the fracture, a newly formed piece of cortex can be seen running continuously across the site of the injury. The external callus originally seen as a haze outside the fracture will now have disappeared.

In deciding when to abandon splintage and when to permit graded use of the limb, we usually accept the clinical tests for union without too much dependence on X-ray. On the other hand, we wait for radiological proof of true union before permitting the patient to bear weight, especially in the case of the long bones of the lower extremity, and before discarding crutches completely.

The tests for clinical union are, of course, the converse of the Five Signs of Fracture: i.e. (2) no abnormal mobility (i.e. not even 'sticky'); (3) no crepitus; (4) return of function; (5) no local tenderness over the callus. Where is (1)?—if the fracture has been adequately treated, there should be no deformity!

First-aid Treatment of Fractures.—The cardinal principle is to immobilise the joint above and below the fracture. In the upper extremity, being relatively light in weight, the arm can be held to the side of the body with slings and bandages, and the forearm is splinted against any flat piece of

wood suitably padded. The elbow and shoulder may need a sling. The lower extremity is best splinted on a Thomas's splint passed over the outside of the trousers and held on with a clove-hitch round the boot (but not left in strong traction for more than about four hours or the skin on the dorsum of the foot may slough). The tibia and ankle are easily splinted with a wooden 'back splint and footpiece', but take care to protect the heel from pressure sores.

Care should be taken not to convert simple fractures into compound fractures, and bones protruding through the skin should not be reduced at the scene of the accident, but the whole wound should be dressed with some bland antiseptic such as Dettol, Cetavlon or acriflavine.

PRINCIPLES OF THE TREATMENT OF FRACTURES

The treatment of a simple fracture falls under three headings, i.e. (1) reduction ; (2) fixation ; and (3) rehabilitation.

(1) **Reduction.**—A fracture needs reduction only if it is displaced. Not so obvious is the degree of displacement which can be tolerated without reduction being necessary. Speaking very broadly, reduction is needed only if deformity can be detected on clinical inspection ; there are many exceptions to this sweeping statement, but the principle is still true that we do not reduce X-rays, but only clinical deformities.

We do not reduce fractures to make them unite ; we reduce them so that when they are united they are in good position for function. Unreduced fractures often unite very quickly, but they give deformity—i.e. 'mal-union'. Fractures fussily over-treated by elaborate mechanical means in order to get a perfect radiological reduction often unite with the greatest difficulty. To some extent non-union is a product of civilisation ; rapid mal-union is the natural state of the uncivilised fracture. Reduction cannot be achieved with accuracy unless muscle spasm is abolished by anæsthesia. The manipulative reduction of a fracture is always easier if done without unnecessary delay, before the surrounding tissues have become swollen and turgid.

(2) **Fixation.**—In most fractures the reduced position can usually be maintained by the use of a skilfully applied plaster cast, in spite of the return of normal muscle tone which follows recovery from anæsthesia. Fractures of the shaft of the femur are, however, a special exception, being unsuitable for this sequence of 'manipulative reduction and plaster fixation', because the powerful thigh muscles will continue to cause shortening, and because the soft tissues of the thigh are so bulky that no plaster cast can control the contained fragments. This is an instance where continuous traction must be employed to prevent shortening.

The technique of manipulative reduction and plaster fixation can only be learned by apprenticeship, but certain guiding principles are worth stating :

(a) Displacement indicates the tearing of soft parts which allow the fragments to move away from each other.

(b) Usually the torn tissues are confined to one side of the fracture—the *convex* side.

(c) By making use of the intact tissues on the *concave* side of the fracture, the fragments can be aligned to each other.

(d) Reduction consists of attempting to hold the fracture in an 'over-corrected' position; by this means the intact tissues are stretched and the torn tissue relaxed.

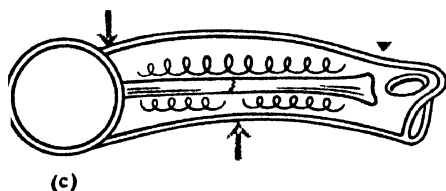
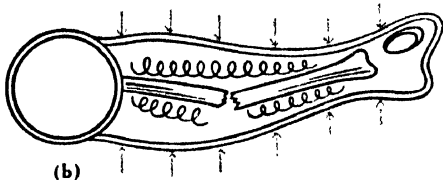
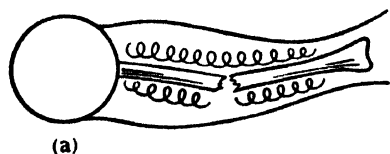


FIG. 210—'Three-point' action of a plaster cast: (a) Original deformity. (b) Original deformity recurring when pressure equal at all parts. (c) Three-point action holding correction.

(e) Fixation by plaster is based on perpetuating the slightly over-corrected position by moulding the plaster. Therefore, paradoxical though it may sound, it needs a plaster slightly curved in the direction of over-correction to produce a straight limb (fig. 210).

(f) Plasters which exert a positive moulding force do not exert an even pressure over the whole surface of the limb. A positive moulding force can always be resolved into a 'three-point' system, where the two distal points of pressure simulate those applied by the surgeon's hands, and the third point is situated in the most proximal part of the plaster.

(3) **Rehabilitation.**—The general principle and aim of rehabilitation is to return the injured person to work in the shortest period, with the

minimal residual disability. It is dangerously easy, however, to think of rehabilitation as a separate subject, often in a different building, generally with a different medical staff which takes over the patient when the main part of the 'hospital treatment' is finished. Such a concept shows a complete lack of understanding of the meaning of modern fracture treatment. Rehabilitation, physical as well as psychological, should start as soon as the patient is over the main discomfort of the early days of treatment.

Böhler was the first to teach the importance of encouraging the patient to "use the splinted limb" (figs. 211 and 212). By this means the circulation of the limb is maintained, atrophy of muscles is minimised, and the duration of joint stiffness is reduced. In other words, this teaching emphasised that the ill-effects of prolonged disuse are avoidable. Böhler vigorously drove home the lesson that many bad results of fracture treatment were not the results of the fracture itself, but the results of bad splintage.

The first step in rehabilitation is, therefore, the training of the surgeon to apply a plaster which will be comfortable and which will, at the same time as immobilising the fracture, permit the limb to be used. Pain from an

inadequate plaster, or a joint fixed in an unsuitable position, may make it impossible for the patient to follow the surgeon's command that he should "use the splinted limb". Thus, to take as an example a fracture of the forearm, the shoulder should be exercised from the start, the wrist should be fixed in the anatomical position of 'cock-up', and, whenever the fracture will allow, in the middle position of rotation; similarly, the plaster should be so

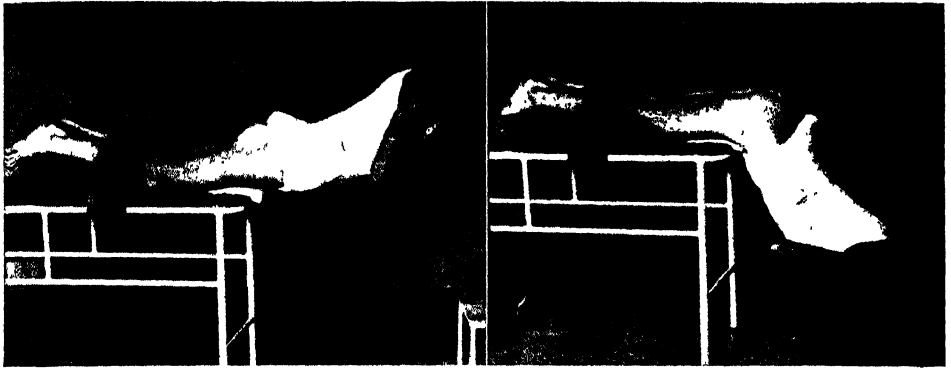


FIG. 211.—Rehabilitation in plaster—extreme example. (Dr. Lorenz Böhler, Vienna.)

moulded round the fingers and palm that it is possible to use fingers without restriction. In the lower extremity, the foot should as far as possible be fully plantigrade and a good walking heel should be used so that the patient can walk with a normal 'heel-and-toe' action. A pressure sore from a badly fitted plaster may render it impossible for the patient to use the limb.



FIG. 212.—A rehabilitation class.

While still in plaster, patients should be made to attend fracture rehabilitation centres in groups and classes. The physiotherapist or remedial gymnast can then scrutinise their attempts to use their splinted limbs and encourage the weaklings. The psychological aspect of this type of rehabilitation is of

paramount importance ; cheerful personalities in the fracture team, interested in patients as human beings, get patients back to work more quickly than gloomy intellectuals who concentrate on X-ray appearances and interesting complications. By all means let surgeons encourage the honest intellectual approach and let us candidly discuss our not-so-good results, but never let it be done in the hearing of the patient. With imaginative patients one careless word may hold up rehabilitation for many months.

Under this regimen the fractured limb will do more than half its rehabilitation while still inside the plaster. The plaster should be retained until the patient can walk in it almost as well as in a boot, so that when the plaster is removed he should be able to walk away with relatively little difficulty. This idea of functional activity in plaster thus puts the patient in the position of merely requiring the residual stiffness removed in the rehabilitation centre. This is quite a different story from that of those rehabilitation centres which see the patient for the first time weeks after the injury, by which time they are faced with the almost insuperable problem of overcoming both the physical and the mental ill-effects of mediocre treatment.

Under the emergency conditions of war, where large groups of patients lived together in communities away from their homes, rehabilitation was much easier to organise than in peacetime. Organised games play a very useful part in rehabilitation. Billiards, darts, quoits, skittles, etc., interest most patients. Competitions can be arranged, during the excitement of which a patient will often involuntarily exercise a limb far more efficiently than if he were conscious of his effort.

Close liaison with a medical social worker (almoner) during this phase is important in solving financial and domestic worries produced by the accident and which, if allowed to persist, may have an ill-effect on recovery. Vocational training is provided by the Ministry of Labour for patients who have suffered from permanent disability, as a result of which they cannot follow their original employment.

COMPLICATIONS OF A FRACTURE

I. General

(1) *Surgical Shock*.—Shock which does not recover within an hour of injury by such simple methods as warmth, morphine, and splintage is probably of the 'oligæmic' variety. A fracture of the tibia rarely produces severe shock, because not much blood is lost locally in the calf ; in the thigh, on the other hand, as much as 3 pints (1·7 l.) of blood can be accommodated without arousing any more comment than the fact that the thigh is very swollen. To give an anæsthetic to such a case, too early and without fluid replacement, may abolish the vasoconstrictive mechanism which maintains the blood pressure, and a fatal fall of pressure may occur (p. 77).

(2) *Fat Embolism*.—See p. 133.

(3) *Hypostatic Pneumonia*.—This is particularly liable to occur in elderly patients confined to bed for the treatment of fractures of the neck of the femur.

2. Local

The local complications of a fracture can be summarised :

Early	Late
1. Nerve injury	4. Avascular necrosis
2. Arterial injury	5. Ischæmic contracture
3. Infection	6. Delayed union
	7. Non-union
	8. Mal-union
	9. Pressure sores
	10. Joint stiffness

(1) **Nerves.**—Nerves, particularly those which lie adjacent to bones, are liable to be involved as a result of contusion, traction, or laceration by the jagged fragments. Appropriate tests must always be carried out when the patient is first seen in order to test the function of any nerve which may have been injured.

Contusion, which gives rise to transient block (neurapraxia) is the commonest lesion, and recovery usually occurs within two or three weeks.

Axonotmesis, or lesion in continuity, occurs if a nerve is crushed so that the axis cylinders only are damaged. The sheath of the nerve is intact, so a good functional result is to be expected after a few months, as axons reunite with a minimum of 'shunting'.

Neurotmesis, or complete division of the nerve, only occurs when there is considerable displacement of fragments.

Nerves which are especially liable to be injured at the time of the fracture are the median, in the case of supracondylar fracture of the humerus, and the radial in fractures of the shaft of the humerus as it lies in its groove on the bone.

(2) **Blood-vessels.**—Arteries or veins are sometimes ruptured at the time of the fracture (p. 123 and fig. 40). Vessels may be occluded by spasm due to bruising, or by the pressure of displaced fragments. The commonest example is occlusion of the brachial artery in association with supracondylar fractures of the humerus, but pressure on the popliteal artery by the diaphysis of the femur in the case of separation of the lower epiphysis may also occur. If the circulation of the limb is unsatisfactory after reduction of a fracture, exploration of the adjacent artery is often advisable if attempts to block the sympathetic chain with local anæsthetic produces no improvement (p. 122).

(3) **Infection.**—The infection of a compound fracture is a very serious matter because, if osteomyelitis results, there may be a persistent discharge of pus for months or even years. With antibiotics, and the ability to operate within a few hours of the injury, the serious effects of osteomyelitis are much less common than formerly. Before the advent of antibiotics, infected compound fractures were particularly prone to *secondary hæmorrhage* by the erosion of a large vessel in the neighbourhood of the fracture; fortunately this complication is now very rarely encountered.

Sequestration of dead fragments of bone may occur in the course of the

later stages of osteomyelitis, and until all the sequestra have been removed, the discharge cannot be expected to stop.

Gas gangrene (p. 44) is a serious complication of a dirty compound fracture, and though this also is less frequently seen to-day than formerly, it still occurs despite the use of antibiotics. The development of gas gangrene is favoured by erroneous judgment in closing wounds which contain large amounts of dead muscle (i.e. deprived of blood supply by the injury), and by overlooking radio-translucent foreign bodies, such as clothing and pieces of wood, which are buried in the depths of the wound.

(4) **Avascular Necrosis.**—This occurs when the fracture completely interrupts the blood supply to one of the fragments. The anatomical arrangement of the blood supply to special bones makes this complication common in certain sites, as in the head of the femur after fracture through the neck, and in the proximal fragment of the carpal scaphoid after a fracture through the waist. If the fragments are closely coapted and rigidly fixed the blood supply may re-establish itself, but more often the changes of aseptic death supervene and the bone crumbles and collapses. This is later followed by osteoarthritic changes.

(5) **Ischæmic contracture** is a rare sequel to arterial complication of a fracture. The best-known example, fortunately uncommon, is *Volkman's contracture* of the flexor muscles of the forearm after fractures at the elbow or in the forearm in children (p. 201), but even more rarely it can occur in the leg and calf. If arterial spasm persists long enough to cause aseptic necrosis of the flexor muscles, these will eventually be replaced by fibrous tissue which will contract. At the same time ischæmia of nerve trunks may cause changes which impair conduction. The reason why the arterial spasm can persist, despite all attempts to dilate the arteries, is still obscure. Though the arterial spasm is usually precipitated by inexperienced handling of a fracture, cases have been encountered where adequate skill and care have been used and undoubtedly cases must exist where the fracture itself is responsible for the initial spasm.

Volkman's contracture is notoriously prone to follow the reduction of supra-condylar fractures in children. The child always has persistent severe pain after the reduction, and the whole of the damage is sustained within the first twenty-four hours after the reduction (and quite probably within six to twelve hours). The hand can be white or dusky. There will be anæsthesia of the whole hand if tested by pinprick (the child, of course, not being allowed to see the pin).

Perhaps the most important physical sign—because if detected early it is still not too late to institute remedies—is inability of the surgeon passively to straighten the patient's fingers and at the same time extend the wrist (fig. 213). If this can be done, no damage is occurring in the flexor bellies.

It goes without saying that the radial pulse will be absent in these threatened cases, but an absent radial pulse by itself need not always cause serious worry if severe spontaneous pain is absent (i.e. not just 'tenderness' evoked on being touched but spontaneous pain when the limb is immobile) and especially if the fingers can be passively extended. In these cases the local spasm of the brachial artery obviously has not spread to the collateral vessels which is the fundamental danger of a Volkman's contracture. If the radial pulse is absent, it is important to release the elbow from too acute flexion to see whether this may restore the pulse.

If danger signs are present, the following routine should be adopted: (1) release all

flexion from the elbow and allow it to extend beyond the right angle (2) immediately block the stellate ganglion with Novocain without any undue delay in trying trivial remedies; (3) heat the child's body with a radiant heat cradle but expose the

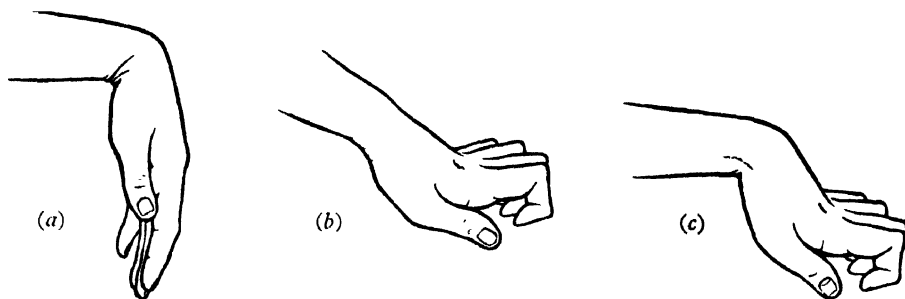


FIG. 213.—Volkmann phenomenon: (a) Mild case: wrist flexed—fingers straight. (b) Mild case: wrist extended—fingers flexed. (c) Severe case: wrist flexed—fingers flexed.

naked arm to the air to keep it cool; (4) administer a vasodilator drug (in appropriate dosage for a child); (5) if all these measures fail after two or three hours, consider exploration of the brachial artery in the antecubital fossa.

Exploration of early cases may be of value. The segment of the artery in spasm, which is contracted to the diameter of a piece of string, is bathed in a warm solution of one per cent. papaverine, or $\frac{1}{2}$ per cent. procaine, for thirty minutes (p. 132). It is no longer considered wise to perform arterectomy on the theory that a damaged segment is the source of irritation causing the spasm.

Late treatment in the established case is mainly concerned with stretching the contracted fingers and preventing further contracture.

Tight Plasters.—Patients have frequently suffered tragic losses of limb, all too often after relatively trivial fractures, and surgeons have had heavy damages awarded against them when there has been evidence of circulatory obstruction after the application of plaster. The problem is complicated because arterial spasm may sometimes, though rarely, be caused by the injury itself, and the surgeon who fails to examine the circulation and sensation in the hand or foot *before* applying a plaster may be penalised. If a tight plaster, or a tourniquet, is left in position too long, the circulation does not return when the constriction is released, because a secondary arterial spasm supervenes, which may resist all measures at relaxation.

Apart from remembering to examine the circulation and sensation before commencing treatment, the most important advice to the inexperienced is not to make the common mistake of thinking that *pain is to be expected* as a normal accompaniment of a recent fracture, either reduced or unreduced. Persistent, spontaneous pain (i.e. not pain evoked merely by movement) is positive evidence of a serious complication, and usually a vascular complication, even if the circulation in the digits 'seems adequate'. Pain after a fracture, especially if it returns in unabated form after partial relief by an analgesic, must be investigated instantly; the mistake must never be made of ordering another dose of analgesic by telephone and 'seeing it in the morning'.

After orthopædic operations it is a sound rule always to split the plaster if the soft tissues of the limb are not already swollen at the time of the operation.

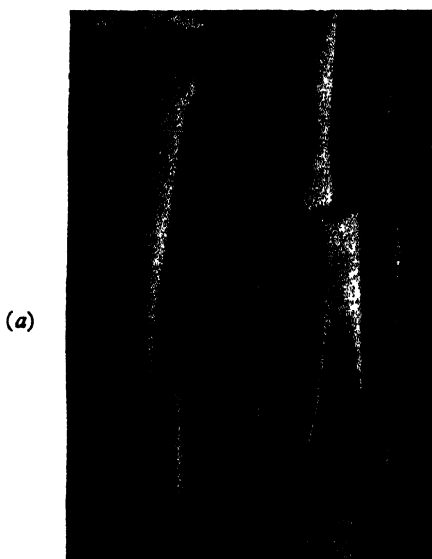
(6) **Delayed Union.**—There are wide variations in the time taken for bone to demonstrate 'clinical union', and wide differences may occur even in

different bones in the same patient. Clinical union should be present in six to ten weeks. If a fracture does not feel solid three months after an injury, we can certainly diagnose 'delayed union'.

Radiographically, the characteristic feature of a delayed union is that no evidence of repair is present. The outline of the bone fragments is almost as clearly defined as in a recent fracture and no callus will be visible. The only indication that the fracture is not recent may be slight generalised disuse osteoporosis. Such a fracture may easily be mistaken for a fracture six weeks old, when in actual fact it is six *months* old; it seems to be 'in cold storage' (fig. 214 *a*).

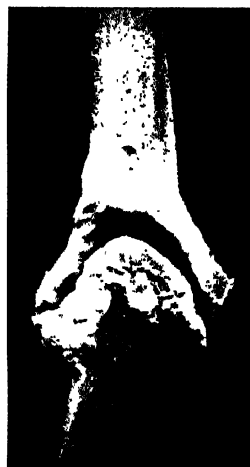
Delayed union implies that spontaneous union is still possible, and treatment consists of continuing the fixation and of encouraging the patient to use the limb.

(7) **Non-union.**—'Non-union' is the end-result of a minority of cases of 'delayed union' which do not eventually unite spontaneously by continued



(a)

FIG. 214.—(a) Delayed union—no sclerosis. (b) Non-union—sclerosis.



(b)

FIG. 215.—A false joint following an ununited fracture of the shaft of the humerus. (R.C.S. Museum.)

immobilisation. Non-union is a permanent end-result; union can only be obtained if operative intervention is undertaken and some form of bone graft is performed.

Radiographically, 'non-union' is diagnosed by evidence of sclerosis, by rounding-off of the bone-ends, and by closure of the medullary canal by dense bone at the site of the fracture. In its fully established state (figs. 214 *b* and 215) this rounding-off of the bone fragments shows that a false joint or pseudarthrosis has formed. Clinically, a pseudarthrosis is shown by the presence of 5 or 10 degrees of free mobility in a fracture six months or more old. In these cases the false joint is lined with irregular cartilage and contains synovial fluid and synovial membrane.

Causes of Non-union and Delayed Union.—It is customary to mention *general causes* of delayed union, such as old age, debility, vitamin deficiency, and systemic disease, such as syphilis, but none of these is a constant factor, and everything seems to point to the greater importance of some local factor.

Local Factors are numerous and may be summarised as follows :

- (a) Infection.
- (b) Inadequate immobilisation.
- (c) Impaired blood supply.
- (d) Over-distraction.
- (e) Interposition of soft parts.

(a) *Infection.*—Severe osteomyelitis undoubtedly delays union, but a mild infection which clears up in a month or two usually is without ill-effect.

(b) *Inadequate Immobilisation.*—If the bone fragments are continuously in motion, the new cellular tissue attempting to bridge the gap may develop a plane of cleavage and the attempt to bridge this gap may be permanently foiled by the ends becoming insulated from each other by mature fibrous tissue. Once this has happened, the bone tends to become rounded off and sclerosed.

Failure of immobilisation, however, does not seem to be the whole story because we know that fractures in the shaft of the femur and the humerus are often capable of uniting in the presence of movement. It seems that certain bones—especially the lower one-third of the tibia—have no natural capacity for generating profuse periosteal callus. In these cases it would seem reasonable that a scanty growth of callus could be compensated by greater attention to immobilisation, but no external splint applied outside the skin (such as a plaster cast) can prevent a certain amount of movement in a fracture.

A fruitful source of non-union, especially at sites such as the lower one-third of the tibia where callus production is scanty, is repeated late attempts to readjust the position of the fracture. Ideally, all manipulations and adjustments to a fracture should be completed within the first week, and thereafter the plaster should be left untouched until the time when the fracture is likely to be clinically solid ; a final change can then be made to a close-fitting plaster in which weight-bearing can be safely permitted.

(c) *Lack of Blood Supply.*—In certain parts of the skeleton the fracture will itself deprive one of the fragments of its natural blood supply and so cause non-union. It is doubtful whether any displaced subcapital fracture of the neck of the femur would ever unite without absolute fixation, and the same applies to fractures through the waist of the carpal scaphoid.

(d) *Over-distraction.*—A fruitful cause of delayed union and non-union is the excessive use of skeletal traction. Excessive traction pulls the bone-ends apart and so favours the growth of fibrous tissue which insulates the bone-ends from each other. Modern ideas emphasise the use of the minimum traction force to achieve the desired result.

(e) *Interposition of Soft Parts.*—Fascia or ligaments appear to be a more effective barrier to the growth of callus across a fracture than the interposition of muscle. Examples of callus growing considerable distances through muscle are not uncommon. In fractures of the patella, the medial malleolus,

and the olecranon, a flap of ligamentary tissue is often found to have fallen into the fracture line, so that accurate apposition of the surfaces cannot be obtained unless the flap is removed. Without operation there is a strong tendency for fibrous union in the patella and olecranon, and the fibrous tissue may stretch under the tension of the traction tendons.

Treatment.—In *delayed union* all that is necessary is to persist with plaster fixation and to encourage the use of the limb. If there is no evidence of sclerosis and rounding-off of the bone-ends, there is always a chance that further fixation will still produce union. If, however, the fracture is rather freely mobile, i.e. is not just 'sticky', and there exists considerably more than 2 or 3 degrees of movement, it is frequently economically wise to treat the case operatively by bone grafting, as if it were already a non-union, rather than to waste further time and then eventually to find that operative treatment is necessary.

Three techniques of *bone grafting* are in common use :

(a) *Massive Onlay Graft.*—A heavy piece of autogenous bone is cut from the normal tibia and screwed across the surface of the non-union, after preparing a suitable bed (fig. 216). In a number of cases, where the ununited fracture is at the junction of thirds, it may be possible to perform a 'sliding graft' without taking bone from the opposite leg. In these cases the graft is taken from the longer fragment, slid across the area of the non-union, and recessed into the shorter fragment.

(b) *Cancellous Bone Graft.*—Modern practice is tending to supplant the cortical graft by the cancellous graft because of its greater power of union and its resistance to slight infection. If there is only a little movement in the non-union, the Phemister technique can be used ; the periosteum is elevated on one or two sides of the fracture, without disturbing the fibrous union of the bone-ends, and slices of iliac bone are laid across the non-union and the wound closed. After three months in plaster the fracture will be solid, and the site of the fibrous union will disappear slowly and spontaneously. It is to be noted that no attempt is made to resect the bone-ends and cut away sclerosed bone as was often done with massive onlay grafts in the past. Cancellous bone grafting by this technique is a very safe and reliable operation and has a high success rate, even in cases which have only recently overcome local infection (where a massive onlay graft would be disastrous).

(c) *Metallic Fixation plus Cancellous Chips.*—If there is excessive mobility at the non-union, it is often desirable to procure fixation by stainless steel or vitallium plates and screws and then to stimulate osteogenesis by means of chips of iliac bone packed round at the same operation.

It takes approximately three to four months for a bone graft to become incorporated and strong enough to abandon all external fixation.

Bank Bone.—Recently there has been a wide use of bone stored in the 'deep-freeze' refrigerator. This 'homogenous' bone (commonly ribs removed at thoracoplasty) does not have such good powers of incorporation as autogenous bone, and

therefore is not to be recommended for non-union in fractures of long bones. Stored bone is, however, very useful in filling gaps, and is often successful in spinal fusion, though taking longer to consolidate than autogenous bone.

(8) **Mal-union.**—Mal-union is the union of a fracture in defective position. The two most serious deformities of mal-union are (a) angulation and (b) excessive shortening. Angulation throws out of line the correct transmission of

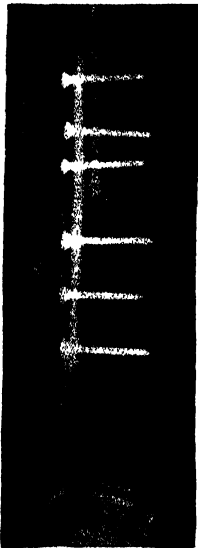


FIG. 216.—Massive onlay cortical bone graft.

stress through joints above and below the fracture and, in the lower extremity, leads to osteoarthritis in the disturbed joints. Gross shortening can always be corrected by a raised boot. An adult can usually compensate for as much as $\frac{3}{4}$ inch (2 cm.) of shortening without needing a surgical raise on the shoe. It is also important to observe that a shortening of 1 inch (2.5 cm.) does not necessarily need the full raise of 1 inch; in such a case, $\frac{1}{2}$ inch on the heel alone is often enough to avoid a noticeable short-leg dip when walking, and the minimum amount can be determined by experiment.

If angular deformity is great, it will need surgical correction by osteotomy, because no surgical appliance can compensate for it.

(9) **Pressure Sores.**—Pressure sores are produced by ridges or local pressure. Ridges are due to the uneven application of a bandage, or to the alteration of the position of a joint after a loose plaster has been applied. Local pressure occurs if bony prominences are unprotected, or if pressure is allowed to indent the cast before the plaster has set. Persistent localised discomfort or pain must not be ignored, and the gradual diminution of the pain should not lull the surgeon into a sense of false security, lest it indicate the onset of gangrene (as with gangrenous appendicitis). Recurrence of œdema of the digits after initial swelling has subsided usually indicates a pressure sore. If infection occurs, it can be recognised by a local patch of warmth or discoloration of the plaster, and later by the odour which emanates from beneath it. When a sore is suspected an inspection window is cut in the plaster, and if no sore is discovered the gap is packed with cottonwool and firmly bandaged in order to prevent œdema. If a sore has occurred, the window must be enlarged beyond the area affected, and suitable antiseptic dressings are applied (p. 37).

(10) **Joint Stiffness.**—Some temporary joint stiffness is to be expected after a fracture; what is most important is that there should be no permanent stiffness.

Fractures involving joint surfaces, or very close to joints, are more prone to cause stiffness than fractures of the shaft of the bone at a distance from the joint. Thus fractures involving joints must be moved earlier than fractures of the shaft of long bones. Anatomical restoration of joint surfaces by open operation in theory is the ideal procedure if combined with early movement, but strangely it often does not give as good mobility as does early movement without operation and with acceptance of some deformity.

INDICATIONS FOR OPERATION ON FRACTURES

The indications for operative intervention in connection with a fracture can be classified as immediate, intermediate, or remote.

Immediate.—(a) *Complicated fractures*, as in the case of rupture of, or pressure on, the main vessels of a limb, may require operative intervention in order to control hæmorrhage, or to diminish or obviate the risk of gangrene or ischæmic contracture.

(b) *Compound fractures* must be operated upon immediately in order to

diminish the risk of infection. Each hour's delay allows infection to become more firmly established and devitalised tissues are particularly susceptible to bacterial invasion. A delay of six hours usually means some degree of infection.

The aims of the treatment in compound fractures have been considerably modified since the introduction of antibiotics. Open fractures received in a fully equipped fracture service within a few hours of injury are nowadays ideally treated by suturing the skin wound and so converting the fracture into a closed fracture. In suitable circumstances skin cover by immediate skin grafting may be advisable. In a few cases (though certainly not as a routine) the experienced operator may with advantage use internal fixation in open fractures, just as he would in fractures which were originally closed.

If a compound fracture is twelve or more hours old, and especially if it is obviously contaminated with road dirt or agricultural earth, it is unsafe to close the wound completely, and this applies even with the protection of antibiotics. In these cases the Winnett Orr method can be used, following the technique popularised by Trueta in the Spanish Civil War. In this technique the wound is surgically debrided, left open, and packed with petroleum jelly gauze, and the whole limb is fixed in plaster after reduction. The plaster is left untouched for two or three months, and any accumulation of foul-smelling discharges is counteracted by various nursing manœuvres (open-air wards and odour-absorbent dressings containing activated charcoal). When the plaster is eventually removed the gauze will be found to have been extruded on to the surface and the wound itself will appear as a flat area of healthy granulation tissue under which the united fracture is sealed off.

Very severe compound fractures of the leg, if complicated by nerve damage and especially if at the same time it is doubtful whether the blood supply is adequate to nourish the foot, are best treated by *immediate amputation* without wasting further time. Failure to make a firm decision in these cases invites the risk of gas gangrene; also a limb may perhaps be saved, after weeks of grave illness to the patient, which is of little use and which later must be amputated because it is painful and useless (p. 42).

Internal Fixation.—It must be admitted that the internal fixation of recent fractures is more often governed by the personal experience of the surgeon rather than by any absolute surgical rules which at the moment can be clearly enunciated. Thus, some surgeons may advise the internal fixation of all fractures of the tibia by plates and screws, while others will not consider the tibia suitable, yet will advise the plating of fractures of both bones of the forearm. Sometimes the combination of two fractures sustained simultaneously may be difficult to treat conservatively and the problem is simplified if one of the two is operated on; thus a fracture of the femur and the tibia on the same side is commonly treated by operative fixation of the tibia and conservative treatment of the femur or *vice versa*.

Certain fractures give such excellent results by open operation and internal fixation that there is no disputing that these are best treated in this way rather than by closed methods. The best known are :

(1) Fractures of the neck of the femur, (2) the patella, and (3) the olecranon.

The intramedullary nail (Küntschner) has gained a permanent place in the treatment of fractures of the shaft of the femur in the *middle and upper thirds* (fig. 217), though it is unsuitable in the neighbourhood of the knee. It often enables a patient to leave hospital in three weeks and to have a full range of knee movement and a perfectly straight femur; the alternative conservative method necessitates three months of bed-rest with splintage, and a total stay in hospital of four or five months.

FRACTURES OF CHILDHOOD

Childhood fractures are quite different from adult fractures, and the principles of treatment also have very little in common with these. Moreover, the treatment of childhood fractures is one of utter simplicity (with one or two notable exceptions), and this simplicity is obscured when overshadowed by the complex problems of the adult fracture.

The simplicity of fracture treatment in the child is the result of the following features :

(1) Delayed union practically does not exist. In childhood all the osseous tissues (endosteum, periosteum, and possibly even the cortex itself) are strongly osteogenic and callus can bridge wide gaps. There are only two exceptions to this statement : non-union of the capitellum (due to rotation of the fragment out of the elbow joint) and 'congenital pseudarthrosis of the the tibia', which in all probability has some obscure pathological basis.

(2) Permanent joint stiffness is unknown. Thus prolonged fixation (if ever needed) can be countenanced and early movement is unnecessary.

(3) Spontaneous correction of deformity. The younger the child the more easily does a fracture deformity diminish or even disappear. A child of five years, unlike an adult, will not have the same bones by the time he has reached fifteen or twenty years of age.

It is sometimes stated that at all costs *angular deformity* should be corrected because this might persist. Though a *severe* angular deformity may never completely disappear with growth, it will certainly diminish. The nearer the age of the child to puberty the less the chance of complete correction. The fear of angular deformity in children arises from the unwarranted belief that growth will continue *in the line of the deformity*, in which case it would obviously get worse.

Spontaneous Correction of Length.—In a growing child some obscure mechanism exists which stimulates the growth of an extremity which has been made shorter than its fellow by an over-riding fracture. The success with which this occurs depends on the age at the time of the fracture (fig. 218).

Ease of Manipulation.—The *greenstick* fracture of children is malleable, and even when it is converted into a complete fracture by over-correcting the de-



FIG. 217.—Küntschner intramedullary nail.



Fig. 218. — Over-riding femur in child, age six, with $1\frac{1}{4}$ inch (3.2 cm.) shortening; which was eventually neutralised by increased growth.

formity there is much less tendency for the fracture to become displaced than in adult fractures. The reason is that the child has relatively much stronger connective tissues, and the periosteum is stronger than that of the adult as it is five or six times thicker and very much tougher.

Epiphyseal Arrest and Ischæmic Necrosis.—It might be thought that danger to growth by involvement of epiphyses would be common. In fact it is very rare—the only well-recognised site being the uncommon complication of fracture through the lower tibial epiphysis. Similarly, ischæmic necrosis of epiphyses might be imagined as a likely sequel, but in fact it is exceptionally rare in children after injury.

Immunity of Certain Bones.—In children some of the sites which cause the greatest trouble in adults are almost immune from fracture. Thus the spine in children is so resilient that after ordinary injuries it is rarely fractured and paraplegia is practically unknown. Similarly, the pelvis is seldom injured and visceral complications are remarkably rare, even after being 'run over' by car wheels. The carpal scaphoid never fractures in young children, and so never suffers from ischæmic necrosis.

Difficult Childhood Fractures.—There are two common fractures in childhood likely to cause difficulty; i.e. the supracondylar fracture of the humerus (p. 201) and the fracture of the capitellum (p. 203). In the former the vascular complications are the main problem; in the latter the late development of cubitus valgus and ulnar paralysis (p. 330). A much rarer injury than either of the preceding, though very sinister when it occurs, is separation of the lower femoral epiphysis. This can cause pressure on the popliteal vessels and gangrene of the foot (p. 39).

SPECIAL FRACTURES

FACIO-MAXILLARY INJURIES

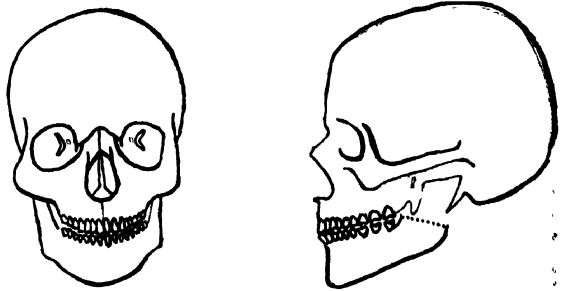
These injuries are all too frequently the result of road accidents. The victims are usually car passengers, thrown forwards against the windscreen or dashboard. The wearing of safety harness, now compulsory equipment in new cars, reduces the incidence of this mutilation.

Fractures of the maxilla are classified according to Le Fort¹ (fig. 219). The likelihood of a fracture being present is suggested by the type of accident, the facial injury (dish-face deformity, split-open face, or swelling), the inability to close the jaws (open-bite deformity), or diplopia due to upward displacement of the floor of the orbit. An X-ray confirms the type of fracture.

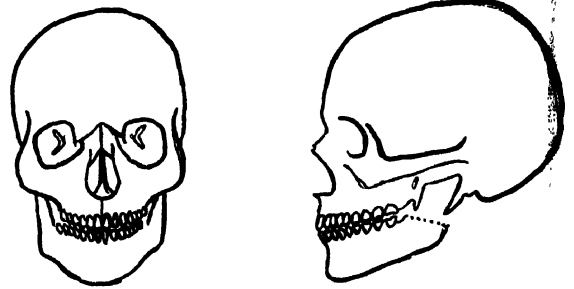
¹ René Le Fort, Surgeon, Paris, defined these fractures as early as 1901, by macabre research, in which he dropped rocks and other heavy objects on the faces of cadavers.

Treatment is best carried out in a facio-maxillary centre. In addition to the repair of the face (p. 458), the displaced maxilla is freed, by rocking with bone forceps, and restored to its normal position. According to the type of

Le Fort I. Fracture involving the maxilla only. The fracture line passes through the base of the antrum on either side and across the nasal floor through the septum.



Le Fort II. Posteriorly the fracture line passes up behind the nose in the direction of the malar-maxillary junction, across the posterior wall of the antrum, and across the pterygoid process in the middle or upper third. The anterior fracture line passes into the orbit in the area of the lachrymal bone, and soon emerges again to break across the fronto-nasal process of the maxilla and the bridge of the nose to meet a similar fracture line on the opposite side. The posterior wall of the orbit is not fractured in this type.



Le Fort III. There is a cranio-facial disjunction, the fracture line passing through both zygomatic arches, both external angular processes of the malar bones, across the back of both orbits, and the back of the nose in the ethmoid arch. There is a separation of the nasal bones from the frontal bone. Posteriorly the septum is fractured high up and likewise the pterygoid processes. There is a complete separation of the maxilla, nose and ethmoids and the palatal bones from the base of the skull.

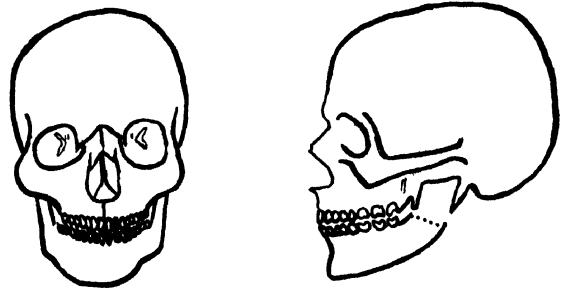


FIG. 219.—Naso-maxillary fractures, Le Fort classification.

fracture, stability is maintained by either (1) wiring the upper and lower jaws together in proper occlusion, (2) by wiring a pliable metal bar to the teeth, or the edentulous alveolus, and fixing this by a system of extra-oral rods (scaffolding) to a plaster of paris head-cap, (3) by internal wire sutures (maxilla to malar bone).

The **Zygomatic Arch** may be fractured and depressed by a direct blow. The important physical signs indicating the necessity for reduction are diplopia and obstruction to opening of the mouth. Unsightly deformity results (fig. 220), and anaesthesia of the cheek may occur owing to pressure on the infraorbital nerve. Pain on mastication is commonly noticed. A small incision is made in the hair

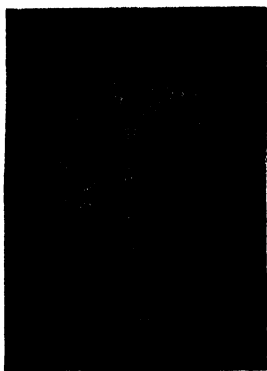


FIG. 220.—Depressed fracture of left zygomatic arch. Motor cycle accident. (T. McNair, F.R.C.S., Edinburgh.)

margin above the bone, through which a blunt instrument is inserted. This is manipulated beneath the arch so that the fragments can be elevated. Reposition of the depressed arch should always be performed, otherwise the cheek is permanently flattened.

The Nasal bones are commonly fractured as a result of direct violence. The fracture usually occurs near their lower margin, but in severe injuries the root of the nose may be driven in towards the base of the skull and the septum is commonly fractured and displaced. Epistaxis, considerable swelling, and surgical emphysema may result. Consolidation speedily occurs, hence replacement should be undertaken without undue delay, although reduction may be successful within two weeks of the injury. Under anaesthesia one blade of a pair of long forceps, protected with rubber tubing, is introduced into each nostril alternately, and the fragments levered back into position. It may be necessary to exert external digital pressure on the fragment in the direction of the deformity, so as to disimpact it, before reduction is possible. Fixation is not necessary.

The Mandible is usually fractured in one of three situations (fig. 221):

(1) The *neck of the condyle* is occasionally fractured, in which case it is displaced forwards and inwards by the pull of the attached external pterygoid muscle. Localised pain occurs on movements of the jaw, and crepitus is detected by the patient or surgeon.

(2) The *ascending ramus* may be fractured, usually in the region of the angle of the jaw. Little displacement occurs, as the masseter on the outside and internal pterygoid muscle on the inner aspect sandwich the fragments between them. The injury is suspected on account of persistent localised pain, and is confirmed by an X-ray. Mastication is limited to soft food for three weeks.

(3) The *body of the jaw* is the part most commonly fractured, sometimes as the result of a blow with a fist. The fracture frequently occurs at the site of the socket of the canine tooth, the cavity of which weakens the bone, and also this region is the junction of two curves. Occasionally the fracture is bilateral, in which case the central portion of the jaw is displaced downwards by the anterior bellies of the digastric muscle and the muscles attached to the genial tubercles. Owing to the firm attachment of the mucoperiosteum to the bone, the fracture is nearly always compound.

Diagnosis is usually obvious, as speech and swallowing are impaired. Blood-stained saliva trickles from the mouth, and irregularity of the line of the teeth is apparent. Crepitus can be elicited with ease.

Treatment

As a first-aid measure the fracture is supported by a barrel bandage,¹ which is a great improvement on the 'four-tail' method, in that it supports the jaw without

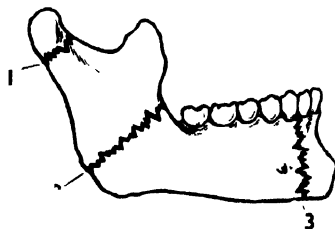


FIG. 221.—Lower Jaw.

1. Neck of condyle.
2. Through the angle or ascending ramus.
3. Commonest site, anterior to the mental foramen, through the canine fossa.

¹ So-called, as it is the method by which draymen secure barrels when lowering them into vaults.

any backward pull, which pull maintains the deformity instead of overcoming it. A 2-inch (5 cm.) bandage is used. It is passed under the jaw so as to support it firmly, and tied in a simple knot over the vertex. The knot is then opened, and one loop is manipulated over the forehead and the other over the occiput. The twist in the bandage on one side thus comes to lie above one ear, and on the opposite side the long end of the bandage is passed beneath the part encircling the head so as to form a corresponding twist. The two long ends are then tied over the vertex (fig. 222).

As soon as possible the patient is referred to the dental surgeon for fixation, the most satisfactory appliance being an interdental splint which fits over the teeth adjacent to the fracture. A corresponding splint is fitted to the upper jaw, and the two splints are fixed together, in occlusion, by wire or elastic bands. Any obviously infected teeth, or an interposed tooth, are removed during the fitting. In edentulous patients, either dentures or acrylic moulds are wired to the alveolus and thence together.



FIG. 222.—The barrel bandage.

Post-operative Care.—As the fracture is compound, infection from the mouth is likely, particularly if pyorrhœa or infected teeth are present. Warm antiseptic mouth-washes are therefore used almost continuously. The diet is confined to fluids, which are taken temporarily through a rubber tube.

Complications are mainly due to infection. Thus necrosis of bone and delayed union are not uncommon, while submaxillary cellulitis may require operative intervention. Aspiration pneumonia is a grave danger, particularly in old or alcoholic patients with pyorrhœa.

UPPER EXTREMITY

The **Clavicle** is said to be the commonest long bone to fracture, indirect violence by a fall on the shoulder, usually at sport, being the usual cause.

Fractures occur in two principal situations (fig. 223).



FIG. 223.—Clavicle.

1. Acromial end.
2. Commonest site, at junction of outer flattened and inner pyramidal portions.

(1) *Acromial End.*—Displacement is slight in this position because the fracture occurs between the trapezoid and conoid ligaments, and thus the two fragments are held in position. Localised pain and tenderness suggest the presence of the fracture, which often requires a radiograph for confirmation. The application of strapping and a sling for three weeks is adequate.

(2) *Mid-shaft.*—This is an exceedingly common fracture, and is usually due to indirect violence, such as falls on the hand or shoulder. It is frequently met with in the hunting-field and on the football ground.

A greenstick variety occurs in children, and is liable to be overlooked, but the definite localised tenderness and reluctance to move the arm should

suggest the necessity for an X-ray. The frequency of fracture at this site is accounted for by the fact that it is the junction of two curves.

Diagnosis is usually obvious, even at a distance, as the patient is seen supporting the elbow on the injured side with the opposite hand, and flexing his head to the affected side in order to relax the sternomastoid muscle. The outer fragment is displaced in three directions—downwards by gravity, forwards by the pull of the pectoral muscles, and inwards by contraction of the muscles inserted into the bicipital groove of the humerus, notably the latissimus dorsi. The inner fragment is tilted slightly upwards by the sternomastoid muscle, and is made the more prominent by the displacement downwards of the lateral fragment.

If displacement is slight, all that is necessary is to place a pad in the axilla, steady the fragments by passing strapping over the site of fracture, and apply a sling for three weeks. If deformity is pronounced, the Three-slings method is adopted.

The Three-slings Method.—Though a number of rather elaborate splints have been devised in an attempt to introduce an element of precision into the treatment of a fractured clavicle, for general use it is difficult to improve on the simplest method of all, the Three-slings method.

The slings are ordinary calico triangular bandages (fig. 224). Two of these

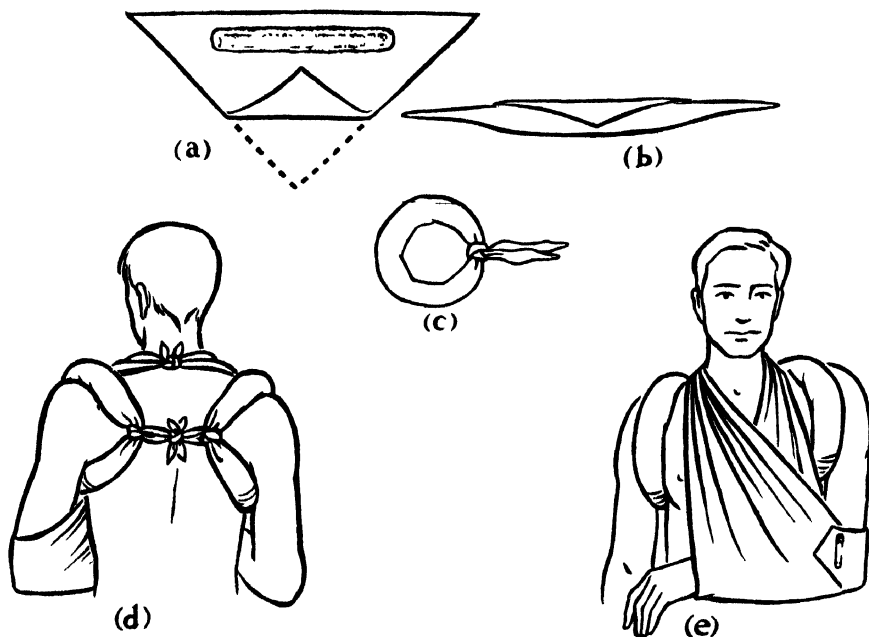


FIG. 224.—Three-slings method for fractures of the clavicle.

are rolled up enclosing cottonwool to make two sausage-shaped pads which are tied round each shoulder as indicated in the diagram. With the patient sitting on a low stool and with an assistant pulling the patient's shoulders backwards (using his knee as a fulcrum), the nurse ties the ends of the two slings together and then pads them so that no pressure is caused on the patient's back.

The third bandage is used to support the elbow on the affected side and to elevate the shoulder. The first two bandages thus brace the shoulder backwards and outwards, while the third supports the shoulder; thus the three deformities are corrected.

Average period of disability—light work five weeks, heavy work eight weeks.

Some degree of mal-union is common, but no disability results. Any cosmetically offensive bony boss can be removed by a simple local operation if the patient specially desires it, but if time enough is allowed to elapse (one to two years) even the most objectionable bony lumps become unobtrusive by the remodelling of bone. If fracture of the clavicle is caused by direct violence, the subjacent structures are sometimes injured, e.g.

the subclavian vessels, brachial plexus, or pleura.

Fractures of the **Scapula** occur in the region of the neck and the body of the scapula. No clinical deformity occurs, and unless crepitus should happen to be felt the diagnosis can only be made radiologically (fig. 225).

Treatment consists in supporting the weight of the arm and forearm by a sling and movements of the shoulder joint are begun after about ten days, as stiffness is more to be feared than inaccurate apposition of the fragments.

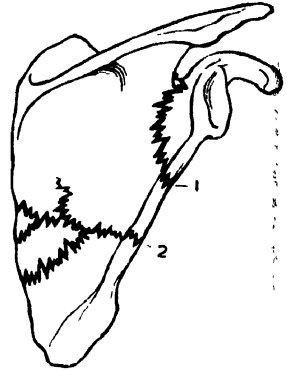


FIG. 225.—Scapula.
1. Surgical neck.
2. Stellate fracture of the body.

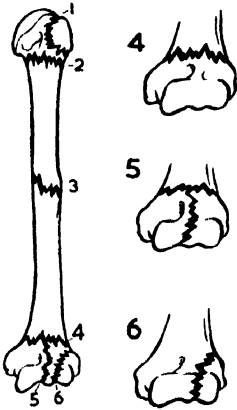


FIG. 226.—Humerus.

1. Greater tuberosity.
2. Surgical neck.
3. Midshaft.
4. Supracondylar.
5. Y-shaped into the elbow joint.
6. External condyle.

Fractures of the Humerus (fig. 226).

(i) *Upper End*.—Fractures of the upper end of the humerus occur most commonly in elderly persons, and particularly women, as a result of a fall on the outstretched hand. Various classifications have been used, such as 'surgical' neck and 'anatomical' neck fractures, or as *abduction* or *adduction* fractures, but as there is no very great difference in their method of treatment, they are all best grouped together as 'fractures of the surgical neck'.

The dominating factor in fractures of this region is that the fracture is close to a joint and early movement is to be encouraged at all costs. The fracture is usually comminuted but, being in cancellous bone, it unites readily in the presence of movement. There is rarely any necessity to attempt accurate reduction of these fractures, because the mobility of the scapula makes up for some residual stiffness in the shoulder joint itself (fig. 227). If the fracture is very grossly displaced, an attempt may be made to improve the position by a manipulation, consisting principally of powerful traction, but otherwise to break down any impaction which may be present is meddlesome.

The type of elderly patient sustaining this injury is prone to develop a stiff shoulder even from a simple contusion; thus early movement by the most conscientious attendance at a physiotherapy department is imperative. Ab-

duction splints are exceedingly uncomfortable and, though quite logical and popular in the past, have now been abandoned in favour of the axillary muff and collar and cuff (fig. 228). This simple device abducts the arm 30 degrees



FIG. 227. — Fracture-dislocation of shoulder in elderly subject. Early movement—position accepted.

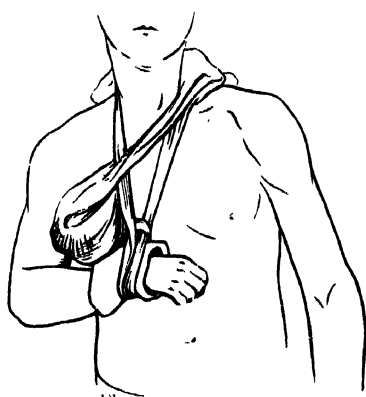


FIG. 228.—Collar and cuff with axillary muff.

from the side and the arm is easily freed for exercise. Swinging movements with the arm hanging vertically should be practised after one week of rest, and thereafter more and more enterprise with 'auto-assisted' movements whereby the patient moves the injured shoulder with the sound arm.

Fracture-dislocation.—This is a severe injury, and the shoulder joint is usually more or less disorganised. In elderly patients, in whom it is common, the shoulder should be abducted to about 30 degrees for three weeks and the radiological appearance ignored. In the case of young and active patients, if traction and manipulation fail, exploration is indicated, so that the dislocation can be reduced and bony fragments approximated.

The *greater tuberosity* may be avulsed in association with dislocation of the shoulder joint (fig. 226). A crack in the greater tuberosity merely requires a temporary sling. If the tuberosity is avulsed and separated by some distance, it is usually impossible to close the gap by abducting the shoulder (as will be seen by attempts under X-ray). Moreover, many separated great tuberosities complicate dislocation of the shoulder, and they should never be treated in abduction lest the shoulder re-dislocates.

(ii) **Shaft.**—Fractures of the shaft of the humerus occur most frequently near the midpoint. They are easily diagnosed by the obvious deformity and abnormal mobility.

Immediate injury to the radial nerve is not uncommon, and to test for the presence of 'wrist-drop' in the clinical examination of a fracture of the shaft of the humerus must never be neglected.

Treatment consists in the application of a U-shaped plaster slab from the axilla under the elbow joint (fig. 229), after which the arm is supported in a collar and cuff and the whole arm bandaged to the side. Reduction under anæsthesia is rarely required, as the weight of the limb and plaster

maintains the fragments in position. General anæsthesia is particularly to be avoided because the horizontal position obstructs reduction; the sitting position of the patient enormously assists the reduction and application of plaster. Local anæsthetic is ideal if the fracture is recent.

(iii) *Lower End.*—Fractures at this site are either (a) supracondylar, (b) T- or Y-shaped fissured into the elbow joint, or (c) of the lateral condyle alone.

Supracondylar fractures and fractures of the lateral condyle (capitellum) are injuries of childhood; T- or Y-shaped fractures of the lower end of the humerus are adult injuries. Supracondylar fractures result from falls on the outstretched hand, T-shaped fractures usually from direct falls on the elbow.

To the inexperienced a supracondylar fracture may be mistaken on clinical examination for a posterior dislocation of the elbow joint, but in the case of fracture the normal relationship of bony points around the elbow is unaltered (fig. 230).

Less common than the ordinary supracondylar fracture, with posterior displacement of the distal fragment, is the deformity of anterior displacement of the distal fragment.

The *supracondylar* fracture in children is notorious for the serious nature



FIG. 229.—A 3-inch (7-cm.) U-shaped slab extends from the axilla round the elbow to the acromion. It is fixed by one gauze and two plaster bandages.

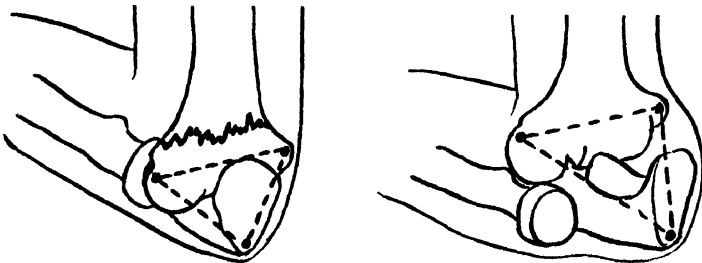


FIG. 230.—Bony points in the elbow: (1) in the normal elbow, or the elbow with a supracondylar fracture, the triangle formed by tip of olecranon and the two epicondyles is roughly equilateral. (2) When the elbow is dislocated the tip of the olecranon is displaced and the triangle is no longer equilateral.

of its nerve and circulatory complications (fig. 231). Sometimes, though by no means always, these complications can be partly attributed to inexperienced treatment, and it is therefore of the utmost importance to record in the preoperative examination the presence or absence of the radial pulse or other signs of serious circulatory embarrassment as well as to test for anæsthesia in the distribution of the median and ulnar nerves.

Treatment.—Reduction is not possible without full general anæsthesia. The danger of causing damage to the circulation in this reduction is so

considerable that only an operator with some experience should be entrusted with the reduction, but as the best results are achieved in the early hours after the injury before swelling is severe, it is incumbent on the casualty officer to

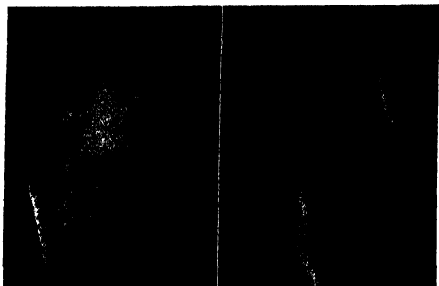


FIG. 231.—Supracondylar fracture. Danger to neurovascular structures between fragments.

study the details, as he may be in a better position to reduce the fracture than a more experienced person the next day. The crucial manoeuvre is the final flexing of the elbow, but this must not be done until the fragments have been disengaged by careful traction. If the elbow is flexed without disengaging the fracture, irreparable damage may be inflicted on neurovascular structures trapped between the displaced fragments.

Firm traction is applied to the extended elbow to disengage the fragments and release any neurovascular structures which threaten to be trapped between the bone-ends. This should be maintained for a minute or so till all lateral displacement has been overcome. Then, still maintaining traction in the length of the arm, the hand is slowly swung round to flex the elbow and draw the displaced lower fragment from its posterior position into line with the axis of the humerus. If a reduction is secured, it should be possible easily to flex the elbow 20 or 30 degrees under the right angle (i.e. 20 or 30 degrees above the horizontal with the patient standing) without obliterating the radial pulse. In this flexed position the reduction is locked by the support of the triceps and only a collar and cuff is necessary to maintain it. If the elbow can only be made to reach 90 degrees, there is every danger that the fracture will slip because it has not been completely reduced.

Depending on the severity of the swelling and the home circumstances, it is so important to keep a careful watch on the circulation of the hand during the next twenty-four hours that the surgeon will have to consider whether or not this apparently trivial injury should be admitted to hospital for at least one night. If allowed to go home, it should only be with instructions to the parents to bring the child back if *persistent, spontaneous pain is evident*. A supracondylar fracture which has been successfully reduced becomes progressively more comfortable with each succeeding hour; not so the limb which has threatened vascular damage. (Ischæmic Contracture, p. 186.)

After reduction, the elbow is left in its flexed position in a collar and cuff under the shirt, for three weeks, and is then given an outside collar and cuff for another two weeks before allowing full movement of the elbow joint. The recovery of full extension sometimes takes one or two years.

Traction.—When a child with a supracondylar fracture has a very swollen elbow it is often impossible to 'lock' the reduced position by flexing the elbow under the right angle because if this is done the radial pulse may disappear. Rather than take the slightest chance of ischæmia it cannot be too strongly urged that the child should be put to bed with the fracture in a 'semi-reduced' position with skin traction on the forearm, as illustrated in fig. 232. By this means it is possible to maintain a reasonable position and

guarantee an intact circulation. After a week in bed on traction the position will no longer slip and the patient can then walk about with the arm in a simple collar and cuff.

Spontaneous Remodelling.—In young children the capacity of bones to remodel themselves after union has taken place in faulty position is nowhere better exemplified than in supracondylar fractures at the elbow. So vigorous is this capacity to remodel that in children under the age of ten years no risk should ever be taken, in the name of perfect anatomical reduction, by performing multiple re-manipulations, or by open operative reductions. The illustration in fig. 233 shows

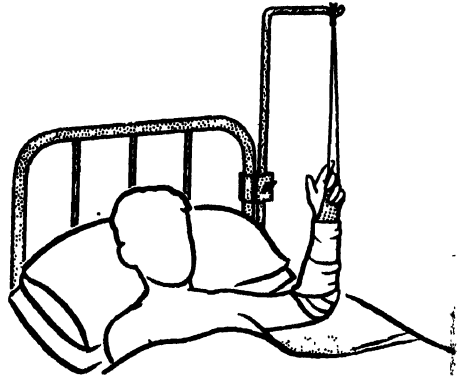


FIG. 232.—Supracondylar fracture treated by suspension with skin traction on the forearm. Note that the elbow is held with a gap of 1 to 2 inches (2.5 to 5 cm.) between it and the surface of the bed.



FIG. 233.—The upper radiograph shows an incompletely reduced supracondylar fracture which remodelled to the appearance seen in the lower illustration over the course of two years with full return of function.

very well the degree of remodelling of a partially reduced fracture which occurred in two years; this is only the radiological appearance of the elbow and clinically a full range of motion was recovered.

Fractures of the Capitellum.—An isolated fracture of the capitellum is a not uncommon injury of childhood and is one of considerable importance. If only slight displacement is present, the elbow can be treated merely in a sling or collar and cuff, and movement started after three weeks or so. Cases with severe displacement are not infrequent, and in these the whole capitellum is often rotated through 180 degrees so that the

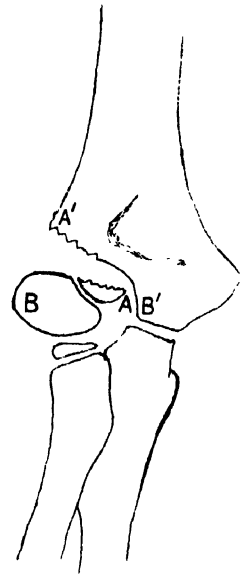


FIG. 234.—Fracture separation of the lateral epicondylar epiphysis of the humerus. The whole capitellum is rotated along a horizontal axis. Reduction aims to restore the relation where A coincides with A' and B with B'.

articular surface of the capitellum faces the fractured surface of the humerus (fig. 234). Unless open reduction is performed, non-union in this fracture is inevitable and non-union of the external condyle results

in the deformity of cubitus valgus with the possibility of late ulnar paralysis (p. 330) due to unequal growth in length of the capitellar and trochlear surfaces of the humerus (fig. 235).

T- or Y-shaped fractures of the lower end of the humerus, usually as a result of a fall on the point of the elbow, are very difficult fractures to treat, and both occur in the adult.



FIG. 235.—Adult non-union of fracture of capitellum neglected in childhood. Eventual ulnar palsy—cubitus valgus.

(i) *Head of Radius*.—Fractures of the head of the radius are most commonly seen in adults, and two main types occur—(a) Chip fractures involving a third or less of the periphery, (b) Comminuted fractures involving the whole of the head with considerable disturbance of the surface articulating with the capitellum. The comminuted type frequently occurs in combination with a transient subluxation of the elbow which may not be recognisable in the X-ray taken later.

Diagnosis is made by X-ray. Small chip fractures are treated by early mobilisation of the elbow joint (fig. 237). Severely comminuted ones are treated by excision of the radial head.

(ii) *Neck of Radius*.—This is usually a green-stick fracture, and if manipulation fails open reduction is necessary. Excision of the head is inadvisable, as cubitus valgus may develop.



FIG. 237.—Chip fracture involving a third of the periphery.

fracture above this level the forearm must be fixed in supination, whereas at all other levels the forearm is best fixed in the mid-position. These positions

Being fractures into a joint, the cardinal principle is early movement, and the modern tendency is to favour this at the expense of the anatomical position of the fragments. Experience has shown that the operative reposition of the fragments may give excellent X-ray appearances, but the functional result is frequently not as good as when the fracture is ignored and early exercise encouraged.

Fractures of the Radius (fig. 236) occur at the following common sites: (1) Head, (2) Neck, (3) Shaft, (4) Colles Fracture, (5) Radial Styloid.

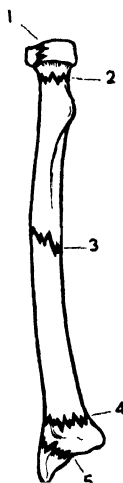


FIG. 236.
—Radius.

(iii) The **Shaft** of the radius can be fractured at any level between the junction of the upper and middle one-thirds, and the middle and lower one-thirds. If above the junction of the middle and upper one-thirds, the upper fragment is supinated by the biceps and supinator brevis, the lower portion being pronated by the pronator radii teres and the pronator quadratus. Therefore with a

are maintained by means of a plaster cast, which extends from above the elbow to the heads of the metacarpals.

When the radius is fractured at the junction of the middle and lower one-thirds (the ulna being intact) there is a strong tendency for the radius to shorten and so cause subluxation of the lower radio-ulnar joint. This does not happen if the ulna is also fractured and shortens an equal amount. This isolated lower one-third fracture of the radius is best treated by open reduction and internal fixation, and this opinion is shared even by fracture surgeons who are otherwise reluctant to employ operative intervention.

(iv) Fractures of the **lower end** of the radius are common, the most important being **Colles fracture**. This fracture commonly occurs in old ladies who fall on the outstretched hand. The fracture is situated 1 inch (2.5 cm.) above the wrist joint, and it is associated with either avulsion of the styloid process of the ulna or rupture of the triangular fibro-cartilage of the wrist joint. The shortening of the radius causes

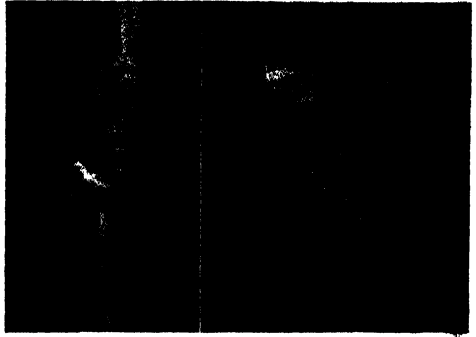


FIG. 238.—Colles fracture. Dorsal displacement—dorsal tilt.

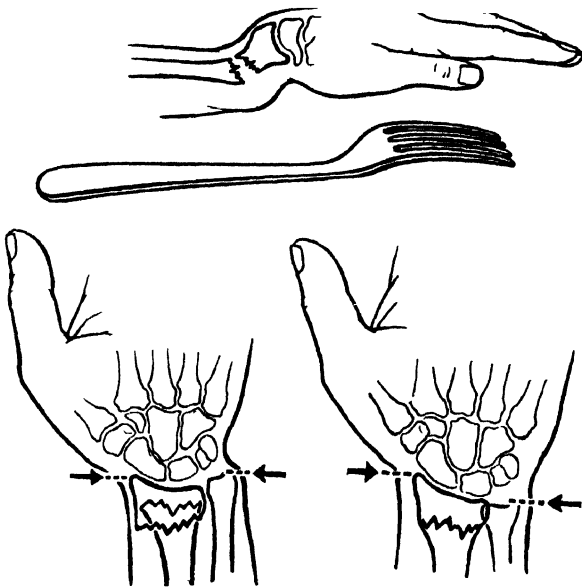


FIG. 239.—Dinner-fork deformity of Colles fracture. Levels of radial and ulnar styloid processes before and after reduction.

subluxation of the lower radio-ulnar joint and excessive prominence of the ulnar styloid. The lower fragment of the radius is displaced backwards and radially, and also rotated so that the articular surface looks backwards, i.e. it is both displaced and rotated in the direction of the violence (fig. 238). These deformities thus produce the clinical 'dinner-fork' deformity (fig. 239). On palpation the radial styloid process, instead of being $\frac{1}{2}$ inch (1.25 cm.) below that of the ulna, is on a level with it.

Smith's fracture, which is very much less common,

is due to falling with the hand behind the body, and the deformity is the reverse of that which occurs in Colles fracture, the distal fragment being

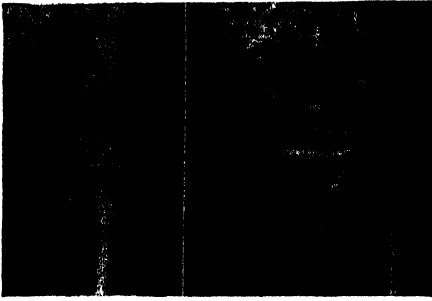


FIG. 240.—Smith's (reversed Colles) fracture.

displaced in front of the lower end of the radius (fig. 240).

Treatment of Colles fracture consists in disimpaction and reduction. General anæsthesia is to be preferred. The surgeon grips the wrist with one hand above and one below the level of the fracture, changing his hands to suit according as the fracture is of the patient's left or right wrist. In the case of the patient's left wrist, the surgeon places the palm of his

left hand on the palmar surface of the patient's wrist above the level of the proximal fragment; the palm of his right hand is then applied to the dorsal surface of the patient's wrist distal to the level of the fracture. It is convenient to have an assistant to hold the patient's elbow and to apply counter-traction.

First Movement.—Disimpaction. The surgeon applies traction with the right hand and gently increases the deformity very slightly by extending the wrist. Still maintaining traction, this is followed continuously by the next movement.

Second Movement.—Palmar flexion. Keeping traction so as to disengage the broken fragments, the distal fragment is gently but firmly flexed. This movement ends by direct pressure being exerted by the right hand on the dorsal surface of the distal fragment and, in an opposite direction, by the left hand against the proximal fragment. To localise these forces they are exerted by the surgeon's thenar eminences over an area of about 1 square inch (6.5 square cm.).

Third Movement.—Still maintaining the differential pressure above and below the level of the fracture, the patient's wrist is now finally pronated by the surgeon forcibly pronating his own right hand. During this movement the surgeon's left hand remains stationary in its original position so as to deter the proximal fragment from following the distal fragment. It will be found that the position of full pronation will also result in ulnar deviation of the patient's wrist, and this is the final position of reduction i.e. (1) slight palmar flexion, (2) ulnar deviation, and (3) pronation.

The reduction is maintained by applying a wet plaster slab to the dorsal and radial surfaces of the wrist and bandaging it in position with a wet gauze bandage. There is very great danger of the fragments slipping during the application of this plaster slab, and it is wise to apply it very quickly so that the surgeon can repeat the previous manœuvre of reduction and can steady the fragments while the slab is setting.

It is important that the slab should be of adequate size. It must extend in length from the heads of the metacarpals to the elbow (but not so long as to prevent flexion of the elbow). In width it must be sufficient to meet at the front of the wrist to leave a gap not more than $\frac{1}{2}$ to $\frac{3}{4}$ inch (1.25 to 2

cm.) wide. To maintain ulnar deviation a tongue of plaster should be passed round the radial aspect of the head of the second metacarpal so as to reach the palm. The final appearance of the plaster in slight palmar flexion and ulnar deviation is seen in fig. 241.

In addition to a routine inspection of the plaster the day after the reduction, it is often necessary to inspect the cast several times during the first ten days, because Colles fracture is often followed by considerable swelling of the fingers, and if tender points develop where the plaster is 'cutting in' the return of adequate finger movements may be delayed.

The patient is encouraged to use the limb, and suitable exercises of the fingers, elbow, and especially the *shoulder* are arranged in the rehabilitation department. Four to six weeks later the cast is removed. Full return of function is usually obtained after two to three months.

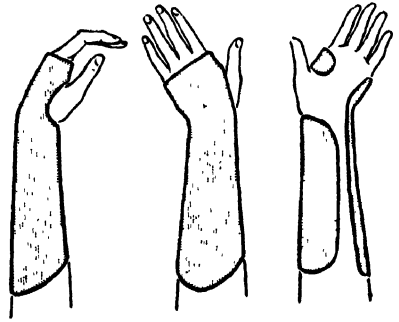


FIG. 241.—Plaster slab in ulnar deviation and slight palmar flexion as used for the Colles fracture.

In the case of feeble old women reduction is often unnecessary, all that is needed being a temporary support to the wrist and encouragement regarding movements of the fingers. In fact, medical advice is not always sought, the condition being regarded as a sprain, and the functional result in these cases is usually very satisfactory though considerable deformity may be evident.



FIG. 242.—Sudeck's osteoporosis.

Sudeck's osteoporosis is an obscure condition which occasionally follows Colles fracture, but which can occur after a mere sprain; it also sometimes affects the foot. As a sequel to Colles fracture it affects the small bones in the neighbourhood of the wrist, and is presumed to be due to reflex hyperæmia occasioned by trauma. It is characterised by pain, vasomotor changes, and atrophy of the bones (fig. 242). It has been mistaken for tuberculous disease, but differs radiologically in that the outlines of all the carpal bones are intact and the bone detail is quite clear, even though so porotic as to appear like a phantom.

Very gradual voluntary movements are indicated, but if the condition persists sympathectomy is rational, provided that relief of pain is obtained to blockage of the stellate ganglion with Novocain.

Rupture of the extensor longus pollicis tendon in about 0.5 per cent. of cases follows a Colles fracture. It is also apt to follow a posterior marginal fracture of the radius. Rupture, due to fraying of the tendon over the ridge of the fracture, occurs a few weeks following the injury. The distal end of the tendon should be sutured to the extensor indicis tendon.

Average period of disability after Colles fracture—light work six weeks, heavy work twelve weeks.

Separation of the lower radial epiphysis is a fairly common injury and in childhood is the counterpart of the Colles fracture. It is most often seen in children from the age of eight to sixteen. The epiphysis is displaced backwards, and the deformity and treatment are similar to those already described in the case of Colles fractures. Usually the displaced epiphysis takes with it a small chip of bone from the dorsal surface of the lower end of

the radius (fig. 243). Reduction of the displacement is usually easy, especially if treated without delay, and the characteristic sensation of 'muffled crepitus' will be felt during the manipulation. This muffled crepitus caused by the grating of bone is against epiphyseal cartilage, which gives a softer sensation than the grating of bone on bone.



FIG. 243.—Slipped radial epiphysis.

One might expect that injury to this epiphysis would cause premature fusion of the epiphysis and late deformity of the wrist due to unequal growth of the radius and ulna, but in fact this is an extremely uncommon complication (unlike the corresponding injury at the lower end of the tibia where arrest of growth of the tibia is common).

Fractures of the **radial styloid** most often occur in men through a fall on the hand, and in the past were one of the so-called 'chauffeur's' fractures sustained by 'back-fires' when cranking internal-combustion engines.

The fracture line, which involves the radio-carpal joint surface, is usually without significant displacement, and often involves a much longer piece of the radius than the term 'styloid process' usually evokes in the mind (fig. 244). Treatment is by a simple 'cock-up' plaster for three weeks and later adhesive strapping.



FIG. 244.—Fracture of radial styloid.

This injury is sometimes associated with dislocation of the carpal semilunar (p. 272) as part of a very serious mid-carpal dislocation—and the inexperienced surgeon may look no farther than the obvious injury to the radial styloid. If the wrist is more grossly swollen than is to be expected from a minor injury such as the radial styloid, the other possibility must be remembered.

Fractures of the Ulna (fig. 245).

(i) The *olecranon process* is usually fractured as a result of falls on the elbow. Separation commonly occurs through the constricted base of the process, and may be almost negligible if the triceps expansion remains unturned. If wide separation is present diagnosis is easy, as the gap between the process and the shaft can be palpated, and the power of extension is lost.

If separation is sufficient to require operation, the fragment is easily fixed in position by means of a vitallium screw (fig. 246), wire, or silk. No plaster fixation is necessary, and active movements are encouraged after two or three days.

(ii) Fractures of the *shaft* of the ulna are due to direct or indirect violence. In the latter case they are sometimes associated with dislocation of the head of the radius (Monteggia's fracture, p. 272). The diagnosis of the ulnar fracture is easy, as the bone is subcutaneous, and displacement or localised tenderness is readily palpable.

(iii) The *styloid process* of the ulna is commonly avulsed in association with a Colles fracture. The maintenance of the hand in a position of adduction, which is an integral part of the treatment of Colles fracture, approximates the position of the ulnar styloid process, but it often remains ununited though usually symptomless.



FIG. 245.—Ulna.

1. Olecranon process.
2. Upper third of shaft, which may be associated with dislocation of the head of the radius.
3. Lower part of shaft, as from direct violence.
4. Styloid process.



FIG. 246.—Fracture of olecranon—internal fixation, early movement.



FIG. 247.—Fracture of radius and ulna treated by plating.

Fractures of the Radius and Ulna are due to direct or indirect violence. In the former case the bones are fractured at approximately the same level, which depends upon the site of injury. If the fractures are due to indirect violence, the radius usually fractures in its lower third, and the ulna about its centre.

Reduction is attempted by an assistant exercising traction on the hand with counter-extension to the upper arm, while the surgeon applies interosseous pressure in order to separate the broken bones. The application of plaster is greatly facilitated by suspending the arm from an overhead support and hanging it by the thumb and index. In this way the traction is maintained while a full plaster is applied. Should manipulation fail, open reduction is advisable and the bones fixed with plates and screws (fig. 247). In some cases operation on the radius alone will be sufficient because the radius suffers more displacement than the ulna.

This injury is very common in children, and is one of the commonest examples of a *greenstick* fracture. The child's forearm is visibly bowed (usually concave dorsally). Reduction is quite simple and it is merely necessary to bend the forearm straight. It is usually advisable to complete the fracture under this bending force in order slightly to over-correct the deformity. If the deformity is not over-corrected, there is a great danger of the original deformity redeveloping inside the plaster. In the forearm frac-

tures of children it is most important to use wool padding as the limb will swell after reduction, especially if the fracture has to be completed, causing the little patient unnecessary pain and even threatening a Volkmann's ischæmic contracture (p. 186).

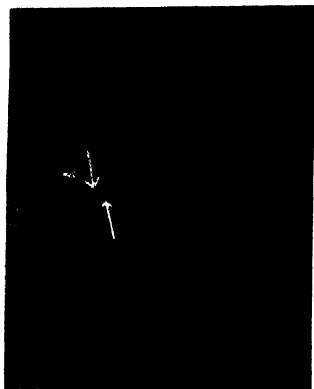


FIG. 248.—Fracture through the waist of the scaphoid. There is abnormal density of the proximal fragment indicating ischæmia of that fragment and threatened non-union.

Carpal Scaphoid.—The commonest bone to be fractured in the wrist is the *scaphoid*. A 'sprained wrist' in a young man after such an incident as falling on the hand, should arouse suspicion. If a fracture is present, inspection of the backs of the hands, with fingers and thumb fully extended, will reveal some fullness of the anatomical 'snuff-box', and on palpation of that area local tenderness is experienced by the patient. X-rays will confirm the diagnosis (fig. 248), and many fractures show best in an oblique X-ray. Some fractures are only visible after two or three weeks (when absorption has occurred along the fracture line). Unless treated efficiently, a fracture through the waist of the bone will pass inevitably into non-union, causing permanent weakness of the wrist and subsequent osteoarthritis. This is a

tragedy in a working man, and the more so since in the majority of cases adequate treatment can prevent non-union. The erroneous diagnosis of a 'sprained wrist' when the scaphoid has been fractured is an oversight which is so well known that courts of law have little sympathy for a medical man making this mistake. The very words 'sprained wrist' should immediately arouse the suspicion of a fractured scaphoid. If localised tenderness in the 'snuff-box' suggests a clinical diagnosis of a fractured scaphoid, even if the X-ray is negative, it is advisable to treat the wrist in plaster and X-ray again after three weeks. The type of patient who sustains a fractured scaphoid greatly narrows down the diagnostic field: a fall on the outstretched hand in an elderly woman or a child is most unlikely to cause a scaphoid fracture; the vast majority of these injuries are in men between the ages of twenty and forty.

As soon as the condition is diagnosed, the hand must be fixed in a 'cock-up' plaster cast. The plaster must not interfere with movements of fingers, and the metacarpal bone of thumb must be included. The cast embraces the sides of the forearm and wrist so as to prevent lateral movement. If 'shearing' movements are allowed to occur between the fractured surfaces, the capillaries attempting to bridge the fracture will be ruptured and bony union thereby discouraged. The plaster is retained for eight to ten weeks. A leather or plastic support may be worn with advantage for another three to six months if there is evidence of delayed union. If the diagnosis is made late, i.e. six weeks or more after the injury, there is no point in applying plaster, and these cases should be encouraged to use the wrist in the hope that a pain-

less pseudarthrosis of the scaphoid will develop, and that symptoms of traumatic arthritis will not occur in the wrist for many years.

Cases of *delayed union*, or *non-union*, of the carpal scaphoid are often treated by bone grafting. There is some doubt whether the results are worth the trouble or whether this operation prevents the development of arthritis. Similarly, operations to excise fragments are nowadays regarded with less favour than formerly. Patients with ununited scaphoids can often enjoy surprisingly long periods of relative freedom from symptoms if their work does not involve heavy strain, and, if comfortable, are perhaps best left alone. In the past it was customary to insist on plaster fixation for six months, or even a year, if radiological evidence of union was not forthcoming; this is no longer considered necessary.

Fractures of the Tubercle of the Scaphoid are often erroneously confused with fractures through the waist and a period of six weeks of irksome plaster fixation inflicted on them. They are fractures which have a good blood supply on both sides of the bone and show no tendency to non-union. Plaster fixation is quite unnecessary and the support of strapping alone is needed for three to four weeks.

Metacarpal fractures involving the shaft of one or more of the inner four metacarpals are produced by the knuckles striking objects, as in boxing, or by objects striking the dorsum of the hand, as in industrial injuries.

Fractures of the shafts of the inner four metacarpals are to a large extent splinted by the adjacent metacarpals. Emphasis should primarily be on early movement, because radiological deformity is of little moment. A little recession of the normal prominence of a metacarpal head is of no significance if full movement and a good grip is present.

Transverse fractures of the metacarpals sometimes give trouble with delayed union, but all other fractures unite well.

If reduction is deemed necessary, simple manipulation of the fractured metacarpals is attempted and the hand enclosed in a 'cock-up' plaster to leave the fingers free.

Fracture of the first metacarpal requires special mention, as a fracture of the base (**Bennett's fracture**), due to a blow on the point of the thumb, may be overlooked. This fracture is sometimes called the 'stave' fracture, which indicates its origin in boxing. Unless efficient treatment is instituted, permanent weakness may result. An oblique fracture occurs through the articular surface of the metacarpal, which allows subluxation of the joint. The shaft of the bone is drawn backwards and outwards (fig. 249).



FIG. 249. — Bennett's fracture-dislocation.

This fracture-dislocation is easily reduced by traction and pressure over the base of the thumb. Continuous traction is best obtained by means of a plaster cast applied to the forearm, in which is incorporated a loop of thick wire to take the adhesive traction applied to the end of the thumb. It is often possible to reduce and hold this fracture without traction, provided the metacarpal is in full extension and local pressure moulds over the base of the bone to hold it in.

Phalanges are commonly fractured by direct violence, e.g. by a blow with a hammer, or crushed by a door or slipping window-sash.

Fractures of the proximal phalanges are more difficult to treat than metacarpal injuries because the flexor tendon sheath will be involved if the fracture is displaced. It is often difficult to align the injured finger with the other digits if splinted separately, but these errors can be minimised if the fractured digit is splinted side by side with an adjacent normal finger. A slab of plaster, suitably curved so as to give a fulcrum on the palmar aspect of the proximal phalanx over which the finger can be flexed, will eliminate the dorsal concavity which is the constant deformity in these fractures (fig. 250). It is rarely necessary to immobilise the digit more than three weeks, and thereafter the callus will be strong enough to allow movement without redisplacement occurring. In the later stages of rehabilitation the simple manoeuvre of fixing the finger to the next digit with adhesive strapping is useful in recent fractures with little or no displacement.



FIG. 250.—Typical angulation in fracture of shaft of phalanx.

Ribs and Sternum.

Fractures of the **Ribs** occur as a result of direct or indirect violence. In certain nervous diseases, e.g. tabes dorsalis, very slight trauma may cause a fracture and pathological fracture occurs in secondary malignancy, multiple myelomatosis, and osteomalacia.

(i) *Fractures due to indirect violence*, by crushing of the chest, may be single or multiple and usually involve the fifth to the eighth ribs a short distance in front of the angle of the rib. This site is the junction of the long anterior curve and the short acute posterior curvature.

The fracture is suggested by the history of injury and localised pain on deep inspiration. Pain is referred to the site of fracture if simultaneous pressure is exerted upon the sternum and spine.

(ii) *Fractures due to direct violence* affect the ribs most exposed to injury. The first and second ribs are rarely fractured, as they are protected by the clavicle, and trauma sufficiently severe to smash the clavicle and upper ribs is likely to inflict fatal injuries on adjacent structures. Similarly, the lower two ribs are protected by muscles, and enjoy a degree of mobility which diminishes the risk of fracture.

Fractures due to direct injury are more serious than those due to indirect causes, as bone fragments may be driven inwards and damage the pleuræ, lungs, diaphragm, liver, kidneys, spleen, pericardium, or heart. Surgical emphysema is likely to follow laceration of the lung.

The treatment of fracture of the ribs depends on the severity of the pain and distress. Often no treatment is needed. Major damage to the thoracic cage, 'stove-in' chest, is quite a different matter from ordinary rib fractures and is so serious that its urgent treatment takes precedence over any other injury. (This is described on p. 647.)

In elderly patients chest complications may endanger life. Local anæsthetic injected at the site of the fractures gives a temporary relief from pain, so that breathing and expectoration can be performed in comfort. Strapping of ribs is no longer popular on scientific grounds, but it may afford the patient some satisfaction.

A close watch is kept for evidence of hæmothorax or pressure pneumothorax (p. 646).

Ribs, in spite of lack of immobility, unite readily, and support for three weeks is usually sufficient.

The **Sternum** is occasionally fractured as a result of direct violence, e.g. by the steering-wheel of a car as the result of a collision. If displacement is gross, death is likely, owing to injury to or pressure on the heart and great vessels. In less severe cases the patient is confined to bed with a pillow or sandbag between the shoulders, and strapping applied to steady the fragments.

Fracture due to indirect violence can be associated with a fracture-dislocation of the spine, due to excessive flexion of the trunk. Treatment is symptomatic, as the more serious injury to the spine takes precedence.

LOWER EXTREMITY

Fractures of the Pelvis comprise fractures of the iliac crests or of the true pelvis.

(i) Fractures of the **Iliac Crests** are not of any significance because displacement is slight, as the iliacus muscle on the inner side, and gluteal muscles on the outer side, support the bone.

A firm flannel bandage is applied to the pelvis and the patient confined to bed till symptoms have settled, which usually will take two to three weeks.

(ii) Fractures of the **True Pelvis** can occur either in the oblique diameter, i.e. through the obturator foramen on one side and the ala of the sacrum on the opposite side, or the pelvic ring can be fractured in two places on the same side. The cause is a severe crush, such as a horse rolling over its rider or a light car passing over a pedestrian.

Occasionally the injury may not be suspected if it results from a simple domestic accident to an elderly patient, such as a fall down the stairs. Clinical examination of the pelvis may not be conclusive if there is no displacement or crepitus and if there are local bruises which might themselves explain the pain; but very suggestive of a fracture of the pelvis in these circumstances is a degree of shock greater than might be expected from a contusion, and inability to walk or stand alone.

The most important aspect of a fractured pelvis is the liability to injury of viscera—the male urethra commonly, the bladder in both sexes occasionally, and the rectum rarely. Therefore the patient should be instructed not to attempt to pass urine until an exploratory catheterisation can be made under fully aseptic conditions (Chap. 46). Blood escaping from the external urinary meatus in the male immediately suggests rupture of the urethra (Chap. 48).

A rectal examination is made, and in rare cases a fragment of jagged bone may be encountered, in which case laparotomy will be necessary, and iliac colostomy. Wounds of the vagina are rare and can usually be sutured.

Having methodically excluded or dealt with visceral lesions, attention

can be directed towards the fracture, but if the patient is seriously ill from the complications, no special measures need be adopted. A plaster cast is unnecessary in fractures of the pelvis, and nursing between sandbags is adequate. When separation of the symphysis occurs the pelvis can be compressed by suspending it in a pelvic sling from an overhead beam. In simple cases nursing the patient on his side will close a separated symphysis without any external apparatus.

The pelvic bones readily unite, but walking is prohibited for two to three months, depending on the site and nature of the fracture.

Fractures of the acetabulum occur either in connection with dislocation of the hip joint (p. 273), when the posterior margin is broken off, or following a heavy fall on the great trochanter, in which case a 'central dislocation' may be produced.

The **sacrum** is fractured by either direct or indirect violence. The latter is usually associated with fracture of the pelvis and anaesthesia of part of the perineum may result from damage to sacral nerves if sacral foramina are involved in a displaced fracture. Direct violence, as by falls or kicks, rarely produces any deformity and often deforms.

The **coccyx** may be fractured as a result of kicks or falls. Pain, which is often severe, occurs on walking, sitting, or actions which cause contraction of the levator ani, such as defaecation or coughing. Rectal examination reveals local tenderness and often deformity.

In some cases these symptoms are produced when no fracture is evident, the condition being known as *coccydynia*, a condition of unknown origin.

If adequate rest and symptomatic treatment by heat or anaesthetic injections are of no avail, excision of the coccyx should be performed, but there should be no haste in making this decision as many of these patients are neurotic and invent some other complaint after operation.



1. Subcapital and mid-cervical fracture of the neck.
2. Basal fracture of the neck.
3. Pertrochanteric fracture.
4. Spiral fracture of upper third of shaft.
5. Transverse fracture of the middle of shaft.
6. Junction of middle and lower one-thirds.
7. Supracondylar.



FIG. 251.—Femur.

Fractures of the femur are of the greatest surgical importance, both on account of the difficulties in treatment and also because of the serious disability if treatment is inefficient.

(i) **Fractures of the Neck of the femur.** Fractures of the neck of the femur since the days of Sir Astley Cooper have been divided into *extracapsular* and *intracapsular* fractures. This broad division is still a good one, as it emphasises the two different problems which treatment presents. In the absence of treatment, *intracapsular* fractures are almost totally devoid of any natural capacity to unite, because

the fracture frequently deprives the head of the blood supply it receives by way of the neck. In a minority of cases where the *intracapsular* fracture is impacted, union may occur without treatment, but in all others it is imperative to reduce the fracture accurately and to hold it completely immobilised by internal fixation with one of the various types of nail or screw.

Extracapsular fractures, on the other hand, occur in the cancellous bone of

the trochanters at the base of the neck, and the ability of the bone to unite is good because there is no disturbance of blood supply. The essential difficulty in this group of fractures is the advanced age of the patients most commonly afflicted. In general, the extracapsular fracture is an injury of the senile (seventy years or more) and these patients have such disuse atrophy of their femoral necks that it takes only the slightest trip or stumble to cause them to fracture. Therefore the problem in treating this fracture is often the problem of old age rather than any inherent difficulty in the treatment of the fracture itself. The problem of the *intracapsular* fracture is not so much the age of the patient but rather the technical difficulty of its strong tendency to non-union.

Whenever possible, the ideal method of treating both these fractures is operative because (1) in the intracapsular fracture, operation is necessary to get fixation and union, and (2) in the extracapsular fracture, operation is necessary to get the patient out of bed early so that the ill-effects of decubitus in the elderly will be prevented.

Intracapsular Fractures.—These are anatomically subdivided into subcapital and mid-cervical fractures. The danger of ischæmia of the femoral head is greater in the subcapital fracture than in the mid-cervical. This is because the synovial membrane of the hip-joint does not extend as far down the back of the neck of the femur as down the front, and therefore some blood can still supply the head in most mid-cervical fractures.

The deformities which result from all fractures of the femoral neck (at whatever level) are twofold: external rotation and coxa vara (and hence shortening). The element of coxa vara (*syn.* adduction fracture) is the more serious because the fracture line is thus exposed to a shearing strain if the patient attempts to take weight. The shearing strain in coxa vara is an important factor in the tendency to non-union in the fracture. In impacted fractures the deformity is usually negligible. Sometimes a coxa valga is produced (*syn.* abduction fracture), and in this position the strain of weight-bearing will further impact the fracture. This latter group (unfortunately not common) needs no special treatment other than bed-rest and protection from full activity for three or four months.

Treatment.—The treatment of mid-cervical and subcapital fractures is operative. If the patient is very old and shocked, it may be necessary to delay surgery a day or two until resuscitated.

The technique of internal fixation in universal use is the insertion of a Smith-Petersen tri-flanged nail under X-ray control. Under gas and oxygen anæsthesia this is not an operation which causes shock, as the operative exposure is very small.

The hip is manipulated and the patient fixed on a special operating table incorporating traction to the feet and counter-traction via a post in the perineum. The hip is held with traction in abduction and internal rotation. If the reduction is satisfactory (as checked by X-ray of the neck of the femur in two planes), a small lateral incision is made over the great trochanter and a guide wire 2 mm. in diameter is inserted into what is estimated as being the line of the axis of the femoral neck. X-rays are taken, and if the direction of the guide has to be changed, various devices are available for directing a second wire in relation to the first so as to be exactly in the axis of the neck. The tri-flanged nail is provided with a central cannula, 2 mm. wide, which enables it to be slid over the guide wire and hammered into the head. After finally

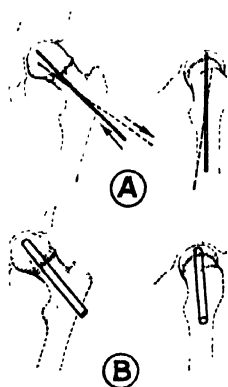


FIG. 252.—A. Insertion of guide wires. The one in good position is retained. B. The nail is inserted and the wire withdrawn.

(A.P. and lateral views.)

impacting the fracture, the wound is closed and the patient is returned to bed without any fixation (fig. 252).

After operation the patient can be allowed out of bed in a few days, but weight-bearing is not permitted until some evidence of osseous union is suggested in the X-ray—which may take three to four months. Sixty to seventy per cent. of these cases achieve a result which is indistinguishable from normality.

Complications.—Non-union of the neck of the femur occurs in approximately 30 to 40 per cent. of all intracapsular fractures, even when skilfully treated by the Smith-Petersen nail. This is in part due to mechanical inadequacy of the nail as a method of fixation in very comminuted fractures, and in part the result of ischæmia of the femoral head.

Non-union can be treated by the following methods :

(1) *Subtrochanteric osteotomy* with displacement of the shaft under the head. In this way the shearing strain of weight-bearing is taken off the fracture line (fig. 253). This is only to be advised if the head is thought to be viable.



FIG. 253.—Smith-Petersen nail being extruded from intracapsular fracture. Treatment by displacement osteotomy.

(2) *Replacement of the head* by a prosthesis of stainless steel (Thompson or Moore prosthesis) especially if the head is ischæmic.

(3) *Insertion of a bone graft* (suitable only in cases where the head is viable with an intact blood-supply and the patient is relatively young).

Extracapsular Fractures.—Two subdivisions of this fracture are recognised on anatomical grounds, though the distinction has no bearing on treatment: (1) *Basal fractures* are those occurring just where the base of the

neck springs out of the trochanters; (2) *Pertrochanteric fractures* where the fracture lies entirely within the trochanteric mass. In the latter the degree of comminution may be very great and the neck, lesser and greater trochanters, and the upper end of the shaft may all be separate mobile fragments.

These injuries are becoming more common than the intracapsular fractures because they usually occur in the oldest age groups and the average age of the population is steadily increasing.

Clinically the diagnosis is made, as in the case of the intracapsular fracture, by the combination of external rotation and shortening (which indicates traumatic coxa vara). In the pertrochanteric fracture the degree of external rotation is considerably greater than in more proximally sited fractures of the neck. In some pertrochanteric fractures the foot of the affected extremity will be found to be lying with its outer surface flat on the examination couch, so indicating a full 90 degrees of external rotation. On the other hand, an intracapsular fracture of the femoral neck will have an external rotation of perhaps only 45 degrees.

This important clinical point can be remembered if the mechanical explanation is understood. External rotation after fracture of the neck occurs in the long axis of the femoral shaft, and the 'stub' of the fractured neck will rotate so that the broken end tends to face forward. If the stub is short or non-existent (extracapsular), there is nothing to prevent full external rotation, but if the stub is long (as in a subcapital fracture) it would have to sweep round in an arc of considerable radius and the presence of the encircling capsule will prevent this.

Treatment.—It is quite a simple matter to treat a pertrochanteric fracture, without operation, merely by applying weight-traction to the limb and confining the patient to bed for three months until the fracture is clinically united. The traction will restore the length, and if the leg is abducted and internally rotated this will correct the deformities of coxa vara and external rotation. Because many of these old patients are incontinent, it is difficult to prevent bedsores if the patient cannot be turned periodically on to the face, also senile dementia is common and may make the maintenance of an efficient traction system impossible.

Internal fixation of the pertrochanteric fracture is therefore more attractive than conservative treatment, but it requires a rather larger internal splint than the Smith-Petersen tri-flanged nail. The apparatus used is a 'blade-plate' which has one limb screwed to the shaft of the femur and the other limb driven into the neck of the femur (fig. 254). Without the angle-iron principle the long proximal fragment in the extracapsular fracture, comprising head and neck, offers such leverage that the force causing coxa vara is

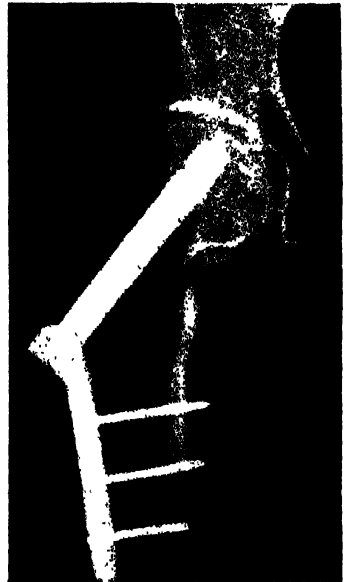


FIG. 254.—Pertrochanteric fracture treated by 'blade-plate'.

too great to be resisted by a simple Smith-Petersen nail. The instrument is inserted with X-ray control very much in the same way as described for intracapsular fractures. The patient is allowed out of bed in a chair within a few days. There are no failures of union after this method, as the blood supply of the fragments is good and the only mechanical failures are technical.

Nélaton's Line, Bryant's Triangle.—It is convenient at this point to describe two classical measurements which formerly were important in the clinical diagnosis of hip conditions in the pre-radiological era. Though not often needed at the present day, every educated surgeon should know them.

Both these tests demonstrate the presence of shortening between the pelvis and the great trochanter of the femur; that is to say, shortening which could be caused by dislocation of the hip, fracture of the neck of the femur, coxa vara, tuberculous destruction of the femoral head with 'wandering acetabulum', etc.

Nélaton's line is a line drawn between the anterosuperior iliac spine and the most prominent part of the tuber ischii; in the normal hip the tip of the great trochanter should lie on this line, but if the neck of the femur is short the tip of the great trochanter will be at a higher level.

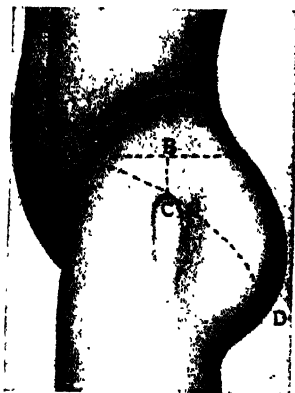


FIG. 255.

A D = Nélaton's line.
A B C = Bryant's Δ , of which B C
is the essential measurement.

To carry out the test the patient should lie on his side with the affected hip uppermost and slightly flexed so that the knee falls slightly forwards. The tip of the great trochanter is palpated and a skin mark made to indicate the level. Nélaton's line is then marked by passing a tape measure from the anterosuperior iliac spine round the great trochanter to the tuber ischii (fig. 255).

Bryant's triangle is another way of arriving at the same result. The patient lies supine and the tips of both great trochanters are palpated and skin marks made to indicate their levels; perpendiculars are then dropped from the anterosuperior iliac spines and the distances from the tips of the trochanters to the perpendiculars are measured on both sides. If the neck of the femur is short on one side, the distance to the perpendicular will be correspondingly reduced. It will be noted that though it is traditional to call this Bryant's 'triangle', the hypotenuse of the triangle is not needed.

In obese patients it may be difficult to detect the precise position of the tips of the great trochanters to within an inch, and as the test is only of value for differences of this magnitude, it will be realised that the modern usefulness of the test is limited, even though the instructional value and clinical exercise is still of importance.

Separation of the epiphysis of the head of the femur is not an uncommon occurrence, but it is not a simple traumatic lesion. The centre appears during the first year, and unites at the age of twenty. The clinical features resemble those of an intracapsular fracture, see *slipped epiphysis* (p. 338).

Avulsion of the lesser trochanter is sometimes caused by sudden contraction of the ilio-psoas. The commonest age for this accident is about puberty. The condition is unlikely to be diagnosed without the assistance of X-rays. Treatment consists in immobilising the limb for four weeks in slight flexion.

(ii) Fractures of the Shaft of the femur.

Displacement depends on the direction of violence, muscular contraction, and gravity. In the upper third the upper fragment is flexed by the ilio-psoas, abducted by the gluteal muscles, and everted by the external rotators. The lower fragment is adducted by the adductor muscles, drawn proximally by the hamstrings and quadriceps, and everted by the weight of the limb.

In middle-third fractures and lower-third fractures, the deformity is one of backward angulation and shortening. The distal fragment is the main element in the backward angulation (fig. 256).

In rare fractures just above the condyles, the distal fragment is often rotated backward through 90 degrees and may press upon the popliteal artery to cause gangrene of the foot if the obstruction is not quickly rectified.

Aims of Treatment.—There are three essential aims in the treatment of a fracture of the shaft of the femur: (1) restoration of alignment, (2) restoration of length, (3) prevention of knee stiffness.

(1) *Restoration of alignment* is essential in the femur because mal-alignment throws an abnormal strain upon the knee joint, and osteoarthritis is prone to develop in later life.

(2) *Restoration of adequate length* is essential unless the patient is to wear an ugly raised shoe. Even so, one need not fear even as much as $\frac{3}{4}$ inch (2 cm.) of shortening, as this amount is easily concealed by tilt of the pelvis, and slight shortening greatly favours rapid and sound osseous union. Almost all the ill-effects of treatment can be traced to over-anxiety to restore full length, and these ill-effects are the result of delayed union.

(3) *Severe knee stiffness* is a common complication of fractures of the shaft of the femur, especially in the lower half, as a result of fibrosis of the muscles of the extensor mechanism. Delayed union is especially prone to result in permanent knee stiffness. It is preferable to have a femur which is a little short with a fully mobile knee, than a femur which is full length with delayed consolidation and limitation of knee movement.

Traction and Counter-traction.—Because of the muscular contraction by the powerful muscles of the thigh, an untreated fracture of the femur may easily develop 4 or 5 inches (10 or 12.5 cm.) of shortening ('over-riding') and the maintenance of length against this constant tendency to shorten is the special problem presented in this fracture. It is therefore essential to have a clear understanding of the principles of traction and counter-traction in fracture treatment.

In most fractures, other than that of the femur, it is a common practice to overcome shortening by a manipulation under anaesthesia and, if the bone-ends have been successfully 'hitched' against each other, then to maintain this length merely by encasing the limb in plaster. This is quite impossible

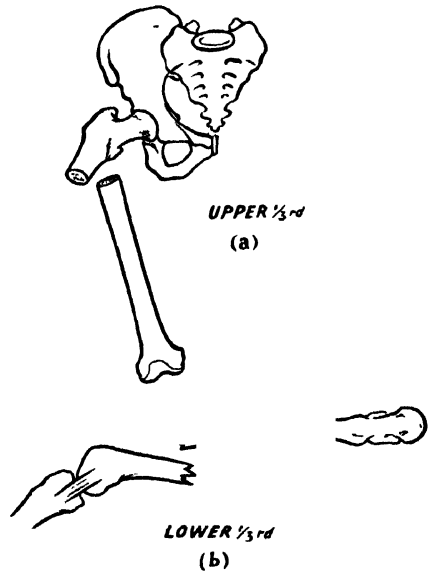


FIG. 256.—Characteristic deformities of fractures in upper and in lower thirds of shaft of femur.

in the femur because, even if the fragments could be 'hitched', the soft bulk of the thigh would allow them to slip again soon after the plaster was applied. If the fracture is oblique or comminuted it would be impossible to obtain stable end-to-end contact against muscle tone.

In applying *traction* to the distal fragment so as to hold the thigh muscles at normal (or nearly normal) length, it is always necessary to use *counter-traction* to the proximal fragment to prevent the trunk and pelvis following the traction force and again allowing the thigh to shorten.

There are two entirely different methods of applying traction and counter-traction and both have their own special spheres of usefulness: they are (1) Sliding traction and (2) Fixed traction.

(1) **Sliding Traction** (*syn.* Weight Traction, Balanced Traction).—There are dozens of modifications of detail in applying sliding traction, but only the essential principles need be considered:

(a) *Traction* is applied to the distal fragment by weights and pulleys attached to the limb either by skin strapping or by skeletal traction. Skeletal traction is to be preferred as it is more comfortable and more precise; a

4-mm. stainless steel Steinmann pin is driven into the upper end of the tibia for this purpose and a Böhler stirrup attached.

(b) *Counter-traction* is provided by using the weight of the body; this is brought into action by elevating the foot of the bed so that the patient's trunk and pelvis tend to slide away from the source of traction. With a traction force of 20 lb. (9 kg.) the foot of the bed is raised about 9 inches (22.5 cm.) (fig. 257).

(c) *Support for the Back of the Thigh*.—This can take almost any

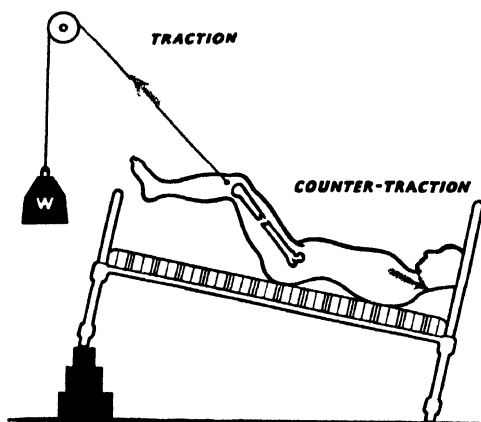


FIG. 257.—Principal elements in sliding traction: traction and counter-traction.

form and is the source of most of the detailed variations in any sliding traction system.

Because of the tendency for the thigh to sag under the influence of gravity, the original deformity of backward angulation tends to persist and must be controlled by slings or supports under the thigh to restore its normal anterior bowing. Frequently support to the thigh is further complicated by apparatus for permitting knee movement, for holding the foot in dorsiflexion (to prevent equinus deformity), and to counterpoise the whole apparatus so that it floats with the patient as he moves in bed.

Sliding traction is attractive in that it is very comfortable. It is especially useful in fractures of the shaft of the femur at, or above, the upper third (perthrochanteric fractures also). The main danger of this method is that bone contact may be lost ('distraction') through excessive traction, and if the traction force has therefore to be reduced, the fixation and alignment of the femur is prejudiced.

(2) **Fixed traction** is quite unlike sliding traction, and because it depends entirely on the use of the Thomas splint, its modern application hardly differs from the original method used by H. O. Thomas nearly one hundred years ago.

In this method of traction, cords are attached to the distal fragment, either by skin extensions or by a skeletal pin, and after passing the Thomas splint over the limb so that the padded leather ring takes a purchase against the ischial tuberosity, traction is applied by tying the cords to the foot of the splint. Countertraction is thus exerted by the pressure of the ring against the soft tissues and the tuber ischii (fig. 258).

The tendency to backward angulation of the fractured shaft of the femur is combated by slings passed between the side-bars of the splint behind the thigh. Fixation of the fracture is obtained by enclosing the limb and the splint in a bandage.

It is evident that this method can exert only a relatively slight traction force, because the skin of the perineum would not be able to withstand the equal and opposite counter-traction force as great as that used in sliding traction. The great art of this method is to get results with minimum force. It is a fundamental requirement, therefore, that the fracture should be capable of reduction under anæsthesia and that the Thomas splint with fixed traction be used merely to *retain* a reduction already achieved.

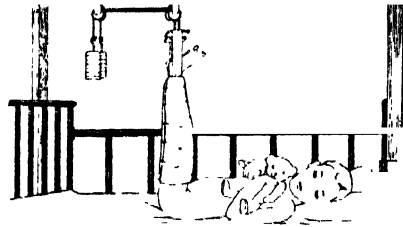


FIG. 259.—A 'gallows' splint.

A *gallows splint* (fig. 259) is useful for children below the age of five years. Traction is applied by means of strapping, and the legs are slung up to the cross-piece, so that the pelvis is just lifted from the mattress. The child's weight acts as counter-traction, and this position is very convenient for nursing purposes.

Internal fixation is becoming increasingly popular but is best avoided in comminuted fractures. The intramedullary nail is the best form of internal fixation because no periosteal stripping is needed to insert it. The exposure should be by the postero-lateral route to avoid damaging the quadriceps muscle and causing knee stiffness.

Separation of the lower epiphysis was more common in the days of horse-drawn vehicles, when children enjoyed the excitement of riding on the rear axle. Entanglement of the foot in the spokes caused violent hyperextension of the leg and forward separation of the epiphysis. The lower end of the diaphysis projects backwards, and gangrene sometimes followed pressure on the popliteal vessels. The deformity is reduced by traction on the flexed knee with the patient lying on his back on the floor, the pelvis being fixed by assistants. After reduction the limb is bandaged in slight flexion, the pulsations of the dorsalis pedis artery being a guide

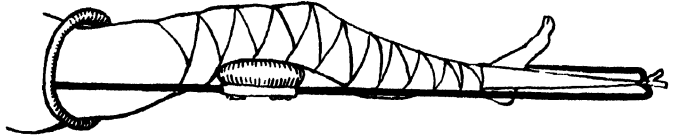


FIG. 258.—Thomas splint with fixed skin traction. Counter-traction against ring.

to the circulation of the limb. If manipulation fails, open operation must be performed.

Fractures of the Patella are due to direct or indirect violence.

If due to *direct violence*, a comminuted or star-shaped fracture usually results. Separation of the fragments is not extensive, and may be absent, owing to the intact aponeurosis of the quadriceps expansion and periosteum, which hold them in position. Considerable bruising and effusion into the joint are to be expected. Aspiration is often advised, but is rather meddlesome as the joint is full of solid blood-clot. A posterior plaster slab is applied for a week, after which active movements are encouraged.

Fractures due to *indirect violence* occur when the knee is semi-flexed. The fracture in this case is typically *transverse* (fig. 260). In this position



FIG. 260.—Transverse-fracture of patella.

the patella is balanced on the front of the condyles, and sudden contraction of the quadriceps, as in an effort to regain balance, snaps the bone in the same manner as a stick is broken across the knee. Local pain and loss of power to extend the leg are prominent symptoms. The joint rapidly fills with blood, and the gap between the fragments can sometimes be seen, and in any case is readily palpable, this feature distinguishing the condition from rupture of the quadriceps tendon (p. 305). Owing to separation of the fragments and the interposition of torn aponeurosis, fibrous union will occur unless operation is undertaken. Moreover, in cases treated conservatively, the band of fibrous tissue which unites the fragments will stretch, and eventually the fragments will be widely separated.

Treatment is operative, and for transverse fractures with separation the aim is to coapt the fragments by internal fixation.

The flaps of aponeurosis which cover the raw surfaces of the fragments are excised and blood-clot scraped away with a sharp spoon. The bone is drilled and approximated by strong braided silk or wire; some surgeons prefer to use a screw. After the fragments are approximated the torn quadriceps expansion is carefully sutured on each side of the patella and the wound closed. Though the internal fixation may be strong, it is advisable to splint the knee for about four weeks before starting active flexion movements.

Excision of the whole of the fractured patella is often indicated if the fracture is comminuted (Brooke), but the cosmetic effect is unsightly, especially in young women, and this may prohibit the wearing of a mini-skirt!

The fragments are easily dissected from the tendinous attachments, and the torn aponeurosis is sutured. The gap resulting from excision of the patella is closed with vertical sutures as accurately as possible, but some slight inadequacy is of no moment. As with all operations on the knee joint, a firm bandage is applied over voluminous wool dressings to control oozing. Active contraction of the quadriceps is begun after three days and graduated active flexion in one week. Patients are enabled to return to work in from three to six months, according to the nature of their occupation.

Fracture of one patella is occasionally followed by fracture of the other at a later date; there is no clear reason for this tendency.

Fractures of the Tibia (fig. 261).

(i) Fractures of the *upper end* are usually due to direct violence, one or other tuberosity being separated, and occasionally a Y- or T-shaped fracture is produced. The knee joint is involved and rapidly fills with blood. A 'bumper' fracture is due to a severe blow on the outer side of the joint, e.g. impact with the bumper of a car which strikes the outer side of the leg just below knee level; the external condyle of the femur impinges on and crushes the outer tuberosity of the tibia (fig. 262). Because these fractures involve joint surfaces, the ideal treatment combines accurate restoration of the joint surfaces with early mobilisation. In young people it is sometimes advisable to perform open reduction and fix the fragments at the correct level by means of a screw.

Most cases, however, do remarkably well with early joint movement provided that there is no gross valgus deformity and that weight-bearing is delayed. This 'bumper' fracture is one which is very frequently seen in elderly patients knocked down by motor-cars, and these patients do extremely well with early mobilisation and the acceptance of 10 or 20 degrees of valgus deformity. In these elderly patients a mobile knee, capable of bearing weight in less than three months, is the prime object of treatment and, as most of these patients are women, even a severe valgus deformity is easily concealed by the skirt.



FIG. 262.—'Bumper' fracture.

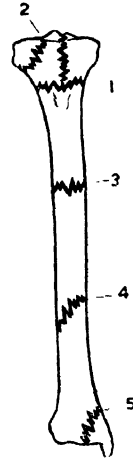


FIG. 261.—Tibia.

1. Y-shaped fracture involving the knee joint.
2. Outer tuberosity.
3. Transverse fracture due to direct violence.
4. Oblique and spiral fractures due to indirect violence.
5. Internal malleolus.

(ii) Fractures of the *shaft* of the tibia, without implication of the fibula, are usually due to indirect violence, in which case the fracture is oblique or spiral. Diagnosis is easy, as

the bone is subcutaneous and irregularity or localised tenderness is readily palpable.

Treatment.—When the fibula remains intact, the displacement of a fractured tibia is relatively slight, and as angulation is rarely more than 5 degrees, it is usually sufficient to apply a simple plaster without any attempt at reduction. Manipulative reduction is obviously unlikely to succeed (even if needed) as the intact fibula would obstruct manipulative manœuvres.

Though it might appear that fractures of the tibia when the fibula is intact would be unlikely to give rise to difficulty in treatment, in fact delayed union is not uncommon. It is probable that the intact fibula acts as a 'strut' to prevent the fragments of the tibia coming into close contact. For this reason there are many surgeons who believe, with some justification, that these fractures are best operated on, and fixed with screws and plates. This operation is certainly to be considered in the adult, but is unnecessary under the age of about twenty years, because the powers of fracture union in youthful bone are very great.

Fractures of the shaft of the Fibula alone are usually due to direct violence. The procedure of 'springing' the fibula assists diagnosis, and consists in compressing the fibula against the tibia, local pain being referred to the site of a fracture. A radiograph confirms the diagnosis, and treatment, if any is required, need be nothing more rigorous than the application of an adhesive support.

Fractures of the Tibia and Fibula commonly occur either as a result of direct or indirect violence and are very frequently seen following traffic accidents. In direct violence the bones are fractured at the same level, but if due to indirect violence the tibia is usually broken at the junction of its lower and middle thirds, and the fibula at about its centre. In the fractures of civil life the tibia is the most common *compound* fracture because it is subcutaneous bone in the whole of its length, and displacement is likely to cause one of the fragments to penetrate the skin.

With the help of modern antibiotics, the treatment of open or closed fractures of the leg bones is very much the same. An open fracture must be converted into a closed fracture by cleansing the wound, excising contused tissue, and then closing to cover exposed bone. In some cases an immediate skin graft may be necessary.

In some severely comminuted fractures, with gross soiling by road dirt, it may be better to amputate forthwith if complicated by nerve or vascular injury.

Fractures of the leg bones can be immobilised either (a) by plaster after manipulative reduction, (b) by skeletal traction through the lower end of tibia or the os calcis, or (c) by internal fixation with plates and screws or an intramedullary nail.

There is a strong argument for delaying for a week or two the internal fixation of fractures of the tibia in order to avoid the suppression of callus formation which can result from very early intervention.

In general, the tibia takes about twelve weeks to unite, and it is rarely fit to take weight without external support under sixteen weeks.

Fractures involving the Ankle Joint

Specialised text-books of fracture surgery devote much space in attempts to classify the fractures which involve the ankle joint; the classification is based on the direction of the force causing the injury. Even the most elaborate classifications fail to cover every fracture encountered, and therefore, for the sake of simplicity, only a broad generalisation will be given.

There are four principal directions in which injurious forces can be transmitted to the ankle: (1) *Internal rotation*, (2) *External rotation*, (3) *Inversion*, (4) *Eversion*. Some text-books use the terms 'abduction' and 'adduction', and in this description these are taken as synonymous with 'eversion' and 'inversion'. The joints of the tarsus are much more flexible in the direction of inversion and internal rotation than in eversion and external rotation. Inversion and internal rotation therefore produce the common *sprained ankle* in which the external lateral ligament of the ankle joint is partially torn. On the other hand, external rotation and eversion forces the tarsal joints into a solid bone-block, and the force is thus expended on the ankle-joint to produce a fracture. By far the commonest fracture, and the least important, is the isolated, and undisplaced, fracture of the *external malleolus*. This is generally an oblique or spiral fracture involving the lower end of the fibula for about 2 inches (5 cm.) and extending into the malleolus itself.

Fracture of the external malleolus is diagnosed clinically by the site of maximum tenderness being over the bone of the lower end of the fibula; this simple clinical test differentiates it from *sprain of the ankle*, where the maximum tenderness is just below and in front of the tip of the external malleolus.

Treatment.—Fractures of the external malleolus do not need reduction because they are undisplaced. If very little swelling is present, strapping is all that is necessary and, with active movements, full function returns in six weeks. If excessive swelling is present, it may be advisable to apply a walking plaster for four weeks, and this will enable the patient to get about his business more quickly than with strapping. Whatever the treatment, if the fracture is undisplaced the result will be 100 per cent. successful.

Pott's Fracture.—The essential features of the Pott's fracture are that (1) it is a fracture-dislocation of the ankle joint, and (2) it requires accurate reduction and fixation. Various patterns of Pott's fracture are recognised, and these differ as the result of the different types of violence causing them.

The common Pott's fracture results from a continuation of the same force which causes an uncomplicated fracture of the external malleolus—i.e. external rotation and eversion. The external malleolus is fractured spirally (because of the rotation element) or obliquely; this is then followed by fracture of the tip of the medial malleolus and thereafter by postero-lateral subluxation of the talus from its surface of contact with the tibia (fig. 263 a). In this movement a chip of bone is frequently avulsed from the posterior surface of the tibia, the so-called 'third malleolus'.

Percival Pott, 1714–1788, Surgeon to St. Bartholomew's Hospital, London, wrote surgical papers while recovering from a compound fracture of the tibia and fibula.

Two other types of ankle fracture, not quite so common, are also easily recognised :

(1) *Eversion Fractures* (*syn.* abduction fractures).—Here the talus presses against the lateral malleolus and the fibula breaks transversely 2 or 3 inches (5 to 7.5 cm.) above its lower end. If the talus subluxates laterally, the tip of the medial malleolus is avulsed (fig. 263 *b*).

(2) *Inversion Fractures* (*syn.* adduction fractures).—Here the talus inverts and tends to move medially. The medial malleolus is fractured vertically through its base and the external malleolus is fractured transversely at the level of the ankle joint (fig. 263 *c*).

A variant of the eversion injury includes rupture of the lower tibio-fibular ligaments with 'diastasis' of the ankle joint—i.e. widening of the mortise by separation of the malleoli, with or without fractures of the lateral malleolus or fibular shaft (fig. 263 *d*).

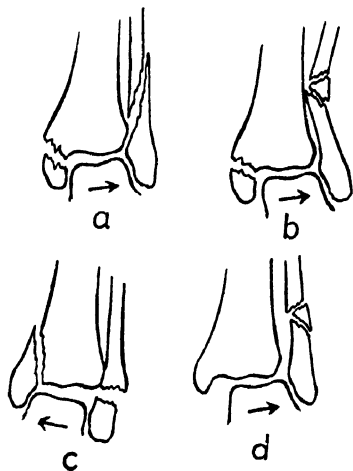


FIG. 263.—Types of ankle fracture.

- (a) Abduction—external rotation.
- (b) Eversion or abduction.
- (c) Inversion or adduction.
- (d) Diastasis of tibio-fibular joint.

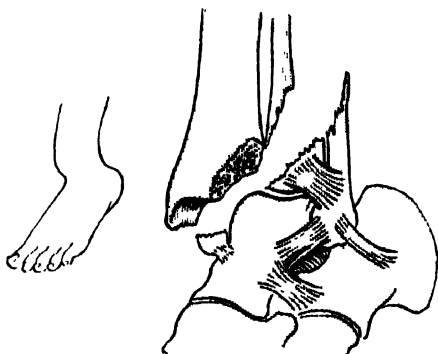


FIG. 264.—Gross mechanism of Pott's fracture. Foot displaced in relation to shaft of tibia.

Treatment.—The reduction of a Pott's fracture requires the complete relaxation of full anæsthesia. As judged from the pre-reduction X-rays of a severely displaced fracture, to the inexperienced it may appear unlikely that a satisfactory reduction could ever be obtained without operation, but the result of a skilful manipulation is often surprisingly good. The essential feature of the reduction is to concentrate on restoring the alignment of the *foot* to the *tibia*, rather than on entertaining the idea of reducing the separate fragments by local manipulation directly on the ankle joint. In the ordinary Pott's fracture with postero-lateral displacement of the talus, the separated fragments of the medial and lateral malleoli move as one piece with the foot and talus, because they are attached to these structures by their ligaments (fig. 264). The manipulation thus consists of reversing the direction of the forces which caused the original injury (a general principle in all fracture work). The common Pott's fracture is caused by external rotation and eversion ; therefore reduction is produced by internal rotation and inversion,

assisted in this case by correcting the backward displacement of the foot in relation to the tibia.

The reduced Pott's fracture is immobilised by encasing the ankle in a closely fitting plaster cast with the foot as near as possible in the plantigrade position. Weight-bearing is best avoided for six to eight weeks in badly displaced cases, and then only permitted after a new plaster has been applied. It is of paramount importance to check the maintenance of the reduction by weekly X-ray examinations during the first month after reduction.

There is a growing tendency to try to eliminate late re-displacement of a reduced Pott's fracture by operating on the medial malleolus. Once the medial malleolus has been fixed, the key to the reduction is held. This operation is certainly to be advised in fractures with gross displacement (fig. 265), but if a perfect reduction is obtained by manipulation it is possible to obtain an excellent result with careful conservative treatment.

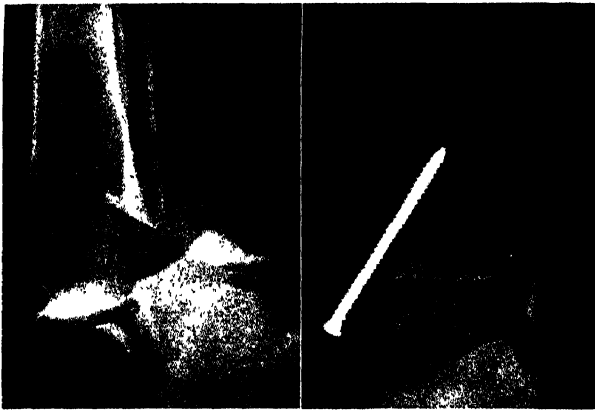


FIG. 265.—Internal fixation of medial malleolus.



FIG. 266.—Mal-union following a Pott's fracture. (*The late J. A. C. Macewen.*)

Old-standing cases of mal-union occasionally require osteotomy of the tibia and fibula, or even reconstruction of the fracture with more adequate reduction and possibly fixation by screw or peg (fig. 266). Old mal-united fractures are frequently best treated by arthrodesis of the ankle joint if the patient complains of pain due to traumatic osteo-arthritis.

The average disability after Pott's fracture is approximately light work in twelve weeks, heavy work six to nine months.

Fractures of the Tarsal Bones

Os Calcis.—The os calcis is almost always fractured by falls from heights by builders, steel erectors, and window-cleaners. In the last war many compound fractures of the os calcis were caused by the explosion of mines under transport vehicles. The os calcis is usually shattered like an eggshell and the injury thus involves the subastragaloid joint (fig. 267).

The degree of displacement varies according to the violence. In some cases there is little or no displacement; in others the whole os calcis is flat-

tened and widened. The tendo Achillis pulls up the loose fragment of the tuberosity and the heel is always everted to give a severe 'traumatic flat-foot'.

This injury is one which, despite all the different forms of treatment which have been tried, leaves the foot stiff in the subastragaloid region. This means that walking is difficult if the workman has to traverse an inclined plane, such as a sloping roof, because he cannot place the sole of the foot flat on the sloping surface. Similarly, walking out of doors on irregular ground is difficult, though walking on a flat pavement may be fairly comfortable. Frequently pain is experienced for many months, but this is



FIG. 267.—Fracture of the os calcis.

sometimes difficult to assess because heavy compensation claims usually cloud the picture. The fact remains, however, that though this is indeed a very severe injury to a working man, and will prevent him from carrying out his original employment if this involves working at dangerous heights, the end-results are sometimes not as bad as is usually suggested. Very few cases have incapacitating pain if examined three years or so after the injury, and to some extent they will have readjusted themselves to the stiff subtaloid

joint in their new occupation. For this reason the modern tendency is to abandon all the elaborate mechanical methods which in the past have been tried in an attempt to restore the normal anatomy. The best results are achieved by accepting the deformity and concentrating on early mobilisation. Weight-bearing should be encouraged in eight to twelve weeks.

The Astragalus is sometimes fractured as a result of a fall from a height. The neck of the bone may be shorn through by the sharp anterior articular surface of the tibia; comminution is common, and injuries to neighbouring bones are often associated. Frequently the body of the astragalus is extruded from the ankle joint by the force of the injury, leaving the head and neck *in situ*, and the displaced body may be palpable under the skin and defy all attempts at closed reduction. As in the case of the os calcis, considerable swelling rapidly develops and obscures the diagnosis, which is often only established after radiography.

Treatment.—Manipulative reduction, using skeletal traction, is sometimes successful, but usually operative reduction is necessary. If the body of the astragalus is deprived of blood supply, osteoarthritis of the ankle joint may later necessitate ankle fusion.

Fractures of the Metatarsals.—These are frequently caused by crushing injuries, such as weights falling on the foot or the wheels of vehicles running over them. In general, they need little or no attempt at reduction unless some part of the shaft, in a grossly displaced case, is projecting into the sole of the foot. If only one or two metatarsals are fractured, they are already splinted by adjacent bones.

Plaster is not required and early mobilisation with non-weight-bearing exercises is to be encouraged. Return to activity is by taking weight on the heel as soon as the patient feels he can, which may be in two to three weeks. Almost full function is possible in eight to twelve weeks, even after very severe injuries, in patients of good morale.

Avulsion of the Styloid Process of the Fifth Metatarsal.—

This is a minor injury which causes unnecessary inconvenience if treated in a walking plaster. The styloid process is avulsed by the pull of the peroneus brevis and, though the fragment is frequently detached by $\frac{1}{16}$ inch (1.5 mm.) or more, it always unites in the absence of treatment and in the presence of active movement. It is unnecessary to apply anything more incommoding than adhesive strapping.

March fractures (*syn.* *pied forcé*, *pied de jeune soldat*) occasionally occur near the necks of the second or third metatarsals. The fracture occurs spontaneously, and is predisposed to by a short first metatarsal. This common atavism causes undue strain to fall on the heads of the second and third metatarsals during such exertions as standing on the toes. The fracture is also encouraged by loss of muscular tone, which is predisposed to by wearing heavy boots. Sudden pain, localised over the dorsal aspect of the bone, is characteristic. An immediate X-ray will often fail to reveal the crack, but if repeated in three weeks callus will be obvious (fig. 268). Strapping and restricted activity only are required. A plaster shoe encourages further atrophy of muscles and renders rehabilitation necessary. Many cases are doubtless treated as 'foot strain' and more or less ignored, with good results!



FIG. 268. —
March fracture
of the third meta-
tarsal.

CHAPTER 12

DISEASES OF BONES

JOHN CHARNLEY

ACUTE INFLAMMATION

ACUTE PYOGENIC OSTEOMYELITIS

Predisposing causes of this condition are as follows :

(i) *Trauma*.—Before growth has finished, the weakest part of a long bone is at the diaphyseal side of the epiphyseal line. At this level (the metaphysis) loops of blood-vessels penetrate the epiphyseal cartilage, and any strain imposed on the bone may rupture one or more capillary loops with the formation of a hæmatoma.

(ii) *An Infective Focus*.—Such conditions as infected scratches, tonsillitis, or impetigo allow organisms to enter the blood-stream. A hæmatoma in any situation then forms an excellent culture medium, in which organisms rapidly multiply. Frequently the bone foci are multiple and mere incidents in a true septicæmia; these patients are, of course, much more ill than in the ordinary case which follows from a simple bacteræmia.

(iii) *Lowered General Resistance*.—In the past the disease was most frequent in industrial areas, where overcrowding and malnutrition were common.

Pathology.—The causative organism in the majority of cases is the *Staphylococcus aureus*, other organisms which are less frequently responsible being the *Streptococcus*, *Staphylococcus albus* and *pneumococcus*.

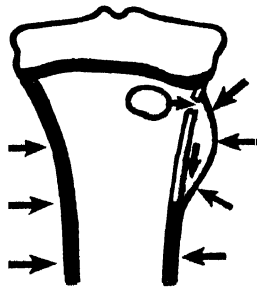


FIG. 269.—Spread of pus in osteomyelitis.

From its commencement as a small abscess on the metaphyseal side of the epiphyseal line, the pus immediately starts to extend to the surface of the bone, to appear under the periosteum; it does not spread from the metaphysis directly along the medullary cavity of the diaphysis as might be expected (fig. 269). The conditions associated with inflammation of bone are unique in that the vessels in the unyielding bony canals become compressed by exudate, and thus the circulation is impeded, with risk of necrosis of adjacent bone. This death of bone by lack of blood supply is rendered even more certain by the presence of the toxins in the pus. As periosteum is stripped up by a subperiosteal abscess, this underlying bone is separated from its source of blood supply and immediately poisoned. In an untreated case the whole diaphysis may be killed by the pressure of pus stripping the periosteum over its whole circumference. Owing to the

firm attachment of the periosteum to the epiphyseal cartilage and the resistance of the cartilage itself, subperiosteal pus is unlikely to invade the neighbouring joint, unless the epiphyseal line is intra-articular, as in the case of the head of the femur, lower end of femur, upper end of humerus, external malleolus, and olecranon process. Pus finally bursts through the periosteum, and tracks under muscles or finds its way to the surface. If the patient survives, the necrosed bone forms a *sequestrum*, the surrounding periosteum becoming extensively thickened with the formation of new bone to form an *involucrum* (fig. 270), which is perforated by *cloacæ* through which pus and spicules of dead bone escape from the cavity containing the sequestrum.

Clinical Features.—The symptoms usually start abruptly, the child complaining of severe pain near the end of a bone, the pain being aggravated by movement. Shivering or rigors may occur, and the general symptoms of a severe infection are present.

The severity of the general signs of infection depends on the virulence of the organism and the resistance of the patient. In septicæmic cases the child may be comatose as a result of profound toxæmia; more usually elevation of the temperature by 2° to 3° F. (1° to 2° C.) with associated increase in pulse-rate indicates a more moderate degree of infection.

The local signs depend to some extent on the depth of the affected bone. If the bone is well covered by muscle, as in the case of the lower end of the femur, vaguely localised tenderness above the level of the knee joint associated with some swelling is discovered in the early stages. In the case of a subcutaneous bone, such as the tibia, redness and œdema of the skin, in addition to exquisite local tenderness, will be present in the early stages. In both cases movements of the limb are painful, and likely to be strongly resented. After two or three days local thickening is palpable, and sympathetic effusion occurs into the neighbouring joint. Unless efficient treatment is adopted, the local signs of pus become increasingly obvious; a painful brawny area appears which gradually softens, and finally the abscess bursts through the skin, the resulting sinus leading down to the bone.

Radiography is of little value in the early stages, as bony changes are not usually visible until the end of the second or third week. Leucocytosis is to be expected, and in severe cases a blood culture is likely to be positive.

Differential Diagnosis.—*Acute Suppurative Arthritis.*—This is an intra-articular condition, and therefore the slightest movement of the joint is painful. In the 'sympathetic' effusions associated with acute osteomyelitis, a considerable range of painless movement can usually be obtained if the patient is given time, and the maximum tenderness is near the end of the bone rather than over the joint. If doubt exists, some of the fluid should be aspirated for examination.

Acute rheumatic arthritis is usually polyarticular. In the past this differential diagnosis was very difficult and the therapeutic response of acute rheumatic joints to aspirin and salicylates was advised. To-day the response to penicillin or wide-spec-



FIG. 270.—
Large sequestrum
and involucrum.
(Sheffield University
Museum.)

trum antibiotics is more useful than salicylates to establish the diagnosis of osteomyelitis.

Hæmarthrosis in children is mentioned on p. 76.

Scurvy.—Subperiosteal hæmatomata are sometimes very tender, and if near an epiphysis the condition may be confused with acute osteomyelitis.

Acute Exanthemata and Typhoid Fever.—These conditions may be suspected on account of the profoundly toxic and even comatose condition of the patient. Careful palpation of the ends of the long bones is necessary, and if pressure over a localised area induces resentful movements or moaning, then the possibility of osteomyelitis should be considered.

Complications.—Adequate and early treatment by penicillin, or wide-spectrum antibiotics, renders serious complications much less common than formerly.

General.—(i) *Toxæmia*.—Some degree of toxæmia is inevitable.

(ii) *Septicæmia* should be suspected if shivering, rigors, or an intermittent temperature are present. Infection of the serous membranes is likely to occur, particularly of the pericardium.

(iii) *Pyæmia*.—In the past this condition was usually fatal. Infected emboli are carried to the lungs and plum-coloured, wedge-shaped infarcts occur, with a small quantity of blood-stained fibrinous fluid in the pleural cavity. Increased respiration, cyanosis, and patches of bronchial breathing are indicative of this complication.

Chronic pyæmia can occur and give rise to abscesses in any part of the body. These abscesses reveal themselves at any time from the first few days of the disease until after the lapse of some months, and often appear like 'cold' abscesses with little or no local discomfort. When detected these abscesses may contain a pint or more of pus.

(iv) *Later complications* are (a) chronic osteomyelitis and (b) amyloid disease from chronic suppuration (p. 35).



FIG. 271.—Manus valga, following osteomyelitis of the radius, and consequent diminution of its growth. A variety of Madelung's deformity.

Local (i) *Joint*.—Acute suppurative arthritis may complicate acute osteomyelitis at sites where the epiphyseal line may be wholly or partly intra-articular.

(ii) *Spontaneous fracture* may occur, especially when a single weight-bearing bone such as the femur is extensively destroyed by an acute osteomyelitis without new bone formation, though in chronic osteomyelitis with sclerosis of bone it is most unlikely.

(iii) *Deformity* sometimes follows from interference with the epiphyseal line, but it is a remarkable thing how very uncommon this is. Growth at the epiphysis directly affected may be diminished (fig. 271), while growth in another at a distance, owing to hyperæmia, may be increased.

(iv) *Brodie's Abscess*.—This is a chronic bone abscess which causes intermittent pain near the end of a long bone, with perhaps transitory effusion into the adjacent joint during an exacerbation. Examination may reveal thickening of the bone, and a radiograph is diagnostic. The amount of sclerosis is variable, ranging from dense sclerosis extending a considerable distance

round the cavity (fig. 272) to, more commonly, a faint line of sclerosis at the junction of the abscess with the cancellous bone. If sclerosis is absent, the cavity may resemble that of a tuberculous abscess. These chronic staphylococcal abscesses were first described by Brodie in connection with the head of the tibia. The abscess may be the sequel to a pyogenic septicæmia from which the patient has recovered some years previously and which has remained dormant in the interval. On the other hand, it may be found years later in a patient who is known to have had osteomyelitis affecting some bone other than the one in which the Brodie's abscess is discovered. Free exposure, curetting, and 'saucerisation' are necessary. Often the cavity is found to contain jelly-like granulation tissue rather than actual pus.



FIG. 272.—Brodie's abscess of lower end of the tibia.

It must be emphasised that nowadays all these complications are, with perhaps the exception of Brodie's abscess, rarely encountered since the use of antibiotics which have completely changed the problem of acute osteomyelitis.

Treatment.—*Penicillin* or *tetracycline* is administered immediately the diagnosis is a probability, but not before a specimen of blood has been taken for culture. In cases treated within two or three days of onset complete resolution is to be expected within a further three or four days, before radiological evidence of disease is present, if the organism is sensitive to the antibiotic. Full doses of antibiotic should be administered daily for a period of three weeks. The patient should lose all signs of spontaneous pain within twenty-four hours if the organism is responding to the antibiotic. Persistent local tenderness indicates the possibility of abscess formation. It is important to immobilise in a splint which permits access for the testing of tenderness. An abscess will maintain the patient's temperature if it is not evacuated, and may erroneously suggest that the organism is not susceptible to the antibiotic.

Operation is indicated in order to evacuate a subperiosteal abscess, or to decompress the metaphyseal region by one or two drill holes on the metaphyseal side of the epiphyseal line. If a subperiosteal abscess is present as indicated by clinical signs, and especially if the toxæmia is not responding to antibiotics, it is unwise to delay surgery lest unnecessary necrosis of bone is produced and the duration of the illness thereby protracted.

The rôle of early surgery in acute osteomyelitis is still frequently disputed. It is the writer's opinion that no harm is ever done by early decompression and that most errors are due to delaying decompression when the child is not showing both general and local improvement on the antibiotic.

Aspiration is inadequate. A small incision should be made so that all the pus can be evacuated. The wound is then dusted with penicillin and sutured. If no pus is encountered under the periosteum at the site where local tenderness was maximal, it is all the more essential to drill the bone.

The fear of infecting underlying bone by drilling, if a subperiosteal abscess is found, is quite unfounded because this pus must have come from the marrow in the metaphyseal region.

It is to be noted that under antibiotic treatment the significance of the X-ray as an indicator of sequestration has slightly to be modified. It is not uncommon to see the X-ray appearances apparently getting worse between the third and fourth weeks in a child who has responded well to penicillin, and who throughout this time has



FIG. 273.—Drilling of the tibia in a case of acute osteomyelitis. An Esmarch's tourniquet has been applied to control hæmorrhage.

been afebrile, eating well, and without local evidence of inflammation. This merely means that the original local toxæmia killed a certain amount of bone which the penicillin then sterilised, and with the passage of time the process of demarcation of the living bone from the dead goes on as a late aseptic process only when the patient has passed the dangerous phase. In these cases the 'sequestrum' will be reincorporated if there is no discharging sinus.

ACUTE TRAUMATIC OSTEOMYELITIS

This condition arises as a result of infected wounds, e.g. compound fractures, operations on bones, following amputations, etc. The constitutional disturbances are less severe than in cases of infective osteomyelitis, as the causative wound provides some measure of drainage. More extensive opening of the wound, removal of dead bone, or even amputation may be necessary.

CHRONIC INFLAMMATION

CHRONIC PYOGENIC OSTEOMYELITIS

Acute hæmatogenous osteomyelitis may pass into chronic osteomyelitis if early treatment is not available, or is inadequate. The risk of this condition developing is now greatly minimised by the modern treatment of the acute infection, but many cases still remain as a legacy of the pre-penicillin era, and more will probably occur if staphylococci become penicillin resistant.

Chronic osteomyelitis may remain quiescent for months or years, but from time to time acute or subacute exacerbations recur, especially if the patient's resistance is undermined by worry, overwork, or other debilitating conditions. Brodie's abscess (p. 232) is a type of chronic osteomyelitis which is specially liable to periodic exacerbations.

An exacerbation is ushered in with constitutional disturbances and local evidence of inflammation, which may culminate in discharge of pus from a pre-existing sinus. An X-ray sometimes reveals a sequestrum which has separated from the surface of the bone or which lies in a cavity (fig. 274).

The chronicity of chronic osteomyelitis is the result of the physical characteristics of bone, in that the abscess cavity can never close by shrinking of the walls as happens in the soft parts. Moreover, the effect of inflammation is to kill the hard, bony walls of the abscess, thereby causing sclerosis, and so preventing the antibacterial action of the blood getting to

the contents of the cavity. The contents of the cavity thus continue to decompose very much in the same way as would animal matter in a warm, moist test-tube.

Treatment consists in immobilisation of the limb and the administration of antibiotics, under which regimen many cases subside for a variable period. Surgical intervention has to be considered if an X-ray indicates the presence of a sequestrum or cavitation. If a sinus is present, a sequestrum may be detected with a probe which grates on the loose fragment of dead bone. Penicillin is administered for some days prior to the operation, and access to the bone is usually gained through a previous scar. The soft tissues are stripped from the bone with a raspatory, and the involucrum is removed as necessary in order to gain access to the sequestrum. If a cavity is present, the overhanging walls are removed with an osteotome, until it is efficiently 'saucerised'. The wound is dusted with penicillin powder and closed.

It is to be noted that so difficult is it to guarantee that an operation will clear up a sinus of many years' duration, that operative intervention is not to be considered lightly unless a sequestrum is known to be present. If a sequestrum is present and is removed, the sinus will almost certainly close. Clinically a sequestrum is often suggested by the presence of pouting granulation tissue at the mouth of the sinus and this sign is an indication for surgery.

If only a cavity is present in the bone without a sequestrum, the attempt to 'saucerise' may fail and still leave a small sinus. There are many cases where, if the discharge is slight and easily controlled by a dressing, it is preferable to retain the sinus and dressing permanently. Amyloid disease need be feared only when a copious discharge of pus has persisted for some years.

Amputation is advisable if exacerbations are frequent or prolonged in order to rid the patient of recurring periods of painful disability, and to forestall the onset of amyloid disease.

SYPHILITIC DISEASES

Syphilitic diseases of bone are now so rare that it is possible the contemporary student in this country will never see an actual case.

Congenital.—(i) *Osteochondritis* of the nasal septum is the first manifestation. Necrosis of cartilage occurs at the age of about four weeks, and the resulting discharge causes 'snuffles'. Characteristic depression of the bridge of the nose follows destruction of its support (fig. 275).

(ii) *Craniotabes* of the vault of the skull occurs during the first six months as a result of absorption of bone. This condition can also be due to concomitant rickets (p. 242).

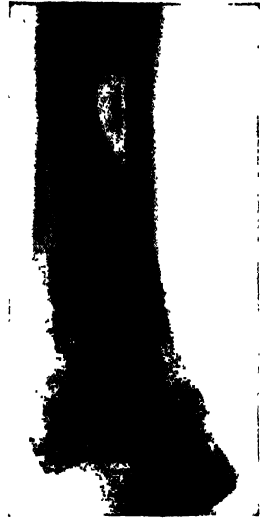


FIG. 274. — Chronic osteomyelitis of the femur with a cavity containing a sequestrum.



FIG. 275. — Frontal bosses and depressed nose of congenital syphilis.

(iii) *Parrot's nodes* may appear on the skull during the early years. They are patches of periostitis, and if the parietal and the frontal bones are also involved, so that there are four bosses, the term 'hot-cross bun' is applied.

(iv) *Epiphysitis* occurs towards the end of the first year, the epiphyseal line being broader than usual and yellowish in colour. Separation may occur, resulting in so-called 'pseudoparalysis'. Periostitis extends from the epiphysis along the shaft of the bone and forms a fusiform swelling, unlike the abrupt expansion of rickets.

(v) *Overgrowth and curvature of the tibia* usually appear towards puberty. The curve is only in an antero-posterior plane, and affects the whole bone.

(vi) *Teeth*.—Hutchinson's notched and peg-shaped teeth affect the permanent incisors (fig. 276), and Moon's turret-teeth, so called from the absence of the central cusp, may occur in the permanent molars.



FIG. 276.—Hutchinson's teeth, characteristic of congenital syphilis.

Acquired.—SECONDARY stage: bone pains are due to localised patches of periostitis. Permanent periosteal nodes may persist.

TERTIARY.—(i) *Periosteal Gumma*.—Single gumma arising in the periosteum characteristically occurs in the tibia, clavicle, and manubrium, although other bones may be affected. A firm, slightly tender swelling appears, which is obviously connected with the underlying bone. As the swelling enlarges, the superficial structures are progressively involved and the skin becomes reddened over the indurated tissues. Eventually the skin softens and sinuses form which allow the

escape of necrotic material. A punched-out or seriginous ulcer results, the floor of which is temporarily covered by a wash-leather slough. Secondary infection is probable, and necrosis of bone then follows.

The nasal septum and hard palate are not uncommonly affected, and extensive necrosis of bone occurs from secondary infection.

Multiple periosteal gummas occur on the skull (figs. 277, 278). The condition is



FIG. 277.—Multiple gummata of the skull. (A. J. King, F.R.C.S., London.)



FIG. 278.—'Worm-eaten' skull. (R.C.S. Museum.)

now uncommon and the 'worm-eaten' skulls of previous sufferers are museum exhibits.

(ii) *Endosteal Gumma*.—This is now uncommon, but when it occurs errors of diagnosis are likely, and a limb may be sacrificed unnecessarily under the impression that the bony enlargement is malignant, e.g. osteosarcoma. Perhaps in no other situation does syphilis more justly deserve its title of 'the great imitator'.

The chief symptom of syphilitic osteomyelitis is an aching pain in the bone, boring

character, particularly when the limb is dependent, or at night when covered by warm bedclothes. Palpation reveals thickening of the bone, due to associated periostitis. X-ray shows marked sclerosis and the formation of new periosteal bone. Biopsy should be undertaken in doubtful cases.

Even vigorous antisyphilitic treatment may fail to relieve the constant pain associated with an endosteal gumma, owing to the protection afforded by the surrounding zone of thickened bone, which prevents remedial substances in the blood from reaching the affected area. 'Guttering' the bone is sometimes necessary so as to open up the medulla and allow an adequate blood supply to the interior of the bone.

(iii) *Diffuse Sclerosis*.—This consists of thickening of all or any of the periosteal, cancellous, or medullary elements of a bone. The skull and shafts of the long bones are affected, and in long-standing cases the medullary cavity is obliterated.

TUBERCULOUS DISEASE

Tuberculous disease of bones and joints has now become a rare condition in the British Isles but it is of such importance in differential diagnosis that the student should be familiar with its manifestations.

Tuberculous disease of bone is always secondary to a primary focus in the respiratory tract or bowel. The disease commences either in the interior of the bone or in the periosteum and the organisms reach these tissues from the primary lesion via the blood-stream.

(a) **Tuberculous osteitis** starts by blood-borne bacilli lodging in cancellous bone. It thus most commonly occurs at the ends of long bones in relation to joints. Very soon a joint is involved and the clinical signs of tuberculous arthritis dominate the picture. The commonest example of tuberculous osteitis, which remains as an osteitis without becoming an arthritis, is Pott's disease of the spine. A rare but well-known example of tuberculous osteitis is tuberculous dactylitis affecting the phalanges of the fingers in children, but it is a condition which nowadays has practically disappeared (fig. 279).

Tuberculous osteitis is insidious in onset, and for some weeks or months the patient may be conscious only of slight weakness or aching, particularly after use. On examination, some puffiness may be noticed, and palpation reveals slight thickening of the periosteum, due to oedema. At a later stage the skin becomes shiny, the bone is thickened and tender, and muscular wasting is evident. Finally, a subperiosteal abscess forms, which erodes the periosteum and finds its way to the surface, the last stage being represented by sinuses which lead down to the bone and allow the entry of secondary infection.

Bone destruction ('caries') is the characteristic feature of tuberculosis and this is seen in the X-ray as a general rarefaction of the bone with blurring of the bone detail and erosion of the shell of cortical bone underlying the articular cartilage. The formation of sequestra is not uncommon, but these are small and not as dense as those in pyogenic bone infections. New bone

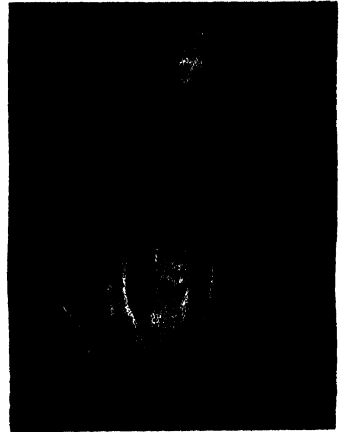


FIG. 279.—Tuberculous dactylitis of third metacarpal and phalanges of fifth finger.

formation and bone sclerosis are not characteristic of a tuberculous lesion, but are seen when secondary infection is present.

(b) **Periosteal tuberculosis** most commonly affects the flat bones, e.g. the sternum, or skull, and especially the ribs. Infection commences in the deeper layers of the periosteum, which becomes œdematous, and is soon separated from the underlying bone by granulation tissue. Caseation and cold abscess formation follow, the superficial structures becoming progressively adherent and invaded, while the bone itself is eroded. In the case of a rib the abscess may extend along the bone to discharge some distance from the site of origin. Finally, the skin is involved and the abscess discharges on the surface, and secondary infection follows. X-rays show erosion of the bone if the condition has advanced sufficiently.

Treatment.—In the treatment of local tuberculous lesions in a bone or joint, the surgeon must never forget that the patient is a tuberculous subject and that the systemic approach to the disease is essential (p. 23).

It is useless to excise a diseased bone or splint a tuberculous joint if the patient is also suffering from some other secondary complication, such as renal tuberculosis.

Only when the systemic element seems to be under control should local surgery be considered. Often spontaneous healing will take place following the improvement in the general condition of the patient. When abscesses are present it is now a common practice to evacuate them and to curette the underlying diseased bone. With antibiotic cover surgery is now employed much earlier than was the case twenty years ago and spontaneous discharge with sinus formation and secondary infection is avoided. Sometimes amputation is inevitable, but much less so since the advent of chemotherapy.

Healing of the local lesion is indicated clinically by diminution of swelling and local heat, and radiologically by the return of bone detail and normal bone density.

POTT'S DISEASE (TUBERCULOUS SPONDYLITIS)

Pott's disease of the spine is the commonest form in which tuberculous disease affects the skeleton. It can occur at any age, and though formerly it affected children more commonly than adults, there has been a steady decline in the incidence among children. This is due to more stringent Public Health measures and improved nutrition. In the past the milk supply¹ was held responsible for most bone and joint tuberculosis, but the organism to-day is of human origin.

The most common site of Pott's disease is in the thoraco-lumbar region; lumbo-sacral and high thoracic diseases are next in frequency with the cervical region as a relatively rare site. The disease starts as an osteitis in the cancellous bone of a vertebral body adjacent to an intervertebral disc. It is characteristic of the progress of the disease that the intervertebral disc is destroyed at a very early stage and there may be radiological narrowing of the

¹ Tuberculosis in cows has now been eradicated in all herds in England and Wales.

Percival Pott, 1714–1788. Surgeon, to St. Bartholomew's Hospital, London.

disc space before loss of detail of the adjacent bone surface can be established in the X-ray with certainty. Further destruction of vertebral bodies occurs by extension of pus as a 'perispinal' abscess under the anterior common ligament and at the sides of the vertebral bodies (fig. 280). Eventually col-

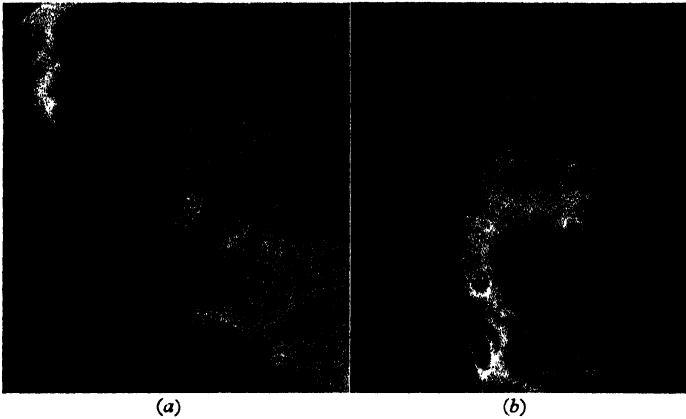


FIG. 280.—Pott's disease: (a) Collapse and fusion of two vertebral bodies with single wedge. (b) Perispinal abscess shadow.

lapse of the bodies occurs and a kyphosis will be produced and the degree of angularity depends on the number of vertebræ destroyed (p. 326). In Pott's disease collapse of vertebral bodies anteriorly always causes a kyphosis and, for all practical purposes, never a scoliosis (figs. 281 and 282).



FIG. 281.—Pott's disease, with gross angular curvature. (London Hospital Museum.)



FIG. 282. — Kyphosis due to Pott's disease.

The perispinal abscess tracks along muscle planes, under the influence of gravity, and often becomes subcutaneous as a cold abscess at a site remote from the source of the disease. Thus twelfth thoracic or upper lumbar disease may track as a psoas abscess, to appear above or below Poupart's ligament in the

François Poupart, 1661-1708. Surgeon, Hôtel-Dieu, Paris.

groin where it may be mistaken for a femoral or, less commonly, an inguinal hernia (fig. 283) and (Chap. 43). If neglected, the natural sequel is for the abscess to discharge and for a sinus with secondary infection to result.



FIG. 283.—A psoas abscess pointing in the right groin.

In Pott's disease in the thoracic region (i.e. above the level of the cauda equina and at the level of the spinal cord), the pressure of a tense perispinal abscess communicating with pus inside the spinal canal can cause compression of the cord with paraplegia (Pott's paraplegia).

Clinical Features.—These vary to some extent with the level of the disease, but the following general remarks apply to all levels.

Symptoms.—It is characteristic of Pott's disease that the onset and initial stages of progress are so insidious that they can pass unnoticed until sometimes the dramatic symptoms of paraplegia, the development of a kyphus, or the appearance of a large cold abscess may be the first evidence of serious trouble. In adults vague symptoms of backache are sometimes dismissed for six to twelve months as 'rheumatism' or 'fibrositis', and then X-ray examination shows extensive destruction of vertebral bodies and perhaps a perispinal abscess.

There will usually be the general systemic signs of lassitude, loss of weight, night sweats, and evening pyrexia.

Signs.—The most important sign, for the early clinical diagnosis of these cases, is rigidity of the spine. A moderate degree of limitation of forward flexion in a patient complaining for the first time in his life of indefinite mild backache is much more sinister than severe limitation of flexion in association with intense back pain in a patient who has had similar acute attacks in the past. The former may well be Pott's disease, whereas the latter is more likely to be an acute disc lumbago.

Palpation of the abdomen and groins should be carried out to exclude the presence of abscesses.

Palpation of the spine may reveal an early 'knuckle' kyphosis.

Examination of the knee-jerks and plantar responses, in dorsal disease, may reveal evidence of early pyramidal tract involvement, often before the patient complains of difficulty in walking.

Radiology.—Radiological examination of the spine will usually establish the diagnosis immediately. In the lateral view narrowing of an intervertebral disc space associated with a little erosion of an adjacent vertebral body is the earliest definite sign. In the antero-posterior view the presence of a perispinal abscess will confirm the diagnosis. Destruction of a vertebral body, without narrowing of a disc space, and particularly if without a perispinal abscess, is more likely to be a secondary deposit than Pott's disease.

In old-standing disease the perispinal abscess may be calcified (fig. 284).

Treatment.—As in the management of a tuberculous joint there are three

phases of treatment to be distinguished:

Phase I.—Recumbency and general systemic measures (fresh air, diet, and antibiotic drugs). During this time the patient is immobilised on a spinal frame or in a plaster bed. In thoracic lesions below the mid-thorax the head can be left free, but in cervical and upper thoracic lesions the head will need restraint.

Abscesses are aspirated, and re-aspirated at intervals if they tend to refill.

In this way it may take one or two years to reach the stage of quiescence, as shown radiologically by the fact that further destruction has been absent for six months and perhaps that some tendency to recalcify may be detected. Quiescence cannot have been reached if palpable abscesses are still present.

Phase II.—Ambulation in apparatus. For most levels a posterior spinal support is sufficient to brace back the shoulders and counteract a forward stooping position. At higher levels a blocked leather collar may be needed. Under the protection of such appliances many cases will pass on to natural spontaneous healing, which (unlike tuberculous disease in large joints) is a true bony fusion between the vertebral bodies. This often may take as long as five years to become complete. When this has happened, all external support can be discarded.

Phase III.—Arthrodesis (spinal fusion). If spontaneous fusion of the vertebral bodies has taken place or seems likely to be taking place, operative

fusion is unnecessary. In cases where no evidence of spontaneous fusion can be seen a posterior spinal fusion is frequently employed so that the patient can eventually discard all external support.

Technique.—Bone grafts are laid on the roughened dorsal surfaces of the spinal laminae and on each side of the roughened spinous processes. The grafts may be taken from the tibia with an electric saw or may be chips of cancellous bone from the iliac crest. The erector spinæ muscle is closed over the graft and immobilisation for

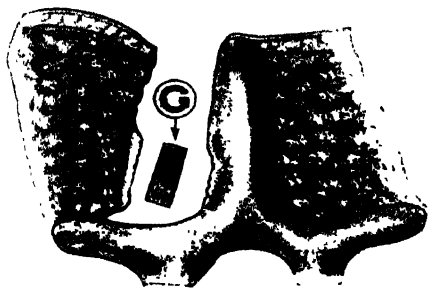


FIG. 285.—G = graft, inserted between spinous process and muscles.

three months will usually be enough for the graft to become incorporated (fig. 285). Protection for another three to six months in an ambulatory appliance is then needed before finally discarding all support.

Early Surgery.—The three phases described above are those inherited from the pre-antibiotic era. There is more and more a tendency to operate after two or three weeks on antibiotic therapy, and to evacuate abscesses, clear out dead bone, and even to implant bone grafts directly into the diseased site. Early surgery is only possible by working through the abscess cavities and this in turn is only possible because of antibiotics. This revolutionary approach has enormously shortened the time of hospital treatment, but it must not be forgotten that the patient is still a tuberculous subject.

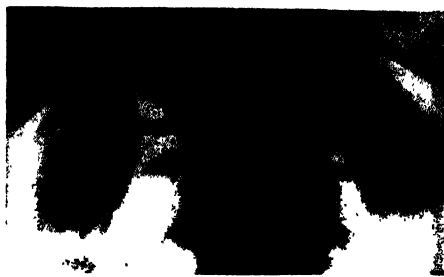


FIG. 284.—Old-standing tuberculous disease of the second lumbar vertebra, with calcified psoas abscesses.

Pott's Paraplegia.—The onset of spastic paresis in Pott's disease is a sign of the utmost gravity, and there are still features of the ætiology which as yet are not sufficiently well understood for a dogmatic statement on the best form of treatment. There are so many possible elements causing the paraplegia that probably no single explanation fits all cases. Thus 'early onset' paraplegia, occurring *pari passu* with the earliest stages of the disease, does not have the same mechanical element which is generally present in the 'late onset' cases with severe kyphus, where paraplegia supervenes years after the start of the disease. Even cases of 'late onset' paraplegia, with acute angulation inviting the idea of pressure on a backward displaced bony projection, can still be due to recrudescence of active disease. In active disease it would be a great step forward if we could distinguish whether the paraplegia is due to mechanical compression by an extradural abscess or by a sequestrum, or whether it is due to inflammatory involvement at the level of active disease, and even, in some cases, to vascular thrombosis in the cord. Obviously mechanical decompression of the cord would help the first but not the last, and if mechanical causes are present it is important to remove them without undue delay.

Historically, it may be said that approximately 50 per cent. of Pott's paraplegias recovered spontaneously on ordinary sanatorium treatment (without antibiotics) and 50 per cent. got worse and eventually died. In those cases where the osseous lesion healed the paraplegia recovered, but where the osseous lesion became a 'chronic grumbling disease' the paraplegia persisted. In an attempt to decompress the cord, the next phase in evolution was a laminectomy designed to evacuate pus from the spinal canal and leave room for dorsal expansion to reduce pressure. The results were so uniformly bad that the method was soon abandoned, and the only surgical procedure advocated was that of 'costo-transversectomy' in which the perispinal abscess was drained by removing the head and neck of a rib. In many cases, especially when a spherical perispinal abscess is visible in the radiograph which suggests internal tension, decompression of the perispinal abscess can decompress the associated intraspinal abscess, and this procedure is still used with considerable success.

In recent years there has again been a revival of interest in the direct surgical decompression of the spinal cord, but more emphasis is now laid on the removal of sequestra or protruded disc material lying directly *in front* of the cord (antero-lateral decompression). Remarkable recoveries from paraplegia have followed this procedure, but it is still too early to make a final pronouncement on the indications for this severe operation.

Undoubtedly, the degree of paralysis at the time of receiving the patient in hospital must have an important bearing on recovery—either by conservative or operative measures. Patients in flaccid paralysis or with extreme flexion contractions, with complete urinary retention, or when vibration sense in the legs is absent, are much less likely to make a recovery, by any method, than others less profoundly affected.

DEFICIENCY DISEASES

RICKETS

Rickets is a disease not exclusively affecting bones, but involving the body as a whole. The characteristic bony changes may be considered with advantage in this chapter.

Rickets is a deficiency disease, the essential cause being lack of vitamin D, which is

component of natural fats and oils. In addition, lack of sunshine and insufficient ingestion of calcium and phosphorus are contributory factors.

Though at one time extremely common in England (called, indeed, the 'English disease' by the French) simple rickets is now one of the rarest. The diseases in which calcium metabolism is disturbed by other causes (see later), at one time considered rare, are probably now the commoner type.

The pathological changes which occur mainly affect the epiphyses, the cartilages of which are enlarged both longitudinally and laterally. The most characteristic radiological abnormality in active rickets is the 'cupping' of the epiphyseal line. The epiphyseal line is blurred and concave towards the epiphysis (fig. 286). Histologically the zone of provisional calcification is either absent or represented by irregular patches, and therefore no definite line of demarcation exists between the proliferating cartilage and the medullary spaces. Instead of being composed of bone, the new-formed trabeculae are of osteoid tissue, which contains no calcium salts, while the medullary spaces are filled with vascular fibrocellular tissue instead of normal bone marrow. The deformities associated with rickets are due to lack of rigidity of this osteoid tissue. As the disease is overcome, calcium salts are deposited, and the deformed bones become normal in texture, or in some cases even denser than normal.

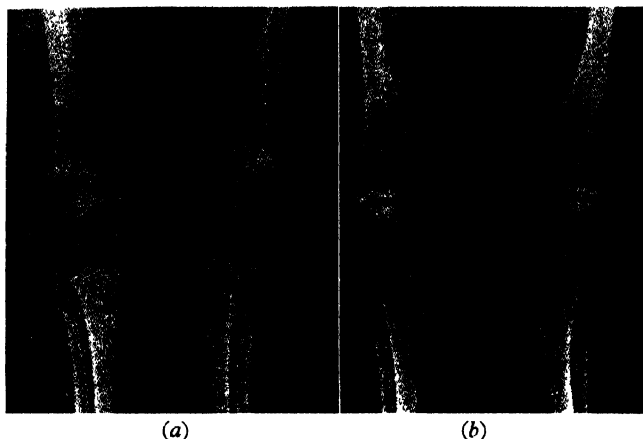


FIG. 286.—Rickets: (a) Florid state; (b) healing state.



FIG. 287.—Rickets, showing the deformed legs and pot-belly.

Deformities are due to the weight of the body, the influence of posture, or to the constant pull of muscles bending the softened bones.

Clinical Features.—The child is usually flabby, and sweating of the head is common. It is particularly susceptible to respiratory and gastro-intestinal disturbances, and the abdomen becomes protuberant owing to enlargement of the liver and spleen. Umbilical hernia is common as a result of prolonged distension.

Bony developments cause restlessness and peevishness, and the child, on account of epiphyseal tenderness, resents being handled. As the disease progresses deformities appear, among which the following are typical:

Epiphyseal Enlargement.—The increased width of the epiphyses can usually be felt (fig. 287). These swellings disappear as the disease fades, and the formation of periosteal bone causes increased width of the shaft.

Ribs.—The 'rickety rosary', due to beading of the costo-chondral junctions, is of the same nature as epiphyseal enlargements. The swelling is more pronounced on the posterior surface. Harrison's sulcus is due to abdominal distension 'spreading' the lower ribs, and not to the inward pull of the diaphragm.

Long Bones.—Bending of the long bones is more marked in the legs because of weight bearing. Natural curves are exaggerated and thus the femur shows increase of the normal anterior curve. Bending of the femoral neck produces coxa vara.

The tibia is characteristically bent in two planes. An abrupt kink occurs in the lower third of the bone, the portion below this being bent backwards and inwards

(fig. 288). A well-marked buttress formation occurs in the concavity, and the anterior border of the bone is sharp, owing to lateral compression of the shaft.

The Skull.—Craniotabes occurs as a result of any severe constitutional disturbance, and if associated with rickets is most obvious in the region of the lambdoid suture. Closure of the fontanelles and dentition are delayed. As the skull develops it becomes broader and flatter than normal, and the increased width between the eyes indicates broadening of the base.

The Spine.—The child is tardy in its efforts to sit up. Kyphosis constitutes the first spinal deformity, which may be followed by scoliosis due to posture or to inequality of the legs.

The Pelvis.—Two types of deformity may result. In the flattened type the conjugate diameter is diminished, while if the lateral walls are approximated, a tri-radiate deformity results.

Stature.—Diminution in stature is due to the following reasons :

(i) The actual growth of bone is retarded, especially that of the tibia and femur, which may be as much as one-quarter shorter than those of a normal child of equal size and age.

(ii) Bending of the bones of the legs, and by spinal deformities.

Treatment.—Early recognition and appropriate treatment are rewarded by ready response. Fresh milk, cod-liver oil, and meat extracts are administered as freely as the child will digest them. Judicious exposure to natural or artificial sunlight or ultra-violet rays is beneficial, as by this means ergosterol, normally present in the skin, is converted into vitamin D.

Early bony deformities respond to appropriate splinting.

Osteoclasis.—In the case of the tibia, manual osteoclasis may be necessary, and should be performed during the third or fourth year. Osteoclasis is performed by resting the leg on a rubber-covered wedge, the leg lying on its outer side. Pressure is applied so that the fibula and then the tibia snap opposite the site of maximum deformity. Care must be taken to grasp the lower end of the bone as close to the deformity as possible, as cases have occurred where the lower epiphysis has been separated. A plaster of Paris casing is applied for three weeks.

In older children or adults osteotomies are sometimes necessary.

Renal Rickets

is a rare condition, due to renal insufficiency during childhood, as a result of chronic interstitial nephritis, or, more rarely, polycystic kidneys. Thirst and polyuria, followed by headache and vomiting, are the symptoms which should suggest renal disease, but cardiovascular changes are absent. The blood urea content may be as high as 300 mg. or more per cent.

Bony deformities appear at any age, and in the early years separation of epiphyses may occur. After the first decade the changes somewhat resemble rickets, and deformity follows.

Death from uræmia is seldom delayed beyond puberty, and is hastened by any operative intervention, such as osteotomy.




FIG. 288. — Rachitic tibia, showing abrupt curvature in the lower third of the bone, and well-marked buttress formation.

Resistant Rickets

This is a condition in which rachitic changes are present in spite of a normal diet and normal intake of vitamin D. It will heal when very heavy doses of vitamin D are given for long periods. The nature of the resistance is unknown. The age of onset is usually about five years and thus considerably later than the appearance of ordinary rickets.

Fanconi's Syndrome

In this condition the calcium and phosphorus metabolism is disturbed by a renal abnormality in which glycosuria and albuminuria are often associated with cystinuria.

There are rachitic changes in the epiphyses if the condition occurs in early life, or in adult life osteoporosis with deformity and fractures. There may be considerable nephrolithiasis due to excretion of calcium through the kidneys.

Late Rickets and Osteomalacia

Later in life the dietary deficiencies which are responsible for rickets, particularly when associated with unfavourable hygienic and social conditions, may be associated with important bone changes which are called late rickets, or osteomalacia, according to the age at which they occur. The lesions of osteomalacia may be regarded as those of rickets in the absence of growth.

Late rickets is a rare disease which occurs during puberty or adolescence. In some cases careful enquiry and examination suggest that this is due to a recrudescence or relapse of the ordinary type of this disease. In a typical case of late rickets the head is not affected, and bending of the bones occurs close to the epiphyses. Severe pain occurs in the bones, which are tender on palpation, and gross deformity occurs in advanced cases. As with infantile rickets, ingestion of substances containing fat-soluble vitamin D results in rapid improvement.

Osteomalacia is rare in this country, although in some localities, e.g. the Himalayas and North China, it is by no means uncommon. Nine-tenths of cases occur in females, mostly during the child-bearing age. The condition usually appears during pregnancy, but the actual cause is an insufficiency of vitamin D and calcium salts. Tetany may occur in advanced cases.

The changes in the bone consist of decalcification of the osseous framework and metaplasia of the resulting matrix and medulla to fibro-cellular tissue. The compact bone may become as thin as paper, and the marrow represented by fatty fibro-cellular tissue of a vascular nature, which has been likened to liver or splenic pulp. Calcium and phosphorus contents of the blood are normal.

The main symptom is pain in the bones, which is deep-seated and aggravated by movements or pressure. Lassitude and asthenia follow, and gross deformities (fig. 289) and fractures become increasingly in evidence, especially with repeated pregnancies.

Treatment demands a food rich in calcium and substances containing fat-soluble vitamin D. Cæsarian section is sometimes necessary for subsequent pregnancies.

CELIAC RICKETS (*syn.* GEE'S DISEASE)

This disease begins in early childhood and is characterised by the passage of offensive fatty stools and changes in the bones similar to osteomalacia. The patient is usually ill-developed, anæmic, and in severe cases tetany may supervene. It is probably due to some gastro-intestinal disturbance which results in deficient utilisation or absorption of some essential factor. In most cases the serum calcium is below normal and the plasma phosphatase is increased.

Rapid improvement follows if a diet is given which is rich in calcium and low in fats, together with some preparation of vitamin D. Anæmia is combated with iron, and deformities require splinting pending regeneration of bones.



FIG. 289.—Pelvic deformity due to osteomalacia.

SCURVY

Scurvy is occasionally seen in children who are weaned at an early age. Most prepared foods are deficient in vitamins, and the anti-scorbutic vitamin (vitamin C) appears to be particularly susceptible to heat. The disease appears in the sixth month and, in addition, rickety changes of a variable degree may be superadded. Scurvy is characterised by subcutaneous or submucous hæmorrhage, subperiosteal extravasations which are markedly tender (they have been mistaken for acute osteomyelitis), and in more advanced cases by sponginess of the gums and even hæmaturia. If neglected, the disease may progress to a fatal issue, preceded by separation of epiphyses, melæna, and emaciation.

Treatment consists in the administration of such anti-scorbutic remedies as fresh fruit juice, uncooked meat juice, cabbage water, mashed potato, and cream.

Scurvy in children is nowadays rarely encountered, and is now most frequently seen in elderly patients on defective diet.

GENERAL DISEASES OF BONE

OSTEITIS DEFORMANS (syn. PAGET'S DISEASE OF BONES)

This condition, first described by Sir James Paget in 1877, occurs rather more commonly in men than in women. The generalised disease as Paget described it is uncommon, but in more recent years the detailed autopsy investigations of Schmorl show that localised forms of the disease occur in 3 per cent. of persons over forty years of age.

Ætiology and Pathology.—The ætiology of Paget's disease is completely unknown. Pathologically it appears to be one of the 'fibrous dysplasias' of bone, and has some histological features very similar to those of osteitis fibrosa, though without the tendency to form cysts and collections of osteoclasts. The marrow spaces between the trabeculæ are filled with fibrous tissue. The dense regular cortex of a normal bone in Paget's disease is replaced by a spongy tissue which is two or three times thicker than the normal cortex and which is not clearly demarcated from the medullary canal which is correspondingly small, or even absent. The blood flow through the diseased bone is so increased as to constitute such an arterio-venous shunt as to cause left ventricular strain and even failure. The exterior of the bone is rough and pitted, due to the dis-

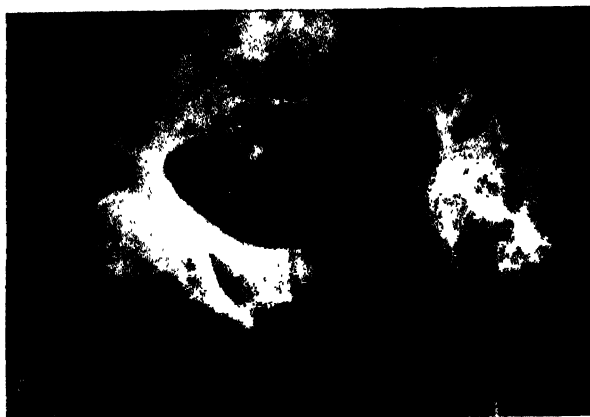


FIG. 290. — Paget's disease of pelvis and hips — 'etched' or striated appearance.

position of spongy subperiosteal bone. Though the bones are much thickened, they tend to bend and deform. **Radiologically** a number of variable appearances are present, often in the same patient, which range from an 'etched' or striated appearance (fig. 290), to those of such extreme density that no accurate bone detail can be seen (fig. 291). Sometimes the pathological process will affect only part of a single bone and the junction between normal and diseased bone may be quite sharp and clearly demarcated.

The pelvic bones, spine, tibiæ, and clavicles are usually affected in the early stages. Enlargement of the skull and the 'cottonwool' appearance in the X-ray are very characteristic. In this respect it is interesting to observe that the base of the skull and the facial bones are usually normal. Involvement of the pelvic bones radiologically often resembles secondary prostatic carcinoma. The conditions are distinguished by estimation of the acid serum phosphatase which is normal in Paget's disease, and, of course, it will only offer difficulty in the male, when a rectal examination of the prostate may clarify the picture (Chap. 47).

Chemical investigation of the blood reveals no abnormality in the serum calcium but the serum phosphorus may be elevated. The alkaline serum phosphatase is usually increased and may reach twenty or thirty times the normal value.

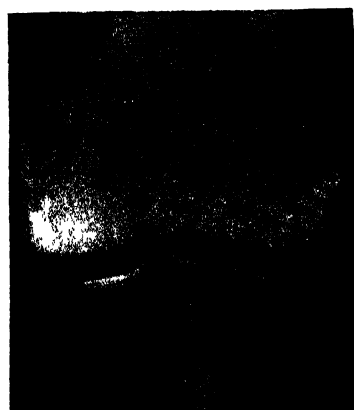


FIG. 291. — Paget's disease—monostotic—in lower end of femur; sclerosed appearance.

Clinical Features.—*Pain* is the most constant symptom, and is usually complained of long before the cause is realised. The tibia is one of the first bones to be affected, and the disease may remain localised in a single bone for years. The tibia in Paget's disease differs from a syphilitic osteitis in that the whole bone bends, whereas in the latter condition the curvature is mainly due to periostitis affecting the anterior part of the bone, so that the palpable inner border is comparatively straight. Pain is intermittent, and in the case of a subcutaneous bone, hyperæmia of the skin occurs during exacerbations.

Diminution of stature, due to kyphosis and bending of the long bones of the legs (fig. 292). A diminution of 13 inches (32 cm.) is recorded. The anterolateral bowing of the femora and the kyphosis give a simian or gorilla-like appearance in the generalised form of this disease.

Increased diameter of the head, an early indication of which may be the necessity for larger hats (fig. 293).

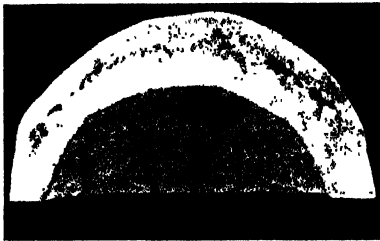


FIG. 293.—Paget's disease of the skull. (R.C.S. Museum.)



FIG. 292. — Advanced Paget's disease, showing large head and multiple bony deformities.

Complications.—*Spontaneous fracture* is common, and may first bring the patient under supervision. Radiographs often reveal partial transverse fracture, and probably a spontaneous fracture occurs in stages. The transverse character of the pathological fractures in Paget's disease is a very distinctive feature and may lead to a suspicion of Paget's disease before confirmatory changes in the radiological texture of the bone is noticed.

In about 5 per cent. of cases Paget's disease is terminated by the development of *bone sarcoma*; this may be either of the bone-forming or bone-destroying type, and its behaviour is not different from bone sarcoma occurring independently of Paget's disease.

Osteoarthritis occurs in joints adjacent to deformed bones, owing to alteration in the mechanics of the joint. Some degree of deafness is common, due to changes in the bony framework of the internal ear.

Paraplegia occasionally occurs, due to involvement of the vertebrae.

Death commonly results from intercurrent pulmonary complications, or from cardiac failure.

Treatment.—There is no known curative agent available to avert the progress of Paget's disease. In some cases of Paget's disease the pain due to faulty mechanics can be relieved by osteotomy, and satisfactory union is the rule. Potassium iodide sometimes may relieve pain in this condition, as with most chronic inflammations of bones, but aspirin mixtures are better tolerated over long periods.

OSTEITIS FIBROSA CYSTICA (*syn.* RECKLINGHAUSEN'S DISEASE OF BONE)

This disease usually becomes obvious in the second decade of life, and generalised active bone resorption results in diffuse cystic changes that are widely scattered throughout the skeleton, involving particularly the long bones and the skull. The destruction of bone is associated with the development of fibrous tissue, and sometimes 'brown tumours' structurally comparable with giant-cell tumours of bone develop. Fractures, pain, bending of bones, and grotesque deformities occur, so that the patient becomes bedridden.

Cases of generalised osteitis fibrosa have an unduly high calcium content in the blood, often as high as 16 to 28 mg. per 100 ml. instead of the normal 9 to 11 mg., and metastatic calcification is sometimes associated with the disease.

Cases sometimes present themselves with bilateral renal calculi, treatment of which is postponed until the possibility of a parathyroid tumour has been considered.

The blood phosphorus is either normal or diminished in amount. Even when a parathyroid tumour is not palpable, i.e. 80 per cent. of cases, careful exploration is rewarded by its discovery, either embedded in the thyroid gland or lying at a lower level in the mediastinum. Removal of the parathyroid tumour is followed by an immediate drop in the calcium content of the blood, and by rapid amelioration of the symptoms. Following such an operation, reconstruction of the decalcified bones takes place.

Polyostotic Fibrous Dysplasia.—This is the fibrocystic disease of bone which manifests itself by softening, bending, and often fracture, in which there is no evidence of hyperparathyroidism. The distribution of the affected bones is asymmetrical and often shows a tendency to affect only one limb (monomelic). The condition differs from the fibrous dysplasia of hyperparathyroidism in that there are no alterations in blood chemistry, no generalised osteoporosis of the whole skeleton, and no bone pain. The condition is present in early life and leads to great deformity of the affected limb. The skull is often affected (p. 474). Radiologically the affected bones are porotic and distorted with a thin expanded cortex and cystic changes in the marrow. In consistency the bones are soft and can often be cut with a knife. The aetiology is unknown.

Albright's Syndrome.—This is a variant of polyostotic fibrous dysplasia associated with areas of pigmentation of the skin. When it occurs in young females it is characterised by precocious puberty.

LOCAL CYST

This condition usually appears at the end of the first decade. The cyst first arises in the metaphyseal region of a long bone and subsequent growth often leaves the cyst at some distance from the metaphysis (fig. 294). Though commonly classified under the heading of 'cystic diseases of bone' the solitary bone cyst is really quite a separate condition. It is the residue of

an abnormal phase of activity affecting one epiphyseal line in a growing child. Subsequent growth is quite normal and if the cyst is treated the child develops normally in every respect.



FIG. 294. — Simple solitary bone cyst.

Coxa vara sometimes follows a cyst in the femoral neck. Cysts in other bones usually attract the surgeon's attention because of spontaneous fracture. Radiography shows a clear cavity in the bone, which later becomes expanded.

Treatment consists in exposure of the bone and curettage of the cyst, which contains straw-coloured fluid, and is lined with a fibrous wall. Frequently the operation is completed by packing with bone chips though this is not always necessary. There is no abnormality of the calcium content of the blood, nor any parathyroid derangement with local cysts of bone.

LEONTIASIS OSSEA

This condition consists of an enlargement of the facial bones and jaws so that the air sinuses are diminished in size, and the shape of the face is grossly altered by the external swellings. In the past the condition was thought to be a 'creeping periostitis' of infective origin, but the present opinion is that it is a localised form of fibrous dysplasia (p. 474).

The early symptoms may be those of lachrymal duct or nasal obstruction. The facial bones then become enlarged, and adjacent bones are successively attacked. Eventually hideous deformity results (fig. 295), and the patient's sufferings are increased by pressure on the eye, brain, and cranial nerves.

Leontiasis ossea has been mistaken for sarcoma of the maxillary antrum, chronic osteomyelitis of the jaws, bone syphilis, and 'frog face' due to displacement forwards of the maxillæ by naso-pharyngeal tumours.



FIG. 295. — Leontiasis ossea. (G. K. Kirkland, Oldham, Lancs.)

OSTEOGENESIS IMPERFECTA (*syn.* FRAGILITAS OSSIUM)

This rare familial condition is due to some congenital defect in the evolution of the connective-tissue cells. Normally, some of these develop into fibrous tissue, and others in connection with the osseous system become bone-forming cells. The blue sclerotics so characteristic of this disease are not simply due to diminution in thickness, and no abnormality can be detected on microscopic examination. The translucency is therefore due to some peculiarity of the fibrous tissue.

The main clinical feature is an abnormal tendency for bones to fracture. Pre-natal and post-natal types are distinguished, the former frequently being incompatible with survival and the latter often being so slight as to manifest the first fracture even as late as five years of age. Thus the foetus may be still-born, the skull being represented by a membranous bag with a few small bony plates, and evidence of antenatal fractures is common. In the infantile type the child is born alive, but the fragile limbs break with distressing ease. In less severe cases fractures begin to occur in childhood or adolescence. Stature is diminished, the skull is commonly flattened, and the ears pointed, so that the patient has an elf-like appearance. The fractures are said to be less painful than those occurring in normal bones, and union occurs readily. Otosclerosis commonly develops about the third decade.

Treatment consists of dealing with fractures as they arise, and of protecting the patient from the risk of injury.

OSTEOCHONDRITIS

Various lesions, the pathology of which is doubtful, are grouped under this term. The essential change is spontaneous fragmentation of the ossific nucleus followed by reconstruction in a slightly deformed shape. Interference to the blood supply following trauma is traditionally given as a causative factor, but it is unsatisfactory when one considers that all healthy

children suffer repeated traumata without ill-effects. The essential ætiology is unknown but the bone changes appear to be a sequel to localised ischæmia.

In most cases symptoms are relatively mild and comprise aching of the affected limb and local tenderness. Relief is afforded by rest and prevention of strain. If the affected portion is palpable, such as the tibial tubercle, enlargement can be detected.

The more important of these conditions are :

Perthes Disease (*syn.* Coxa Plana, Pseudo-coxalgia).—This condition appears between the ages of five and ten, with a preference for the earlier ages, and is three times more common in boys than girls, and 15 per cent. of cases are bilateral. Calvé, of Paris, suggested the name 'pseudocoxalgie,' in order to distinguish the condition from tuberculous arthritis, because before the advent of radiology some cases diagnosed as tuberculous disease often surprised surgeons by their recovery of joint function and these were almost certainly suffering from Perthes disease. Pain and limp, especially after vigorous use, are the early symptoms. On examination, wasting is slight, and movements are restricted according to the extent of bony change. As the head of the bone becomes flatter, so rotation is progressively restricted, and if coxa vara supervenes, then abduction also is limited. Flexion and extension, however, are free and painless, and this feature, combined with negligible wasting of muscles, and the robust health of the patient, should prevent an erroneous diagnosis of tuberculous disease. In these cases a week in bed results in the disappearance of the muscular spasm, after which the characteristic features of the underlying condition can be recognised.

A radiograph shows, in the early stages, slight broadening of the neck of the femur, the upper border of which is *convex*. Later the head of the bone becomes flattened ('mushroomed'), and the epiphysis is represented by two or more fragments, often of increased density suggesting ischæmia



FIG. 296.—Perthes disease of the left femoral head.

(fig. 296). Finally the neck becomes thickened, and the epiphyseal fragments fuse to form an expanded and flattened head.

Surgeons were formerly content to restrict movements and limit weight-bearing, as by the application of a walking caliper. However, it is becoming increasingly appreciated that, although a deformed femoral head causes little disability for many years, osteoarthritis is prone to develop in later life.

Therefore treatment by recumbency and traction is to be recommended, in a form modified from that used for a tuberculous hip. Many surgeons consider that even this precaution will not always prevent osteoarthritis in later life, and that prolonged absence from good schooling and parental influence must be taken into account.

Osgood-Schlatter's disease is much commoner in boys than girls, and appears between the ages of ten and sixteen. It is frequently preceded by some unusual strain, e.g. training for sports. The tibial tubercle becomes unduly prominent and tender on pressure. A radiograph shows partial separation of the tongue-shaped portion of the epiphysis from the shaft (fig. 297). Vigorous activities should be curtailed, and firm strapping provides mild support. If pain persists, a posterior plaster slab should be worn for a few weeks. Symptoms persist despite treatment, frequently as long as twelve months.

Sever's disease, or apophysitis of the os calcis, affects the epiphysis of the heel, which is present between the tenth and sixteenth years. A radiograph shows fragmentation and irregularity of the epiphysis (fig. 298). The child should wear a boot which is cut away at the back (to relieve pressure) and the heel of which is raised (to relax the calf muscles). It would appear that this condition is becoming increasingly rare.

Köhler's disease affects the scaphoid tarsal bone and occurs between three and eight years of age. The bone is at first fragmented and tender, but later is compressed and sclerotic (fig. 299). Strapping support is usually adequate.

Calvé's epiphysitis affects the epiphysis, usually of one single vertebra in infants or very small children. It is often mistaken for tuberculosis. No deformity or disability results in later life.

Scheuermann's disease involves the dorsal vertebral epiphyses in adolescents (p. 327). The development of a kyphosis is the first indication of the disease, which is confirmed by radiography. Five or six vertebræ at the mid-thoracic level become



FIG. 297.—Osgood-Schlatter's disease. Notice the decalcification which occurs in the early stage of the disease.



FIG. 298.—Sever's disease.



FIG. 299.—Köhler's disease, showing disc-like scaphoid.

wedged to give a round kyphosis. In early stages remedial exercises and perhaps a spinal support improve the deformity, but some kyphosis persists.

Freiberg's disease is uncommon and occurs in young adults. It affects the head of the second or third metatarsal, in the region of which tenderness and swelling are

Robert B. Osgood, 1873-1956, Orthopaedic Surgeon of Boston, U.S.A., and Carl Schlatter, 1864-1934, Professor of Surgery, Zürich, described apophysitis of the tibia simultaneously in 1903.
James W. Sever, 1878-1964. Consulting Orthopaedic Surgeon, Children's Hospital, Boston, Mass., U.S.A.
Alban Köhler, 1874-1947. Professor of Surgery, Berlin.
Holger Werfel Scheuermann, 1877-1960. Director of Radiological Department, Military Hospital and Sundby-hospital, Copenhagen.
Albert H. Freiberg, 1868-1940. Professor of Orthopaedic Surgery, Cincinnati, Ohio, U.S.A.

detected. The X-ray shows flattening of the articular surface and irregular sclerosis of the head. Treatment consists of a metatarsal bar, or, if necessary, at a later date, excision of the head of the bone (fig. 300).

Kienböck's disease of the semilunar bone of the wrist occurs in adults of any age (fig. 301). In most cases a history of injury is obtainable, which is followed by pain, tenderness over the bone, and limitation of wrist movements. It is remarkable, and difficult to correlate with the ischæmic theory of origin, that it rarely,



FIG. 300.—Freiberg's disease of the head of the second metatarsal. (F.P. Fitzgerald, F.R.C.S.I., London).



FIG. 301.—Kienböck's disease of carpal semilunar. (J. Hindenach, F.R.C.S., London).

if ever, follows dislocations of the semilunar in severe injuries of the wrist. It is to be noted that though classed as 'osteochondritis' this particular condition of the semilunar is one of adult life. Treatment consists in immobilisation for six months in order to encourage regeneration. Severe cases pass on to traumatic arthritis of the wrist joint which may be controlled with a permanent leather wrist-strap or, in a few cases, necessitate wrist fusion.

ACHONDROPLASIA

This familial condition is due to maldevelopment of bones arising from cartilage. Thus the stature is markedly diminished and the limbs in particular are stunted. The legs are obviously short, and the fingertips reach only to the great trochanters, the arms thus resembling flippers (fig. 302). The fingers themselves diverge, so that they resemble the spokes of a wheel. As the fibula is less shortened than the tibia, it frequently enters into the formation of the knee joint. The base of the skull, being developed from cartilage, is small in proportion to the vertex, so that the prominent forehead causes the bridge of the nose to appear to be depressed. Mental development is normal, and sufferers often find ready employment in circuses.



FIG. 302.—Achon-droplasia.

CRANIO-CLEIDODYSTOSIS

is thought to be due to failure of development of bones arising in membrane. Thus the vertex of the skull and clavicles are ill-formed, so that the head appears flattened, and as the buttress action of the clavicles is lost, the heads of the humeri are approximated to, and may articulate with, the sternum (fig. 303). A curious feature is delayed ossification of the pubic bones.

DIAPHYSEAL ACLASIS (syn. MULTIPLE EXOSTOSES)

This is a not uncommon hereditary condition characterised by the outgrowth of cancellous osteomas near the ends of long bones. Sometimes all the long bones may be affected and dozens of growths may be present, but most commonly six to twelve outgrowths are encountered. The femur and tibia near the knee and the humerus at the shoulder are the most common sites.

The bone may be distorted in growth and a little dwarfing may occur. The outgrowth is often pedunculated with a cartilage cap on the tip from which it grows and a bursa overlying it (fig. 309). The condition is regarded as a failure of 'tubulation' of the growing bone, i.e. a failure of the periosteum to remodel the external surface of the new bone produced by growth at the epiphysis. Some tumours may cause mechanical symptoms and need removal.



FIG. 303. — Cranio-cleidodysostosis.

**DYSCHONDROPLASIA (syn. MULTIPLE ENCHONDROMAS)
OLLIER'S DISEASE**

This differs essentially from multiple exostoses in that the growing epiphysis leaves behind masses of cartilage in the centre of the shaft. The lesion is thus cartilaginous and endosteal—not periosteal and of cancellous bone as in diaphyseal aclasis. There is no hereditary tendency. The condition may be localised to a single bone or single limb. When this happens the affected bone is dwarfed and the skeleton becomes asymmetrical. The long bones are affected—especially the femur and tibia at the knee and the upper end of the humerus and lower end of radius. Occasionally the phalanges of hands and feet are affected (fig. 306).

SCHÜLLER-CHRISTIAN'S DISEASE

is rare, and is a disease of the reticulo-endothelial system and concerned with faulty lipid metabolism. Soft swellings occur in the scalp, beneath which the skull is eroded ('map-like' skull). Exophthalmos and diabetes insipidus develop and growth is retarded. Other bones are sometimes affected, in which case differential diagnosis is often difficult. The condition responds to deep X-ray therapy. Schüller-Christian's disease occurs in adult life, but two similar conditions occur in childhood to which different names are attached. In adolescent and young patients localised lesions in bone having the same pathology are termed *eosinophilic granuloma*. In infancy an analogous condition occurs which is almost always fatal, and is known as *Letterer-Siwe* disease.



FIG. 304. — Osteopetrosis of the lumbar vertebrae. (Prof. Carl Krebs, Aarhus, Denmark.)

OSTEOPETROSIS (syn. 'ALBERS-SCHÖNBERG'S DISEASE') is a rare and familial condition in which the bones become progressively more dense owing to excessive deposition of calcium (fig. 304). 'Marble bones' is a misnomer, as although the bones are sclerotic they are friable and pathological fractures occur. *Osteopoikily* (speckled bones) is a similar but less extensive condition.

ACROMEGALY

Acromegaly is an endocrine disturbance, being the stimulation of bone growth, after fusion of the epiphyses, by the pituitary secretion of an eosinophilic adenoma of the anterior lobe of the pituitary.¹

¹ Hunter's famous example of acromegaly, the skeleton of the Irish giant, now in the museum of the Royal College of Surgeons of England, showed an enlarged sella turcica when the skull was opened at Harvey Cushing's suggestion.

The early signs are enlargement of the hands and feet, at first confined to the soft tissues, but later bony thickening also occurs. The jaws enlarge, especially the lower, which becomes prognathic, and separation of the teeth indicates that the enlargement is partly interstitial. Overgrowth of facial bones also occurs, especially of natural ridges and at the sites of muscular attachment. The frontal sinuses of the skull are markedly enlarged. The lips, nose, and ears show a variable amount of thickening (fig. 462). When the disease is established the spine is kyphotic.

As the disease is associated with new-growth and enlargement of the pituitary gland (p. 393), symptoms of increased intracranial pressure supervene. Vision is affected, partly owing to this general increased pressure, and also as a result of local pressure of the enlarged gland on the optic chiasma giving rise to bilateral temporal hemianopia. More rarely an optic nerve is compressed with consequent blindness of the corresponding eye. Involvement of the fifth nerve, and proptosis from pressure on the cavernous sinus, to which the ophthalmic veins pass, sometimes occur.

If the eosinophilic adenoma develops before puberty, there will be generalised stimulation of growth—gigantism—and later these cases often show the facial signs of acromegaly.

A radiograph demonstrates enlargement of the sella turcica (fig. 460).

Operation should be undertaken only for intolerable headache or threatened blindness.

Harris's Lines.—Radiographic examination of long bones in children and adolescents sometimes reveals transverse lines of compact bone near the epiphyses (fig. 305). These are due to arrest of growth which accompanies some severe constitutional disturbance.

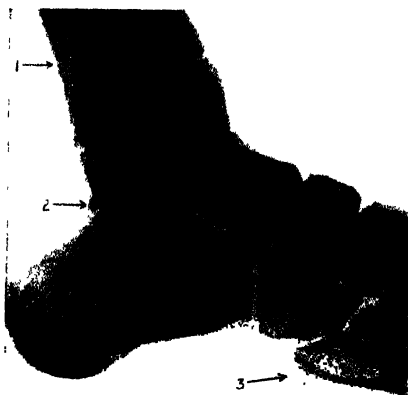


FIG. 305.—(1) Harris's lines. Two abnormalities are also seen—(2) an os trigonum, and (3) an epiphysis at the base of the fifth metatarsal. These may be of importance as alleged fractures in compensation cases, but an X-ray of the other foot will show a similar condition.

TUMOURS OF BONES

The various types of bone tumours are the neoplastic counterparts of the different tissue components that constitute bone. Thus we can relate chondroma and chondro-sarcoma to cartilage, bone-sarcoma to bone-forming osteoblasts, giant-cell tumour of bone to the bone-destroying osteoclasts, fibrosarcoma of bone to the periosteal connective tissues, and other tumours such as myeloma and Ewing's tumour to the hæmopoietic tissue of bone.

It is important to bear in mind that various groups of bone tumours are not to be regarded as absolutely clearly defined

and that although these 'labels' are the ones attached to the commoner types observed, an individual tumour may on occasions show features of more than one of these types (Sissons).

There have been several attempts to classify primary tumours of bone on a pathological basis, but none have been satisfactory. In view of the failure of attempts to make a precise classification a helpful one, Platt recommends the broadest possible approach, as set out below.

CLASSIFICATION OF BONE TUMOURS (PLATT)

Primary	{	Benign
		Osteoma
		Chondroma
		Locally Malignant
		Osteoclastoma
		Malignant
		Periosteal Fibrosarcoma
		Osteogenic Sarcoma (sclerosing and osteolytic types)
		Chondrosarcoma
		Ewing's Sarcoma
		Multiple Myeloma
Secondary		Secondary to primary neoplasm arising in other tissues

CHONDROMA

Cartilaginous tumours, usually benign, may arise in connection with the epiphyseal cartilage.

The tumours are composed of hyaline cartilage, the cells of which are variable in size and shape. All stages of transitional forms link non-progressive developmental abnormalities and benign chondromas to malignant chondro-sarcomas, and no sharply dividing line can be drawn between the two extremes.

Chondromas are conveniently classified according to the type of bone from which they arise :

(a) **Small Bones of the Hands and Feet.**—As these tumours arise within the bone, they are termed *enchondromas*. They most commonly appear during childhood. The affected bone becomes gradually and painlessly expanded, and the local condition may suggest dactylitis. However, the more advanced age of the patient and the absence of evidence of local inflammation or of any general manifestation of disease should prevent an error of diagnosis. A radiograph shows a clear expansion of the bone (fig. 306), and sometimes the presence of small specks of calcification help to distinguish it from a bone cyst. If allowed to grow, destruction of the bone is inevitable, and finally myxomatous degeneration may occur.

It is to be noted that if multiple enchondromas occur, the condition is probably not truly a neoplasm but is a generalised disease of the skeleton (i.e. Ollier's disease, p. 253). The true neoplasm usually occurs as a solitary enchondroma.



FIG. 306. — Multiple enchondromas of phalanges.

Boeck's sarcoidosis may simulate enchondromas in that cavitation sometimes occurs in digital bones. However, the presence of granulomatous swellings in lymph nodes, salivary and lachrymal glands, and elsewhere (p. 158) should clarify the diagnosis.

Treatment consists in scraping out the tumour. In the case of a digit a postero-lateral incision is made, which passes between the extensor tendon and the digital vessels and nerve.

(b) **Long Bones.**—Centrally placed chondromas can occur near the ends of long bones, but in adults there is always a strong suspicion of malignancy. The upper end of the humerus is a common site in the adult.

(c) **Flat Bones.**—These tumours grow from such bones as the ribs, scapula, and pelvis, and form characteristically hard and painless swellings. However, they may remain unnoticed until myxomatous degeneration causes pain and increase in size and a malignant transformation may be suspected and proved. In the pelvis and upper end of the femur these cartilaginous tumours may attain very large dimensions (fig. 307). As there is frequently extensive bone formation scattered among the cartilage (and giving a mottled appearance in the X-ray), these tumours are termed osteochondromas or ossifying chondromas. They usually remain benign and may cause trouble by mechanical obstruction, but sarcomatous changes are always to be feared.

Treatment of these large osteochondromas is by excision whenever possible. The operation may be formidable in the pelvis and there are



FIG. 307.—Osteochondroma of the ilium.

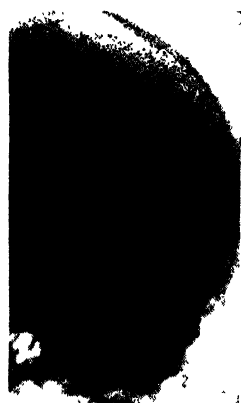


FIG. 308.—Ivory osteoma. An attempt at local removal (abroad), terminated when the osteotome broke—the fragment is seen embedded in the tumour. (Professor Norman Dott, Edinburgh.)

occasions when amputation of the affected extremity is necessary—taking with it the affected half of the pelvis (hemipelvectomy or inter-innomino-abdominal amputation).

OSTEOMA

Osteomas are of two varieties, ivory or cancellous.

Ivory osteomas are uncommon, but are occasionally found on the skull, particularly in connection with bones which form the walls of air sinuses (fig. 308).

Treatment.—Removal through normal bone is indicated if pressure symptoms

result, e.g. deafness from auditory obstruction, displacement of the eye, or involvement of nerves.

Cancellous osteomas are comparatively common tumours and are found near the ends of long bones (fig. 309). It is to be noted that these cancellous 'osteomas' are probably not true neoplasms because growth ceases when ossification of the bone is complete; when multiple, as they frequently are, they are the result of a growth disorder (failure of 'tubulation' of the growing end of the bone) and should be classed as diaphyseal aclasis (p. 253).

A **subungual exostosis** is an irregular bony outgrowth under the nail of the big toe, which is lifted off the underlying phalanx (fig. 310). It is included in this section for convenience, but it



FIG. 310.—Subungual exostosis.
(K. I. Nissen, F.R.C.S., London.)

is not a neoplasm. It is due to pressure of an ill-fitting boot and consequent periosteal irritation. The nail should be removed or displaced, and the bony excrescence excised by means of a chisel or bone-cutting forceps.

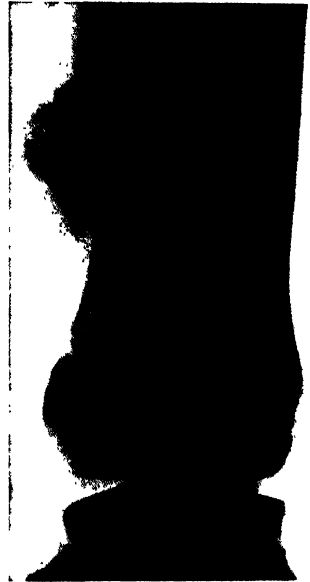


FIG. 309.—Typical cancellous osteoma, growing away from the epiphysis. A fracture has occurred near the base.

GIANT-CELL TUMOUR (syn. OSTEOCLASTOMA)

The giant-cell tumour occupies a middle place in the range of malignancy of bone tumours from the completely benign (the aneurysmal bone cyst) to the malignant. In general it is a *locally malignant* tumour with an occasional tendency to metastasis.

Giant-cell tumours occur most commonly at the end of a long bone, particularly in the vicinity of the knee joint. A very characteristic feature is that they always originate in what originally was the epiphyseal region of the bone. Tumours with similar histological structure occur in relation to tendons (p. 314), where they arise from synovial tissue. On the gums a similar tissue gives rise to one variety of epulis (p. 470), but that which occurs in the jaws is probably not a true neoplasm but a giant-celled granuloma (p. 475).

Osteoclastomas usually occur during the third or fourth decades of life, and are presumed to arise from osteoclasts, which are giant cells normally engaged in absorption of bone. Clinical features depend upon whether the affected bone is subcutaneous or surrounded by muscle. If subcutaneous, a swelling is noticed first which is painless. Expansion of the bone follows, which process consists in destruction of the bone from within, while at the

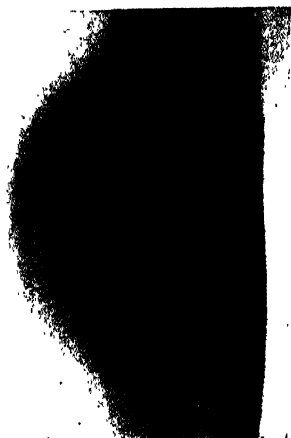


FIG. 311.—Osteoclastoma of the fibula with gross expansion of shaft.

same time a thin shell of new periosteal bone is formed. This new formation of bone occurs more slowly than the destructive process, so that the bone becomes larger but progressively thinner, and eventually 'egg-shell crackling' may be detected. The expansion of the tumour is characteristically eccentric and extends into the articular extremity of the bone to reach a site subjacent to the articular cartilage. Finally, the growth, if ignored, erodes the compact bone, and a soft, pulsating swelling results (fig. 311).

In the case of deep-seated bones, enlargement and consequent destruction of the bone may be unnoticed, in which case the first evidence of the tumour is either a spontaneous fracture or a pulsating swelling.

Owing to the slow rate of growth of these tumours, pain is not a prominent feature though a dull ache is often present. When a large osteoclastoma is adjacent to a joint, e.g. the lower end of the femur, a 'sympathetic' effusion may occur as a result of local hyperæmia. A radiograph confirms the abrupt expansion of the bone, and presents well-marked bony trabeculæ which give the appearance of a multilocular cyst; in fact the cyst is unilocular and the trabeculæ are thickenings in the cyst wall. The appearance often resembles a collection of soap bubbles.

In the diagnosis two features are worthy of stress by repetition: the tumour arises in the epiphyseal region of the bone, and is rare before the age of thirty years.

Macroscopically, the tumour appears as a soft, maroon-coloured tumour, with localised extravasations of blood. Histologically, characteristic giant-cells are strikingly evident; these large cells, about $120\ \mu$ in diameter, are irregular in shape, and contain from twelve to fifteen deeply staining nuclei. The remainder of the tumour consists of spindle cells, extravasated blood, and numerous blood-vessels. 'White' osteoclastomas occasionally occur, usually at the lower end of the radius.

Treatment.—Until recently it was believed that osteoclastomas were only locally malignant, but a more careful 'follow-up' shows that in about 8 per cent. of cases metastases occur in the lungs. Whenever possible surgical removal should be the method of choice. X-ray treatment of osteoclastomas has been used with great claims for success, but the heavy irradiation which is necessary to kill the tumour cells, which are not specially sensitive, at the same time destroys the articular cartilage of the involved joint, and though the tumour may be necrosed the joint is the seat of painful arthritis. In the spine and pelvis, deep X-radiation is usually the only procedure available.

At the knee, and most other sites in the extremities, curettage of the cavity, followed by packing with bone chips, will give excellent results if carried out radically.

Curetting.—The bone is opened with a gouge or chisel, and the growth scraped away with a sharp spoon, care being taken not to penetrate the adjacent articular cartilage. The cavity is swabbed with pure carbolic acid, any excess being removed with spirit. If necessary, the cavity is reduced in size by compression of its walls, and bone chips may be inserted. If the cavity is small, it is sufficient to allow it to fill with blood-clot.

In some cases one of the following procedures will be preferable to curettage or radiation :

(a) **LOCAL EXCISION**.—If removal of the bone will cause little disability, then excision is performed, e.g. rib or fibula. This method is certain in its result, and convalescence is speedy.

In the case of the upper end of the fibula the external popliteal nerve must be isolated above the swelling, as it lies under the tendon of the biceps, since its anatomical relations in the region of the tumour will be distorted. The nerve is traced downwards and held aside, and the fibula is divided and cleared of muscles from below upwards.

(b) **AMPUTATION**.—In the case of extensive destruction of a bone, such as the lower end of the femur or head of the tibia, amputation is usually performed, rather than to attempt to arthrodesis the knee by bridging a gap by a bone graft at the site of a resection.

MULTIPLE MYELOMA (*syn.* **KAHLER'S DISEASE**)

This is a not uncommon condition in which multiple endosteal tumours occur and are associated with marked bone destruction. The lesions involve spine and ribs most frequently and the skull and femora are other common sites. The proliferating tumour cells are plasma-cells, and for this reason the condition is sometimes known as plasmacytoma. The involvement of bone marrow

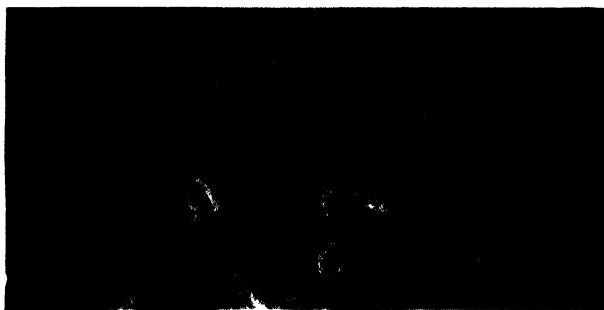


FIG. 312.—Myelomatoses of sternum and ribs.
(Dr. L. S. Carstairs, London.)

is widespread and the sternal marrow is practically always affected even in the absence of clinical evidence, thus the diagnosis can often be confirmed by examination of smears of sternal marrow. The multiple nature of the lesions and the sharply delimited bone destruction gives a radiographic appearance resembling metastatic carcinoma (fig. 312), and it may cause the pathological fracture of affected long bones. The proteose, first described by Bence-Jones, appears in the urine in most cases, but it may absent itself for a period in any patient. It precipitates on the addition of nitric acid and disappears on warming. Owing to extensive bone destruction the blood calcium is usually increased, and if renal insufficiency coexists the blood phosphorus is also raised. The condition is associated with severe anæmia and is encountered most commonly over the age of fifty years.

In a relatively small number of cases only a single bone is involved by a myeloma lesion. Sometimes these cases remain well after X-ray therapy, but more often the development of additional lesions shows the condition to be one of generalised disease. Death may result from uræmia following obstruction of the renal tubules by protein, or some intercurrent disease in a bedridden sufferer.

SARCOMA

Periosteal fibro-sarcomas are spindle-shaped tumours which arise from the periosteum, or from the insertion of tendons or muscles, and strictly speaking should

therefore be regarded as tumours of these structures rather than of the bone itself. Local and free excision, including the periosteum in the neighbourhood, occasionally results in cure, but, as in the case of fibro-sarcoma arising from a muscle sheath, incomplete removal will be followed by recurrence of the tumour with increased malignancy. They are very slow growing, and though tending to recur after local removal the survival rate is favourable even if amputation is necessary.

Osteogenic sarcoma is a highly malignant tumour which is believed to arise from the osteoblastic cells of bone. This, however, does not mean that it always produces new bone—indeed in many cases only bone destruction is in evidence. One presumes that the bone-forming sarcomas arise from the later stages of development of the osteoblast and the osteolytic sarcomas from the more primitive stages of development of the osteoblast. The clinical presentation and the radiographic appearance show great variations, depending on the osteogenic or osteolytic properties of the tumour and on its site of origin. In the same way the histological appearances often show a large variety of cells indicating origins in a tissue which is a common precursor of cartilage, bone, fibrous, and myxomatous tissue. Dissemination occurs by the blood-stream.

Osteogenic sarcoma most commonly occurs during puberty or adolescence, and is practically unknown after the age of fifty, except as a complication of Paget's disease. The favourite sites are the ends of the shafts of long bones. The leg is affected five times as often as the arm, and the majority of these tumours occur in the region of the knee (lower end of femur, upper end of tibia). The patient's general health is not affected until the final stages of the disease.

On inspection of the affected part large distended veins are often seen in the skin overlying the tumour. On palpation a spindle-shaped swelling is detected of bony-hard consistency. Characteristically, the mass is at the end of a long bone arising in the metaphyseal region. Eventually soft tissues are invaded, and finally the tumour involves and fungates through the skin.

Dissemination occurs early, the lungs being commonly affected via the systemic veins (fig. 313). A blood-stained pleural effusion is sometimes the first evidence of pulmonary involvement.

The radiographic appearances of bone sarcomas vary according to the rate of growth of the tumour. In some cases (osteolytic type) erosion of the bone is the principal feature, but in others (osteogenic type) characteristic spiculation of periosteal new bone is sometimes evident (fig. 314). Frequently the production of new bone is seen best where the limits of the tumour are stripping up the periosteum from the surrounding bone. This gives an appearance often known as Codman's triangle, but it



FIG. 313. — Ossifying metastasis in the lung from osteosarcoma of the femur.

has no special diagnostic significance.

Problems in diagnosis.—Radiological signs by themselves are often misleading. Diagnosis can only be by a balance of history, clinical appearance,

biopsy, and radiology. Clinically, the presence of a palpable extra-ossseous mass is essential to the diagnosis if the lesion is in a site capable of palpation. The mass will always be bigger than the radiological changes might suggest



FIG. 314.—'Sun-ray' pattern in osteogenic sarcoma. Rare site. Most common site is the knee. 'Sun-ray' appearance is not common.

(fig. 315). Similarly, in the history, the appearance of a palpable mass for some time before the development of pain is against a sarcoma, where pain always precedes the appearance of a palpable mass.

A tumour commencing endosteally erodes and destroys bony tissue as it expands, and eventually spontaneous fracture may occur or the appearance of a soft pulsating swelling indicates that the bone is extensively destroyed.

Besides the other types of primary bone tumour, such possibilities as an acute inflammatory process, an endosteal gumma, or a metastatic tumour must always be considered.

Biopsy.—If the diagnosis is doubtful and the W.R. negative, then a biopsy must be performed, as exact diagnosis is essential for treatment and prognosis. Biopsy is alleged to encourage dissemination, but if a tourniquet and diathermy are employed, the slight risk is far outweighed by the importance of a diagnosis. Diagnosis can be so difficult that no amputation should ever be proposed without a previous biopsy lest a limb be sacrificed for what later may be found to be a non-malignant condition.

Treatment of an osteogenic sarcoma depends on the site and extent of the growth:

(a) *Amputation* is the usual procedure, providing the diagnosis is accepted and an X-ray shows that the lungs are cleared, and should be performed as high as possible above the tumour. In the case of the femur or humerus disarticulation should be performed through the hip or shoulder joint, or even by a hind- or fore-quarter amputation.

However, the prognosis is extremely gloomy, but amputation relieves the patient of a limb which will become increasingly painful, and fungation and risk of secondary hæmorrhage are obviated.



FIG. 315.—*Osteogenic sarcoma*: Note that the tumour is metaphyseal and eccentric and that the underlying cortex is eroded. The extra-ossseous mass seen here is much larger than what might be expected from the mere trace of new bone which was present in the radiograph.

(b) In certain situations local excision of the affected bone and the insertion of a bone graft have been adopted. Earlier permission is likely to be given for this procedure, as compared with amputation. Also, in the early stages local recurrence is unlikely, and if secondary deposits have occurred, even amputation cannot retard their progress. Hence in situations where bone grafting yields good results, such as the upper limb, the arm may be saved, and cases are on record where this line of treatment has been adopted and the unmaimed patient has survived for many years.

X-ray therapy has little influence in retarding the progress of the tumour, and there are many surgeons who believe that the patient's general condition is rendered more miserable by X-ray therapy than without it.

Prognosis.—Out of 650 cases collected by the American Registry of Bone Sarcoma, only seventeen *appear* to have been cured—sixteen after amputation, and one following treatment by radium. Platt reported twenty-three five-year survivals after amputation in 128 patients.

Recurrence within a year is likely to occur in viscera or other bones if the primary growth is situated near the trunk. In more distant tumours recurrence is usual within three years, although we have known a case in which secondary deposit appeared in the spine thirteen years after amputation through the thigh for periosteal sarcoma of the tibia. Thus the time limit for a 'cure' is almost unlimited. An X-ray of the chest may reveal secondary deposits in the lungs, which indicates that the expectation of life is, at the most, a few months.

EWING'S TUMOUR

This rare tumour has the following characteristics : acute onset with pyrexia, local tenderness, a situation in the middle of the shaft of a long bone, a patient between five and sixteen years of age, a pattern of longitudinal layers of subperiosteal ossification likened to the layers of an onion—and, finally, striking radio-sensitivity. It is probable that a number of pathological entities can give rise to this syndrome, and some authorities consider the eponym should be discontinued. In addition to the 'angio-endothelioma' that Ewing regarded as its corresponding pathological counterpart, there must be mentioned not only other unusual forms of primary endosteal tumour (such as sarcoma derived from reticulo-endothelial tissues) but also metastatic lesions, particularly neuroblastoma, which occur in children of this age.

One of the characteristic clinical features of this disease is the ease with which it can be mistaken for a subacute, or chronic, osteomyelitis. Even the round cells which compose it have been mistaken for pus, and the temperature is often slightly elevated.

The prognosis is poor, even after amputation, as secondary deposits occur in other parts of the skeleton, and eventually in lymph nodes and viscera. Deep X-ray therapy causes striking retrogression of the primary growth, which is an important point both in differential diagnosis and treatment, but secondary deposits are less radio-sensitive.

SECONDARY TUMOURS

Secondary bone tumours are far commoner than primary bone tumours, and should always be considered first before the diagnosis of sarcoma is made.

Carcinoma.—Carcinoma of bone occurs either by direct extension, as in the case of the chest wall following carcinoma of the breast, or by metastasis.

Secondary deposits are liable to occur particularly as a result of a primary growth in the following situations :

(i) *Breast* is traditionally regarded as the commonest source of secondary carcinoma of bone. Secondary deposits occur in about 50 per cent. of fatal

ases, the favourite situations being the spine, pelvis, and upper ends of the femur and humerus.

(ii) *Prostate*.—This gland is commonly associated with osseous dissemination. The usual manifestation is diffuse sclerosis of the pelvis and lumbosacral regions (Chap. 47). This osteoblastic type of metastasis is peculiar to the prostate and, in the early stages, it may be difficult to distinguish the X-ray from Paget's disease (p. 246).

(iii) *Kidney*.—Any bone is liable to be affected, perhaps most commonly the pelvis. A bony swelling or a spontaneous fracture is sometimes the first evidence of a carcinoma of the kidney. Occasionally the deposit is solitary, and may reach a very large size while the primary hypernephroma is still very small. In these cases nephrectomy and resection of the affected bone are feasible.

(iv) *Bronchus*.—Carcinoma appears to be increasing in frequency and secondary deposits in bones are also becoming increasingly common. Some surgeons believe that the bronchus now rivals the breast in frequency of secondary deposits in bone.

(v) *Thyroid*.—The flat bones, especially the vertex of the skull, are likely to be affected. These tumours in particular are very vascular, and apparently are capable of function, as after complete thyroidectomy for carcinoma the post-operative myxœdema has disappeared on the appearance of secondary deposits.

Clinical Presentation.—Secondary deposits in bone usually cause very severe persistent pain, and if occurring in the vertebræ pain is liable to be referred along spinal nerves. In most cases a swelling eventually becomes palpable (fig. 316); the presence of superficial veins and possibly pulsation indicate the vascular nature of the tumour. Spontaneous fracture is common, and if immobilised, union often occurs if the patient survives for a sufficient length of time. The use of the intra-medullary nail for these pathological fractures in the femur is a great help in rendering these unfortunate patients comfortable.

A radiograph (fig. 317) shows irregular destruction of bone with little surrounding reaction unless fracture has occurred, in which case evidence of callus formation is sometimes seen.

If a tumour in bone raises a suspicion of secondary carcinoma, then the common primary sites must be carefully examined. Carcinoma of the breast, prostate, or bronchus can usually be detected by clinical examination. The presence of a slowly growing carcinoma of the kidney is often difficult to diagnose in the absence of hæmorrhage, but a pyelogram will usually show a 'spidery' pelvis. Primary carcinoma of the thyroid gland is sometimes so unobtrusive as to be impalpable, or a tumour which clinically appears to be a simple adenoma may actually be malignant.

Treatment.—Secondary osseous deposits are often sensitive to deep X-ray therapy. Pain is relieved, and a spontaneous fracture or paraplegia may be prevented and much suffering thereby obviated. Therefore, unless metastases are widespread, irradiation should receive consideration.

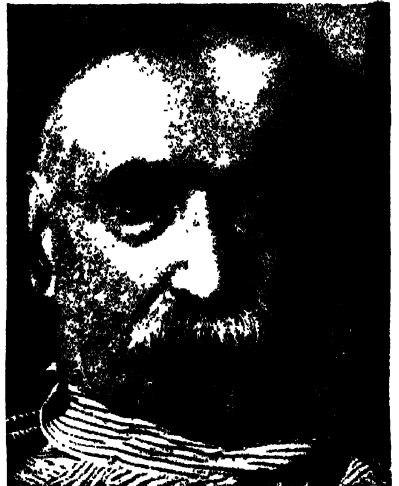


FIG. 316.—Secondary deposit in the skull from a carcinoma of the prostate.

Secondary deposits arising from the prostate are often checked, and may even temporarily disappear, following the administration of oestrogen (Chap. 47) and this can be combined with orchidectomy. Radioactive iodine has been used effectively in controlling secondary thyroid carcinoma. Secondary deposits from mammary carcinoma will often respond initially to large doses of testosterone but invariably recur later, and the value of adrenalectomy in this condition is doubtful.

The estimation of the amount of acid phosphatase in the serum is of value in the diagnosis of prostatic secondaries, and also in prognosis. Normally 0.5 to 2 units of acid phosphatase is present in 100 ml. of serum. Over 5 units is very suggestive of prostatic secondaries, and 10 units or more is diagnostic. The value of treatment by oestrogen is assessed by the degree of diminution in the amount of acid phosphatase. This test is also useful in distinguishing prostatic secondaries from osteitis deformans, as in the latter case the acid phosphatase is not increased, but the alkaline phosphatase is above normal.

OSTEIOD OSTEOMA

This is a rare condition first described by Jaffe, of obscure origin but probably inflammatory, causing an isolated, painful, bone lesion in young adults.

The patient complains of chronic aching pain, usually in the shaft of a long-bone but occasionally in a cancellous bone as in short-bones of the tarsus. No abnormality is found on clinical examination of the affected part. X-ray examination shows gross thickening of the bone affecting the cortex eccentrically. The denseness of the sclerosis may be so extreme that no special feature may be detected in an ordinary X-ray, but if films are made using extreme penetration it is sometimes possible to demonstrate a small central cavity, 0.5–1.0 cm. in diameter.

The condition is almost certainly self-limiting but it can cause pain for several years before spontaneous cure occurs. Operative treatment, which consists of a widespread removal of the bone until the cavity is reached, relieves symptoms immediately. When the central nidus is reached it is recognised by its dark red, or maroon, coloration. In a histological section the lesion is quite characteristic and easily recognised, consisting of a circular cavity in the surrounding sclerosed bone which contains a spherical mass of osteoid tissue and vascular stroma.

Henry L. Jaffe, *Contemporary*. Consultant Pathologist, Hospital for Joint Diseases, New York.

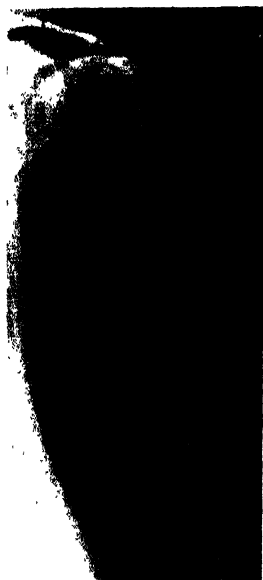


FIG. 317.—Secondary carcinoma, showing deposit in the humerus and a pathological fracture. The primary was in the breast.

CHAPTER 13

INJURIES TO JOINTS

JOHN CHARNLEY

Sprains are due to overstretching of ligaments with consequent partial rupture. Perhaps the ligaments most commonly affected are the external lateral of the ankle and the internal lateral of the knee. Localised pain, which may be sickening in severe cases, and tenderness over the site of the torn ligament, are immediate features. Extravasation of blood occurs in the neighbourhood of the torn ligament, and a sympathetic effusion occurs into the associated joint.

Treatment consists in the immediate application of a cold compress, followed by a pressure bandage made of one or two layers of wool and flannel bandage. The bandage must be applied firmly so as to limit further effusion. After two or three days graduated movements are instituted, and subsequently care is taken to relax the damaged ligament, e.g. the foot raised on its inner side in order to relieve strain on a torn internal lateral ligament of the knee joint.

DISLOCATIONS

Dislocations are either complete or partial (subluxation). Three types of dislocations are recognised : congenital, pathological, and traumatic.

Congenital dislocation most commonly occurs in the hip joint, and is considered in the chapter on Deformities.

Pathological dislocations are due to :

(1) *Destruction of the joint by disease*, e.g. 'travelling' acetabulum in advanced tuberculous arthritis of the hip joint, or subluxation of a tuberculous knee in cases of triple deformity.

(2) *Paralysis* of muscles which support a joint, as in the case of infantile paralysis of the shoulder girdle, or of the muscles around the hip joint. In spastic paralysis (Little's disease) the hips may dislocate as a result of persistent adductor spasm.

(3) *Neuropathic*, e.g. Charcot's joint. Softening of the ligaments predisposes to stretching, and pathological dislocation is liable to occur, even in the hip joint.

Traumatic dislocations occur most commonly in the middle part of adult life. In children separation of an epiphysis is more likely, while in older people atrophy of bone predisposes to fracture. Traumatic dislocations are always accompanied by tearing of the capsule and injury to surrounding tissues, especially to muscles and ligaments which are attached to adjacent bones. Nerves and blood-vessels are occasionally injured, e.g. the circumflex nerve following dislocation of the shoulder joint, or the sciatic in dislocation of the hip.

The likelihood of any individual joint suffering dislocation depends upon the shape of the articular surfaces, and the support given by muscles and ligaments. The shoulder joint is commonly dislocated, as the glenoid cavity is shallow, and the support given to the head of the bone by muscles and

ligaments is lax. Conversely, in the case of the hip, the acetabulum is deep, and muscles closely support the joint; therefore dislocation is uncommon.

Clinical Features.—(a) Pain—due to local trauma, or pressure on nerves, e.g. the head of the humerus may press on the brachial plexus.

(b) Loss of function.

(c) Deformity. The limb is shortened or lengthened, or malalignment is present.

(d) The end of the bone may be detected in an abnormal position. This is the *absolute* sign of a dislocation. Unless the dislocation is accompanied by a fracture, movement of the shaft of the bone causes corresponding movement of the articular end.

(e) Restricted mobility, as distinct from a fracture where abnormal mobility is present.

Treatment.—Reduction is obtained by manipulation, traction, or operation. Manipulation is carried out as soon as any attendant shock has subsided, without avoidable delay. In the case of large joints surrounded by powerful muscles, general anæsthesia is desirable in order to overcome muscular spasm. The path taken by the displaced bone should be visualised, and movements carried out so that this path is retraced without causing additional damage to soft tissues. From two to four weeks of partial immobilisation is advisable after reduction, in order to allow healing of the soft tissues; the larger joints require a longer period of rest than the small ones, e.g. interphalangeal. Operative measures are sometimes necessary, e.g. thumb (p. 273), and in the case of larger joints open reduction is required should manipulation fail.

Attempts at reduction by manipulation are seldom justified after a lapse of three or four weeks. If considered advisable, late cases are treated either by open reduction, with or without arthrodesis, or by excision of part of an implicated bone, or conversion into an arthroplasty.

DISLOCATIONS OF SPECIAL JOINTS

TEMPORO-MANDIBULAR JOINT

The usual cause of dislocation of the mandible is a blow on the chin when the mouth is partly open. Dental operations, particularly those performed under general anæsthesia, and excessive yawning are other causes. As in the case of the shoulder joint, recurrent dislocation is not uncommon.

If the dislocation is unilateral, the jaw is displaced towards the opposite side, and saliva dribbles from the partially open mouth. A hollow is palpable immediately in front of the tragus, and the condyle can be seen in a slightly anterior situation (fig. 318). In bilateral cases the mouth is fixed in a partly open position, and both condyles are displaced in front of their normal situations.



FIG. 318.—Dislocated jaw.

anterior situation (fig. 318). In bilateral cases the mouth is fixed in a partly open position, and both condyles are displaced in front of their normal situations.

Reduction can usually be performed with ease by pressing the padded humbs on the lower molar teeth, at the same time rotating the body of the arm upwards with the fingers. A general anæsthetic is occasionally necessary. After reduction a four-tailed bandage is worn for three weeks.

STERNO-CLAVICULAR JOINT

Violence affecting this joint is transmitted along the clavicle, but in the majority of cases fracture of the clavicle occurs before force sufficient to cause dislocation reaches the sterno-clavicular joint. Moreover, the sturdy rhomboid ligament anchors the inner end of the clavicle to the first costal cartilage. When dislocation occurs the inner end of the clavicle is displaced forwards and downwards, or backwards and upwards, the first of these being the more common. Backward dislocation may cause severe dyspnœa from pressure on the trachea, or congestion of the head or arm owing to obstruction to the great veins at the root of the neck. Owing to the subcutaneous position of the bone, the dislocation is readily recognised.

THE ACROMIO-CLAVICULAR JOINT

Dislocation of this joint is not uncommon as a result of a fall on the shoulder; owing to the obliquity of the articular surfaces, the outer end of the clavicle is forced upwards and over-rides the upper surface of the acromion. The conoid and trapezoid ligaments which anchor the clavicle to the coracoid process must of necessity be ruptured to permit this overriding. The prominence caused by the displaced outer end of the clavicle is readily palpable (fig. 319). The dislocation is easily reduced by elevation of the shoulder, but withdrawal of support results in immediate redislocation. Treatment consists in flexing the forearm, and applying strapping so that pressure is exerted on a pad situated over the outer end of the clavicle (fig. 320); the arm is then supported by a sling.

There are some who advocate immediate open reduction and internal fixation for complete recent dislocations. If an unreduced dislocation causes disability, resection of the outer end of the clavicle is sometimes advised, but it is rarely necessary as the patient can be encouraged by active exercise to generate a painless pseudarthrosis in the new position.



FIG. 320.—The application of pads and strapping for dislocation of the acromio-clavicular joint.



FIG. 319.—Dislocation of acromio-clavicular joint.

SHOULDER JOINT

Owing to the wide range of movement, the shallowness of the glenoid cavity, and the lack of support by ligaments and muscles, particularly on the inferior aspect, dislocations of this joint are of common occurrence, and are caused by sudden violence, not necessarily severe, to the joint with the arm in abduction. In the majority of cases a subcoracoid dislocation occurs.

On inspection of the shoulders an alteration of contour is obvious, unless the patient is very obese. The rounded appearance of the shoulder is lost owing to displacement inwards of the head of the humerus, and consequently

on inspection the 'point' of the shoulder appears more angular than normal (fig. 321). The axis of the arm passes upwards and inwards, and a fullness is noticed below the outer part of the clavicle. On *palpation* the angular shape of the shoulder is even more readily appreciated than on inspection, and loss of resistance is felt beneath the acromion process indicating absence of the humeral head from its normal position. Pain and limitation of movement are complained of by the patient. Injury to the circumflex nerve (p. 443), as indicated by paresis of the deltoid muscle and a patch of anæsthesia over its insertion, must be recorded before reduction is attempted.



FIG. 321.—Subcoracoid dislocation of the left shoulder.

The contour is of importance in diagnosing various lesions of the shoulder (fig. 322).

The following academic tests are rarely necessary. *Hamilton's Ruler Test*.—The acromion process and the external condyle can be connected by a straight line.

Dugas' Test.—Owing to the abduction of the lower end of the humerus it is impossible to place the hand of the patient on the opposite shoulder.

FIG. 322.—Contours of the shoulder.

- (1) Normal—rounded and mobile.
- (2) Dislocation—angular and fixed.
- (3) Fracture—rounded and fixed.
- (4) Paralysis of deltoid with wasting (circumflex palsy)—angular and mobile.

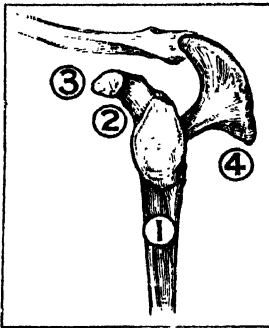
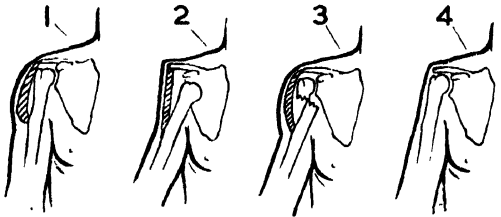


FIG. 323.—1. Subglenoid. 2. Subcoracoid. 3. Subclavicular. 4. Posterior or dorsal.

Varieties (fig. 323).—Dislocations of the shoulder, in the first instance while the arm is abducted, are usually subglenoid but the head of the humerus resting in a precarious manner on a narrow ridge of bone usually slips forward into the *subcoracoid* position. Should the dislocating force continue to act, the head of the bone moves farther inwards, and comes to rest in the subclavicular position. Occasionally the head of the bone is displaced backwards to give a 'dorsal' dislocation of the shoulder.

Luxatio erecta is a rare variety (under 1 per cent.), in which the head of the humerus is displaced into the subglenoid position, the arm being fixed in extreme abduction.

Treatment.—A general anæsthetic is usually advisable, particularly in a muscular subject, or when delay has allowed muscular spasm to supervene.

Kocher's Method.—This is a classical method, and was originally designed for use without anæsthesia. It is possible to do damage under anæsthesia if excessive force is

Frank Hastings Hamilton, 1813–1886. Professor of Clinical Surgery at Bellevue Hospital Medical College, U.S.A.
Louis Alexander Dugas, 1806–1884. Professor of Surgery, Medical College of Georgia, U.S.A.
Theodor Kocher, 1841–1917. Professor of Clinical Surgery, University of Berne.

sed. The patient may be either sitting or lying. The following manipulation is then performed in a smooth and deliberate manner according to the three stages of the illustration (fig. 324).

(1) The elbow is flexed and adducted and the arm externally rotated so as to stretch the subscapularis muscle, which has contracted owing to the inward displacement of the upper end of the humerus. This manipulation must be performed slowly and to its fullest extent.

(2) When full external rotation is obtained, the elbow is brought in front of the chest, at the same time exerting traction.

(3) The forearm is then rotated inwards so that the fingers sweep across to lie on the opposite shoulder if reduction has been successful. Usually reduction will be detected by a 'click' during this last movement.

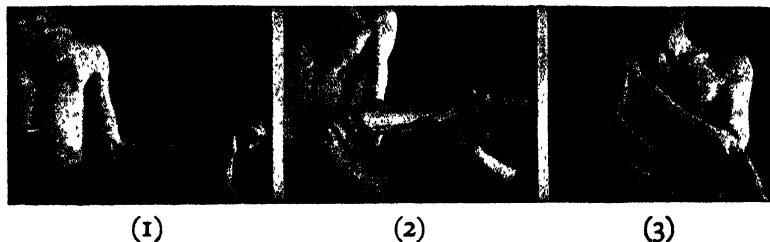


FIG. 324.—Kocher's method for reduction of a dislocated shoulder.

(F. P. Fitzgerald, F.R.C.S.I., London.)

Traction Method (Hippocratic).—Traction is applied by placing the unbooted foot against the chest wall in the axilla with the patient lying supine. A steady pull is maintained on the forearm in an outward and downward direction. If reduction does not result by the time maximum traction has been applied, it will usually be produced by internal or external rotation of the arm while traction is maintained. Quite often reduction occurs long before maximum traction has been applied and it sometimes can be obtained by traction under the pre-operative drugs before anaesthesia is started. The possibility of damage to axillary structures by the pressure of the operator's foot is without foundation.

After-treatment consists in supporting the arm in a sling in a slightly abducted position which is conveniently maintained by an 'axillary muff'. If the arm is kept in an adducted position, e.g. bandaged to the chest wall, the loose inferior portion of the capsule is thrown into folds which become adherent to each other, and thus return of full abduction is hindered. Finger and wrist movements are encouraged immediately, and active movements of the shoulder are commenced a few days later if the patient is elderly and therefore likely to get a very stiff shoulder. In young people abduction to more than a right angle should be prohibited for one month, as this may precipitate recurrent dislocation.

Operation.—In cases which have been dislocated for several weeks manipulative measures fail and open reduction may be necessary. In old people even this may not be necessary if they have recovered from the discomfort of the original injury, and quite good function is possible with the fibrous ankylosis in the dislocated position.

Complications

FRACTURE.—Dislocation of the shoulder may be complicated by fracture of the surgical neck of the humerus. Not uncommonly the great tuberosity is avulsed, but apposition occurs when the dislocation is reduced (fig. 325).

RECURRENT DISLOCATION.—*Vide infra.*

Hippocrates, by common consent, the Father of Medicine, was born in the island of Cos in the Aegean archipelago about B.C. 300. He lived to be 109 years of age in an era when the expectation of life was about 32 years.

NERVES.—Any part of the brachial plexus, or adjacent nerves, may be involved. Owing to its limited mobility and proximity to the head of the humerus, the circumflex



FIG. 325.—Subcoracoid dislocation of the shoulder with avulsion of the greater tuberosity. Good apposition after reduction. (*J. Hindenach, F.R.C.S., London.*)

nerve (p. 443) is most commonly injured (10 per cent. of cases), but recovery usually ensues in from two to six months.

MUSCLES.—The tendon of the supraspinatus muscle is occasionally ruptured, as is evinced by inability to abduct the arm. If suspected, the tendon should be explored by operation, as suture is only possible if the injury is a recent one.

VESSELS.—Damage to vessels is rare, but complete rupture of the brachial artery is occasionally reported.

RECURRENT DISLOCATION

In cases in which after-treatment has been inadequate, or in persons who are subjected to frequent injury, e.g. epileptics, weakness of the capsule persists, and it may even happen that the patient is able to dislocate the joint voluntarily. If disability occurs, Bankart's operation gives consistently good results, but there are many modifications. Bankart postulates that recurrent dislocation is due to separation of the anterior part of the glenoid fibro-cartilage. The joint is exposed, and the coracoid process is divided and displaced downwards with the attached muscles. The subscapularis muscle and capsule are divided and the joint is thus opened. Sutures are inserted so that the detached labrum is firmly stitched to the capsule of the joint. Further stability is obtained by double-breasting the capsule, and the subscapularis is then repaired. The coracoid process is replaced and held in position by sutures inserted into the adjacent soft tissues. External rotation is not permitted for at least a month, after which active exercises are encouraged. It is probable that permanent restriction of external rotation is the basic cause for the success of operations for this condition, and on the strength of this observation more and more frequently the simpler operation of 'reefing' the tendon of the subscapularis (Putti-Platt) is now used in preference to the more complicated Bankart procedure.

FRACTURE—DISLOCATION OF THE SHOULDER

This serious accident is due to continuation of force after the shoulder has been dislocated, the fracture occurring through the surgical neck of the humerus. The condition is difficult to recognise except by X-ray. Crepitus is obtained on manipulation, and more pain and extravasation are present than in cases of dislocation only.

Reduction by manipulation is unlikely to be successful, owing to the small size of the upper fragment and consequent difficulty in controlling it.

In a young patient open reduction is therefore usually necessary, the bone being exposed by an incision which separates the deltoid and pectoralis major muscles. Attachments of soft tissues to the head of the bone must be carefully preserved, otherwise avascular necrosis may follow. The operation is not commonly needed because most of these cases occur in elderly patients, and provided that there is no pressure on neurovascular structures, early movement will eventually give adequate fibrous ankylosis.

ELBOW JOINT

In dislocation of the elbow the forearm is most commonly displaced posterolaterally in relation to the humerus (fig. 326). Forward dislocation is usually accompanied by fracture of the olecranon.

Dislocation of the elbow is distinguished from supracondylar fracture of the humerus by the fact that the normal relations of the bony points around the elbow are distorted (fig. 327). Also on inspection the forearm is apparently shortened, and the measurement between the external epicondyle and radial styloid process confirms this shortening.

Reduction is effected by flexing the arm to a right angle and applying traction to the forearm. The reduction takes place with a loud snap and is immediately stable unless the elbow is fully extended. The joint is kept at rest in a sling for three weeks.



FIG. 326.—Posterior dislocation of elbow.

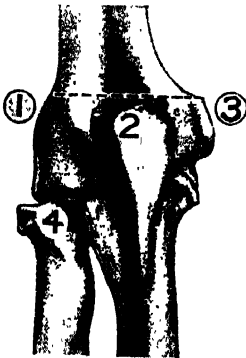


FIG. 327.—1. External condyle. 2. Tip of olecranon process. 3. Internal condyle. 4. Head of radius.

Myositis ossificans is a well-known complication affecting the brachialis anterior muscle after dislocation of the elbow joint (p. 317). When recognised, it is of the utmost importance to limit use of the elbow, and to make sure that the patient is not applying passive stretching movements in order to increase the range of movement.

Anterior dislocations are associated with fracture of the olecranon process. Reduction is obtained by traction with the arm extended, and the olecranon process must then be exposed by open operation and mechanically fixed in position by a screw or wire suture.

INCARCERATION OF THE MEDIAL EPICONDYLE

In children the epiphysis of the medial epicondyle is frequently avulsed as part of the injury of a dislocated elbow, but in rare cases the separated epicondyle becomes trapped inside the joint when it is reduced. Clinically this complication should be suspected if there is anæsthesia or paræsthesia in the distribution of the ulnar nerve in the hand. X-ray will show the epicondyle in the joint, but it is easily overlooked if not carefully searched for.

When recent, the epicondyle is usually easily reduced by manipulation, but a late case may need open operation and excision.

PERILUNATE DISLOCATION OF THE CARPUS

The commonest element in dislocations of the carpus is dislocation of the lunate. When this occurs the lunate is extruded through the anterior capsule of the wrist joint and lies in the carpal tunnel, causing median nerve pressure in this confined space.

The injury occurs in falls from heights on to the outstretched hand. It is important not to miss the injury because when seen in the early stages it can usually be

reduced with the greatest ease by simple traction and hyperextension combined with pressure on the dislocated bone.

The condition is most likely to be missed if associated with a fracture of the radial styloid or a fracture of the carpal scaphoid, because then the inexperienced surgeon may look no further in the radiograph. A well-centred lateral X-ray clearly reveals the dislocated semilunar in front of the carpus, but a poor lateral view may conceal the dislocation. The antero-posterior view at first sight may be passed as normal, but close inspection will show that the bones of the proximal row of the carpus (scaphoid, lunate, and triquetral) are not disposed round the head of the



FIG. 328.—Midcarpal dislocation. Sometimes can be missed in antero-posterior view if not scrutinised carefully.

capitate (os multangular major) with the intervention of a regular joint space as in the normal wrist (fig. 328).

Dislocation of the lunate is always part of a more serious injury which is that of a midcarpal dislocation which has reduced itself, and therefore will not be visible radiologically. In old unreduced cases the lunate must be excised, but if of not more than a few weeks' duration an open reduction is often worthwhile.

Dislocation of the head of the radius occasionally occurs as a congenital abnormality (p. 330). In cases due to trauma the head of the bone

usually passes forwards and hinders flexion of the joint.

Fracture of the shaft of the ulna (Monteggia's fracture) is commonly associated with forward dislocation of the head of the radius, and can be readily recognised by palpation of its subcutaneous border (fig. 329). Traction of the forearm combined with pressure on the radial head usually permits of re-



FIG. 329.—The 'Monteggia' fracture. Combination of dislocation of the head of the radius with fracture of the upper one-third of the ulna.

duction, but as the orbicular ligament is torn the dislocation tends to recur. In children the head of the radius can usually be retained in position in the fully flexed position of the elbow. In adults the key to the reduction is provided by internal fixation of the fractured ulna. Once this has been done, it is usually possible to hold the head of the radius in position by plaster after manipulation.

Metacarpo-phalangeal and **inter-phalangeal** dislocations can be reduced easily by traction and flexion, with the exception of the metacarpo-

phalangeal joint of the thumb. Traction should be applied, and a bandage, placed as a clove-hitch round the thumb, may assist in securing a firm grip. Manipulation frequently fails, the commonest cause of failure being the interposition of the glenoid (anterior) ligament between the two bones. This tough ligament, firmly attached to the base of the phalanx, is carried backwards, and lies like a curtain between the phalanx and the head of the metacarpal (fig. 330 (A)).

Other causes of failure in reduction are buttonholing of the two slips of the flexor pollicis brevis (fig. 330 (B)), and interposition of the long flexor tendon (fig. 330 (C)). If a satisfactory manipulative reduction of this joint cannot be achieved, it should be explored by operation without delay.

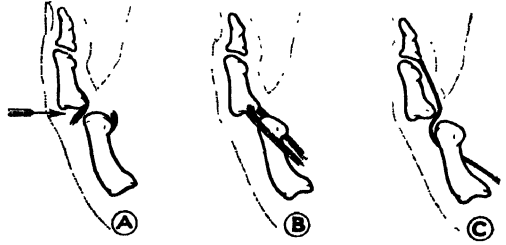


FIG. 330.—Causes which prevent reduction of a dislocation of the metacarpo-phalangeal joint of the thumb.

HIP

Owing to the depth of the acetabular cavity and the strong support afforded by ligaments and muscles, traumatic dislocation of this joint is relatively uncommon.

Dislocation of the hip most commonly occurs when the hip is flexed, and especially so if it is also adducted, because in this position the head of the bone is in contact with the relatively weak under-surface of the capsule. The usual causes are a weight falling on the back of a person in a stooping position, as in a coal-miner struck by a 'fall of roof', and, most common of all, in car and motor-cycle accidents (fig. 331).



FIG. 331.—Traumatic 'dorsal' dislocation of the hip. There is also a fracture of the cotyloid labrum.

Posterior dislocations are the common types of hip dislocation. The head of the bone escapes into the sciatic notch (sciatic variety), and then passes up on to the dorsum of the ilium (dorsal variety). In both cases the leg is flexed, adducted, and inverted, so that the sole rests upon the opposite instep. Pain is sometimes referred along the sciatic nerve, which may be involved by direct injury. X-rays should always be taken to confirm the dislocation and detect any associated fracture.

Reduction is usually accomplished without difficulty if the injury is recent, and provided that the anæsthetist obtains adequate relaxation of the muscles.

The patient is placed supine on a mattress on the floor, and the iliac crests are steadied by an assistant. The surgeon stands over the limb and flexes the knee and thigh, bringing the head of the bone beneath the acetabulum. The femur is then pulled vertically upwards so as to draw the head forward from its posterior position. The essence of the reduction lies in the vertical lifting of the femur with the maximum force the surgeon can exert. Sometimes reduction will occur with this vertical traction alone. Otherwise the hip must be internally and then externally rotated while maximum upward traction is being exerted before the head of the femur suddenly snaps into its socket.

Usually the reduction is quite obvious when it occurs, and if the acetabulum is intact the reduction is stable. If a large fragment of the posterior margin of the acetabulum has been fractured, the hip will easily re-dislocate when upward traction is released, but it will usually remain stable if the hip is extended before releasing traction.

Following reduction, a plaster spica should be applied to below the knee for two months, or longer if the rim of the acetabulum is fractured. In about 10 per cent. of cases avascular necrosis of the femoral head is inevitable, due to damage to the ligamentum teres and the anastomosis nourishing the head, and osteoarthritis is a common sequela.

Complicating dislocations of the hips are fractures of the posterior lip of the acetabulum. Usually they reduce themselves when the hip is reduced, but in a few cases, where the fragment of the acetabulum is large, it may be necessary to fix it by open operation and a screw.

Anterior dislocation of the hip is exceedingly rare and can be either obturator or pubic (fig. 332); in both cases the limb is in a position of flexion, abduction, and eversion.

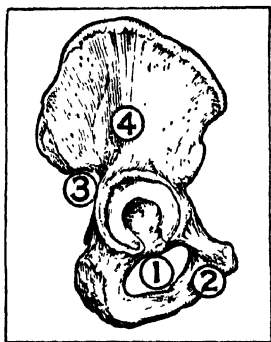


FIG. 332.—1. Obturator.
2. Pubic. 3. Sciatic. 4.
Dorsal.

Central dislocation of the hip occurs as a form of fracture of the pelvis when a blow is delivered to the great trochanter driving the head of the femur through the floor of the acetabulum. This injury is really a comminuted fracture of the acetabulum and often is accompanied by other elements of fracture of the pelvis.

Treatment is difficult and unsatisfactory. The femoral head can often be pulled out to its anatomical position by a combination of traction on the lower extremity and a side pull to a wire or nail in the great trochanter. Only rarely does the weight-bearing part of the acetabulum reduce with the head of the femur and, even if it does, the floor of the acetabulum rarely accompanies the reduction. A good initial reduction can be held for a month or two, but on removing traction the position deteriorates slightly, but enough to guarantee the commencement of arthritis some years later. Operative treatment is difficult and demands great experience and skill.

KNEE

Complete dislocation of this joint is rare, but subluxation is common, following rupture of one or other cruciate ligament, or both. The diagnosis is usually obvious, although rapid effusion into the knee joint tends to render

immediate recognition difficult. The most common direction of dislocation is for the femur to pass forward, and the popliteal vessels are occasionally compressed (p. 130).

Reduction is effected by flexion and traction, and aspiration of the distended knee joint is often advisable. The joint should be immobilised in plaster in slight flexion for three months. Quadriceps drill is instituted to begin with, and weight-bearing is permitted after about a month. Instability sometimes persists owing to rupture of the cruciate ligaments (*vide* internal derangement).

PATELLA

This sesamoid bone may be dislocated by direct violence and by far the most common direction is outwards. The diagnosis is readily made on palpation. Reduction is effected by laying the patient on his back with the leg and thigh extended. The quadriceps muscle is thus relaxed, and the bone can be manipulated into position.

Recurrent dislocation of the patella is not uncommon, especially in girls or young women. Sometimes it is associated with a marked genu valgum which increases the angle between the direction of pull of the patella tendon and the quadriceps tendon and so disposes to lateral movement of the patella. Most commonly recurrent dislocation occurs without genu valgum and is almost certainly due to a lack of development of the lateral femoral condyle. Operative treatment is usually successful, and most commonly the tibial tubercle, with its inserted patella tendon, is transplanted medially, and slightly distally, to prevent the lateral movement. In severe cases excision of the patella has been successfully employed.

INTERNAL DERANGEMENT OF THE KNEE JOINT

This term is used to describe incidents of a mechanical nature in a previously normal knee joint. The 'internal' nature of the derangement is suggested if the mechanical incident is followed by synovial effusion.

Two special conditions exist: (1) tears of the cruciate ligaments and (2) tears of the semilunar cartilages.

(1) **Tears of the cruciate ligaments** are always the result of severe injuries, and frequently at sport. They are the result of a subluxation of the knee, and the lateral ligaments are of necessity ruptured at the same time. The injury is followed immediately by a hæmarthrosis and the joint is usually grossly swollen in a few minutes, the patient being unable to walk on the limb.

Careful testing, especially under anæsthesia, will show abnormal mobility of the joint—the tibia sliding forward on the femur and the knee hyper-extending if the anterior cruciate ligament is torn, and the tibia sliding backwards if the posterior cruciate is damaged.

Provided that the serious nature of the condition is recognised the ligaments usually unite if protected by a plaster cylinder in slight flexion for three months. Thereafter strong quadriceps exercises and physiotherapy to recover flexion will be needed for about six months.

In some cases the patient has a permanently unstable knee which "lets him down" and attempts have been made to repair the ligaments by various operations but with doubtful success. Many of the patients with this complication are compensation cases with no incentive to recovery. In this respect the excellence of recovery in athletes is very significant. The

complete recovery of quadriceps power, which is always in the patient's capability, is the key to full recovery of function in knee-joint surgery.

For the same reason the use of a knee-cage in permanent laxity of the knee should not be encouraged as it perpetuates any hysterical tendency.

(2) **Cartilage.**—Injuries of the semilunar cartilages are very common as the result of accidents at sport, and particularly in football. The medial meniscus is torn perhaps twenty times more frequently than the lateral, also it is often torn in miners working in a kneeling position at the coal-face.

The exact mechanism whereby the cartilage is torn is still not completely explained, but certain facts are quite clear. The tear occurs most commonly as a result of rotation on a flexed knee which is *taking the weight of the body*. In the case of the right knee, the medial meniscus is damaged when the player is turning his body, and his femur, towards the left. This movement abducts the tibia and the medial meniscus is presumably drawn into the joint and pinched between the medial femoral condyle and the medial tuberosity of the tibia. In this position it splits along its length so that the free inner rim is separated from the periphery which is attached to the joint capsule. The lateral meniscus is torn less commonly than the medial because it is more mobile and not so adherent at its periphery to the capsule. Once the longitudinal split occurred, it may extend to one of the three main varieties: (a) anterior pedunculated tear, (b) posterior pedunculated tear, and (c) the complete, 'bucket-handle' tear, which is by far the most common lesion (fig. 333).

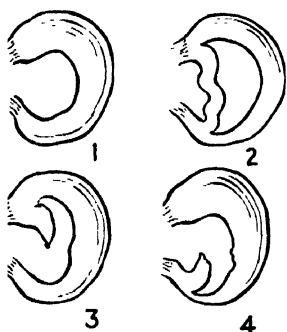


FIG. 333.—Tears of media meniscus.

- (1) Normal.
- (2) 'Bucket-handle.'
- (3) Posterior.
- (4) Anterior.

Diagnosis.—A careful history is of paramount importance, and very few patients will forget the dramatic circumstances of the first injury. Frequently this will disable them for two or three weeks, and will invariably be accompanied by a large effusion. In a few cases there will be no history of injury, but here it is not uncommon to find that the tear occurred when kneeling. In these cases it seems that the meniscus can assume an abnormal position when kneeling, and if the patient suddenly stands up the meniscus fails to get back into position quickly enough and therefore sustains a tear.

If seen shortly after the first injury, the diagnosis will be made on the history, the presence of effusion, the inability to extend the knee fully (though flexion may be possible to some extent), and by localised tenderness over the joint line on the inner aspect of the knee. In such a case the knee is 'locked' because the displaced fragment is obstructing extension, and unless the patient accidentally releases the obstruction it will be necessary to 'unlock' the knee.

Of all the signs and symptoms in making a diagnosis of cartilage injury, the most important *sign* is marked local tenderness over the affected meniscus and the most important *symptom* is a volunteered statement of the ability

to unlock the knee by various wriggling manœuvres which the patient has learned by experience. 'Locking' of the knee is a very unreliable symptom of cartilage injury but, on the other hand, sudden spontaneous 'unlocking' is almost positive proof of a cartilage tear. Patients sometimes think, if the surgeon asks whether the knee locks, that locking means the loss of all movement (in neither flexion nor extension being possible). In fact flexion is possible, but full extension is not possible; this may cause the patient to say the knee does *not* lock when in fact it does.

The significance of accurate localisation of tenderness is indicated in fig. 334.

An X-ray should be taken as a routine to exclude a loose body (p. 301) or Paget's quiet necrosis (p. 302).

Manipulation of the Locked Knee.—Many complicated manœuvres have been described by which the displaced fragment can be made to return to the position from which it came, but in fact the essential feature is effective relaxation of the muscular spasm under full anaesthesia. Frequently the displaced fragment 'goes back' at the first movement of the knee as soon as full anaesthesia has been achieved. If this does not happen, the surgeon manipulates the knee in an endeavour to open up the medial joint line and give an opportunity for the displaced fragment to slip out from the centre of the joint. If a satisfactory 'snap' is not achieved and full extension does not immediately result, it is probable that the tear is irreducible by non-operative means and operative treatment may later be necessary.

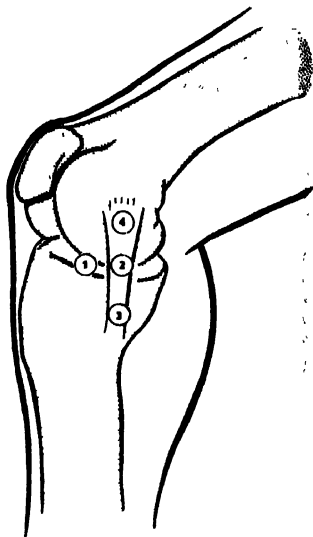


FIG. 334.—Sites of localised tenderness in knee injuries.

(1) and (2) on joint line. Tears of medial meniscus.

(3) and (4). Sprains at tibial and femoral attachments of medial collateral ligament.

When deciding to advise operation the diagnosis should be established beyond all question.

The poor results of cartilage surgery are those in which, at operation, to the surgeon's surprise, the suspected cartilage proves to be normal. Until the diagnosis is proved there is no hurry in advising surgery and every new episode will help to clinch the final diagnosis. There is no place for 'exploratory arthrotomy' in suspected cartilage injuries, because experience has shown that the results of a negative exploration are disappointing. Probably the most common error in diagnosis is to mistake the rather vague symptoms of chondromalacia of the under-surface of the patella for a cartilage tear.

Once a cartilage has been torn there is no likelihood of it healing, even if the reduced fragment lies in contact with the rim from which it was originally torn; this is because the fibro-cartilage of which it is composed is avascular and incapable of healing. The decision whether or not to operate to remove the torn fragment depends on the amount of trouble the injury causes in subsequent months. Even a large 'bucket-handle' tear lying in the intercondyloid notch, which manipulation has failed to reduce, will eventually adapt itself to its new site and may cause no trouble. On the other hand,

the patient may recover from the first injury and thereafter have repeated episodes of locking from minor violence.

It is customary, and proper, to insist on the most absolute aseptic precautions possible during this operation. Careful skin preparations and 'no-touch' technique in the theatre are a *sine qua non*. An Esmarch's rubber bandage or a sphygmomanometer is applied as a tourniquet. The internal cartilage is exposed through either an oblique or transverse incision on the inner side of the joint. The skin knife is then discarded and skin edges draped. The torn cartilage is steadied by a blunt hook and the anterior attachment to the head of the tibia is divided. The cartilage is then grasped by forceps and pulled forward till the posterior attachment can be reached and divided. If the common 'bucket-handle' tear is present, it is necessary only to remove the detached fragment. The synovial membrane, capsule, and skin are sutured, and a firm bandage applied before the tourniquet is removed, so as to limit bleeding into the joint. The limb is steadied between sandbags, and quadriceps drill is commenced on the following day.



FIG. 335.—Cyst of the right external semilunar cartilage.

CYST OF SEMILUNAR CARTILAGE

A cyst of a semilunar cartilage is a myxomatous degeneration occurring in the substance of the cartilage itself. The external cartilage is affected perhaps twenty times more commonly than the internal, and a tense swelling appears over the interarticular groove (fig. 335). The cyst sometimes appears suddenly owing to extrusion from the joint. The cartilage, together with the cyst, should be removed if symptoms are persistent, but, like a 'ganglion' in any other site, if desired it can be ignored and will eventually disappear spontaneously after a number of years. Pain in these cases invariably follows exertion, and rest relieves the tension which is the cause of the pain. Local removal is un-

satisfactory, as recurrence is inevitable; if surgical intervention is necessary, complete meniscectomy must be performed.

ANKLE

Owing to the deep mortise formed by the tibia and fibula, dislocation of the ankle joint, without fracture, is extremely rare. Dislocations of the talus are more common than formerly, owing to accidents associated with high-velocity transport and aeroplane crashes.

Subastragaloid dislocation occasionally occurs as a result of severe twists or wrenches, e.g. the patient being dragged by a horse with a foot in the stirrup. If manipulation fails, open reduction is necessary.

Johann Friedrich August von Esmarch, 1823-1908. Professor of Surgery, Kiel, Germany.

CHAPTER 14

DISEASES OF JOINTS

JOHN CHARNLEY

TRAUMATIC SYNOVITIS

THE term 'traumatic synovitis' is accepted by long use, though scientifically it is open to criticism. The term is valuable in indicating that the initial reaction of a joint to injury produces local physical signs which are almost indistinguishable from those of an early infection.

Traumatic synovitis is caused by such episodes as aseptic penetrating wounds (including operations), direct blows, sprains, or nipping of a loose body or cartilage. An effusion follows, either serous or blood-stained, according to the severity of the lesion. The joint assumes a position of ease, i.e. in which the synovial cavity has the greatest capacity, and palpation or movement is painful and local heat may be present.

Treatment consists, in the early stages, in rest, combined with cold lotions and firm bandaging, in order to minimise further effusion. Plaster fixation is unnecessary, and not advisable, because plaster cannot give continuous gentle pressure as the effusion absorbs. If effusion is excessive, aspiration is sometimes advisable to relieve pain, and the time necessary for absorption is shortened. When the early symptoms have abated, muscle drill and subsequently active movements are employed in order to prevent adhesions and to maintain muscular tone.

ACUTE SUPPURATIVE ARTHRITIS

Acute infection of a joint occurs as a result of :

- (i) Direct infection, as by a penetrating wound or a compound fracture which involves the joint.
- (ii) Local extension, from some neighbouring focus, such as acute arthritis of the hip joint from osteomyelitis of the femoral neck.
- (iii) Blood-borne infection, the usual organisms being streptococcus, staphylococcus, and pneumococcus, and less commonly the gonococcus and *B. typhosus*.

The knee joint, owing to its large size and exposed position, is the commonest joint to be involved by penetrating wounds, while suppurative arthritis from blood-borne infections is more likely to be the cause in other joints.

Clinical Features.—*General.*—The systemic effects of a suppurative arthritis are usually severe. Occasionally the condition is a complication of a septicæmia ('septicæmic joints'), and in this case the severity of the toxæmia can easily be explained.

Local.—Pain, especially on attempted movement. The joint is held in the position of greatest capacity, and swelling is usually evident. Palpation reveals increased heat and tenderness. Movements are limited by muscular spasm, and attempts at either active or passive movement cause severe pain.

It will be seen that in most cases the position of ease differs widely from

the position which is most useful should ankylosis occur. As any case of arthritis may be followed by ankylosis, the first duty of the surgeon is to anticipate this possibility by immobilising the joint in the best position for ankylosis, as indicated in the following table.

Joint.	Position of ease.	Site of maximum swelling.	Position for ankylosis.
Shoulder .	Adducted	Under the deltoid, along the tendon of the biceps, and in the axilla	40° to 50° of abduction, with elbow joint just anterior to the coronal plane and hand in front of the mouth.
Elbow .	Flexed at a right angle and pronated	On either side of the triceps tendon	90° of extension semi-pronated. If both sides, one elbow at 75° of extension, the other at 135° ¹ . These positions may be modified according to occupation.
Wrist .	Slight flexion . . .	Under extensor and flexor tendons	Dorsi-flexed to allow a firm grasp.
Hip .	Flexed, abducted, and everted	Upper part of Scarpa's triangle	20° to 30° of flexion to allow sitting, and in neutral position as regards abduction.
Knee .	Flexed	Suprapatellar bursa, and either side of patellar tendon	5° to 10° of flexion to allow foot to clear ground in walking.
Ankle .	Slightly extended and inverted	Anteriorly and on either side of the Achilles tendon	At the right angle, with slight inversion to discourage flat-foot.

Treatment.—Antibiotics are prescribed, and have greatly reduced the incidence and severity of joint infections, as a result of both prophylactic and therapeutic administration. In the early stages the limb is supported and fixed by a suitable splint or appliance in the correct position, an anæsthetic being administered if necessary. Weight-traction, which was commonly used in the past with the idea of holding joint surfaces apart and preventing damage to the inflamed cartilage, is no longer much favoured. The following lines of treatment are then to be considered.

(i) *Aspiration*, which is useful for both diagnostic and therapeutic reasons. Thus the nature of the fluid can be ascertained, and smears or cultures assist in identifying the causative organism and its resistance to antibiotics, so that suitable chemotherapy can be instituted. Aspiration also reduces the tension within the joint, thereby relieving pain, and limiting the stretching of ligaments and capsule.

(ii) *Aspiration and Injection*.—After fluid has been aspirated, antibiotics may be injected into the joint, but repeated injections into a joint are probably unnecessary. Antibiotics are administered parenterally, and the limb is suitably splinted.

(iii) *Arthrotomy and Drainage*.—In neglected cases with radiological

¹ Thus the patient is enabled to reach the most important orifices of the body.

destruction of bone the joint is opened, washed out, and drainage tubes inserted down to the synovial membrane. This procedure should only be considered if bone destruction has involved the articular surfaces and when some degree of ankylosis is all that can be expected when healing has occurred. Though commonly used during the early part of the Second World War, this technique is today but rarely needed, due to the effect of antibiotics. The knee joint is drained through an antero-lateral incision, combined, if necessary, with a postero-lateral incision which passes behind the ilio-tibial band and in front of the biceps tendon. In all cases adequate immobilisation is essential.

Extra-articular abscesses sometimes require to be opened and drained. In the case of the knee joint, pus is particularly liable to track upwards beneath the quadriceps, where its presence may be overlooked.

(iv) *Excision*.—If the condition of the patient deteriorates in spite of treatment, or if suppuration is prolonged, then drastic surgical ablation of the diseased bone is necessary. In the knee or ankle joint the solution is simply that of amputation, and an excellent functional result can be expected with an artificial limb, and moreover convalescence is rapid. In certain cases, such as the elbow, wrist, or shoulder, being non-weight-bearing joints, an excision of the joint can be life-saving and the resultant flail limb is still useful to the patient. Even in the hip joint, excision is a valuable procedure (Girdlestone's operation) and, despite the unstable pseudarthrosis which results, the patient can walk on a weight-bearing caliper provided with a 'bucket-top'. Needless to say, a patient's life must never be jeopardised in an endeavour to spare a limb, particularly as the affected joint will eventually be more or less disorganised.

(v) *Amputation* is indicated if at any stage the patient's life is threatened, or prolonged suppuration leads to amyloid disease (which is particularly liable to occur in connection with prolonged infection of the hip joint (fig. 33)). Also, in some cases a painful and disorganised joint causes such disability that an artificial limb is preferable.

PENETRATING WOUNDS OF JOINTS

Owing to its size and exposed position, the knee joint is the joint most commonly affected by penetrating wounds. For the purposes of treatment such wounds can be classified according to the risk of subsequent infection.

(i) *Infection is unlikely*, e.g. recent puncture by a small and comparatively clean article, such as a needle. In these cases the needle is removed and the wound is sterilised with an antiseptic. The limb is immobilised in the position of rest and antibiotics administered. Usually a mild, transitory effusion occurs, which subsides in two or three days. Should general and local symptoms or signs suggest that infection is occurring, then the joint is aspirated, and treatment continued along the lines already indicated for acute arthritis.

(ii) *Infection is probable*, such as penetration by a rusty nail, particularly if delay has already permitted infection. In these cases emergency operation

must be performed, and the wound excised layer by layer until the joint is opened. Irrigation is advisable if obvious contamination has occurred, as by fragments of rust. The wound is partially or completely closed according to the risk of infection (the synovium is always closed), and the limb is immobilised in the most useful position should ankylosis follow. A prophylactic course of penicillin is prescribed.

(iii) *Infection is certain*, e.g. extensive laceration of the capsule, or less severe wounds which have been neglected. Recent extensive wounds, which formerly would inevitably have become infected, often do remarkably well following thorough debridement, insufflation of penicillin powder, encasement in plaster, and chemotherapy. If infection supervenes or is already established, the wound is rendered as surgically clean as possible, and drainage is provided if necessary. The limb is immobilised, and further treatment conducted on the lines indicated for acute arthritis.

GONOCOCCAL ARTHRITIS

Owing to more efficient treatment of gonococcal urethritis and conjunctivitis, articular complications are now uncommon. Joint lesions occur at any stage after infection, but are most common when the initial infection is subsiding.

(i) **Acute Arthritis.**—Usually a single large joint is affected, especially the ankle, elbow, or wrist, the last particularly in females. The symptoms and signs of acute infective arthritis are present, and destruction is sometimes sufficiently extensive to cause bony ankylosis. Characteristically the patient never looks as ill as would be expected if the local pain and heat were caused by a suppurative arthritis of staphylococcal or streptococcal origin. On aspiration the fluid may be found to contain gonococci, but in less acute or later cases it is sometimes sterile. Treatment is conducted on the lines already indicated.

(ii) **Chronic arthritis**, in which there is but slight effusion in the joints. Oedema of the synovial membrane and periarticular structures occurs. Troublesome adhesions and stiffness are to be expected.

Treatment.—Limitation of movement arising from fibrosis of extra-articular structures, or fibrous and sometimes even bony ankylosis, are characteristic features of gonococcal affections. A course of chemotherapy or antibiotics is prescribed, and as soon as the acute phase of inflammation has abated judicious physiotherapy and active movements must be instituted.

SYPHILITIC DISEASES OF JOINTS

Inherited.—*Painless Effusion.*—This has been aptly described as ‘symmetrical, silent, serous, syphilitic synovitis’, and is associated with the name of Clutton. The knee joint is most commonly affected and the condition is frequently bilateral, although the swelling of the two joints may not synchronise. The effusion causes a sensation of weakness and insecurity, and on examination the joint is seen and felt to be distended. Movements are only limited if the amount of fluid mechanically prevents the full range.

The condition occurs between the ages of ten and eighteen, and other stigmata of inherited syphilis are usually, but not necessarily, present. This condition is one of the four characteristic signs of inherited syphilis, which appear about puberty, and give rise to ‘the halt, the deaf, the blind, and the impotent’, i.e. halt owing to Clutton’s joints, deaf because of otitis interna, blind following interstitial keratitis, and impotent secondary to orchitis.

If no confirmatory clinical evidence of syphilis is discovered, then the family history is usually suggestive, and finally the W.R. of the patient and parents, or even of the fluid aspirated from the joint, should be tested.

Acquired.—During the *secondary* stage a transient or variable effusion sometimes occurs in the larger joints. The condition is painless and, like most secondary lesions, often symmetrical. Lesions of the *tertiary* stage are rare. Gummatous synovitis in

ome respects simulates tuberculous disease, but the condition is painless, and therefore muscular spasm and wasting are not pronounced.

NEUROPATHIC JOINTS

The most important pathological conditions of joints secondary to affections of the nervous system occur in connection with :

1. Parasyphilis. 2. Syringomyelia. 3. Other lesions of the Nervous System.

1. **Parasyphilis.**—Owing to the more efficient treatment of syphilis, Charcot's joints now occur with diminishing frequency. About 4 per cent. of tabetic patients develop an arthropathy, of which 85 per cent. occur in the lower limbs, the knee being the commonest joint to be affected. Two quite distinct types of neuropathic joint are distinguishable radiologically; the 'hypertrophic' (usually hinge joints,) shows large osteophytes, bone sclerosis, and all evidence of new bone production; the 'atrophic' type (commonly ball and socket joints), merely shows osteoporosis and the resorption of the articular ends of the bones forming the joint. In either case, an effusion, which varies in amount, is present, and complete *absence of pain* is a striking feature. Examination of a typical case, following the routine which should be adopted for all joints, comprises:

(i) *Inspection.*—The joint is distended, and if the effusion is generous, it assumes the position of greatest capacity. In advanced cases the joint is obviously disorganised (fig. 336).

(ii) *Palpation.*—The presence of fluid is confirmed, and in hypertrophic cases irregular masses of bone are readily palpable. Bursæ which communicate with the joint, such as the psoas or semimembranosus bursa, are sometimes distended.

(iii) *Movements.*—Soft crepitus is usually noticed, and the laxity of the capsule and ligaments allows an abnormal range of movement. The patient will often astonish the inexperienced observer by the way he can walk without pain on a joint which is completely disorganised and threatening to subluxate.

(iv) *Measurement.*—Shortening of the limb may occur owing to absorption of bone, and some degree of muscular wasting may result from disuse.

(v) *Radiograph.*—Irregular masses of bone are seen in the hypertrophic variety (fig. 337).



FIG. 336.—Charcot's disease of the right knee. (Dr. Worster-Drought, London)



FIG. 337.—Charcot's knee joint. Hypertrophic type.

The atrophic type will show irregular and eventually almost complete absorption of the articular ends of the affected bones.

(vi) *General Examination*.—A Charcot's joint can present itself as the first cause of a tabetic patient seeking treatment. Lightning pains, Romberg's sign, ataxia, or other symptoms are usually present, while examination commonly reveals Argyll-Robertson pupils and loss of tendon reflexes, particularly the knee and ankle jerks. Deep pain sense is lost, as can be tested by squeezing the tendo Achillis.

Treatment consists in supporting the joint by a suitable appliance, e.g. a knee brace or a walking caliper. Arthrodesis of the knee is the obvious solution of the unstable Charcot knee joint, but it is notoriously difficult to obtain osseous union in *tabes dorsalis*.



FIG. 338.—Neuropathic joint of syringomyelia. Atrophic type.

perature are lost, but tactile sensation and muscular sense remain. Owing to disturbed sensation, slight injuries, notably cigarette burns, occur unnoticed.

(b) *Trophic Changes*.—Wasting occurs of all the muscles of the hand, and later the forearms. Also the tissues are more prone to injury, and healing is delayed. Eventually the soft tissues and finally the phalanges become absorbed (fig. 339).

(c) *Scoliosis* is sometimes present, owing to asymmetrical irritation of the pyramidal tracts and unbalanced action of the spinal muscles.

(d) *Unequal pupils*, due to asymmetrical irritation of the oculo-pupillary fibres, which pass down the cervical cord, and leave it in company with the first dorsal nerve, passing thence via the rami communicantes to the inferior cervical sympathetic ganglion, and along the carotid sheath and ophthalmic artery to the ciliary ganglion.

(e) *Spasticity of the legs*, due to pressure on the pyramidal tracts.

Syringobulbia, as evinced by hoarseness and dysphagia, may subsequently develop.

Treatment of the joint condition entails adequate support, pressure being carefully guarded against, as owing to insensitiveness to pain, pressure sores are particularly liable to occur.

2. **Syringomyelia** is due to gliomatous degeneration round the central canal of the spinal cord, and usually occurs in the lower cervical region. Joint complications occur in about 15 per cent. of cases of syringomyelia, and closely resemble the Charcot's joints described above, except that the arm is more often affected than the leg (80 per cent.) (fig. 338). The shoulder is most frequently attacked and, as with ball-and-socket joints, is of the atrophic type.

Examination of the patient often reveals other evidence of syringomyelia, such as :

(a) *Dissociation of Sensation in the Hands*.—

The sensibility to pain and variations of temperature are lost, but tactile sensation and muscular sense remain. Owing to disturbed sensation, slight injuries, notably cigarette burns, occur unnoticed.



FIG. 339.—Trophic loss of the fingers, due to syringomyelia. Morvan type.

4. Other Lesions of the Nervous System

Long-standing impairment of the trophic innervation of a limb is followed by changes in the small joints of the hands and feet. Such causes include spina bifida and peripheral nerve lesions, e.g. injury, neuritis, or leprosy. Of particular interest are the occasional cases of neuropathic arthropathy produced by *diabetic neuritis*. These diabetic neuro-arthropathies most commonly affect the foot and ankle (i.e. distal part of the extremity).

HYSTERIA (*syn.* MIMETIC JOINTS)

Disability in connection with a joint sometimes occurs in patients of hysterical tendencies, and is most commonly seen in adolescent or young adult females. The history in some cases is suggestive, in that the onset follows some emotional crisis, or mimicry of some joint condition seen in another patient. The larger joints are usually affected, the commonest being the hip, knee, and ankle. There is usually a bizarre quality about the gait and behaviour which does not occur in the known patterns of organic disease.

An especially marked example is 'hysterical inversion of the foot.' The patient walks on the outer border of the foot—the condition coming on quite suddenly in a patient otherwise in normal health.

The more important clinical features are as follows :

(a) *Inspection*.—The limb is often in an extreme position which is unusual for any early pathological condition, e.g. in the case of the hip joint the thigh may be markedly flexed, adducted, and inverted. Wasting of muscles, if any, is slight, and is due to disuse.

(b) *Palpation*.—Gentle palpation may appear to cause intolerable pain, but if the attention is distracted, considerable pressure will pass unnoticed. Otherwise no abnormal features are discovered.

(c) *Movements*.—All movements are restricted, and if the patient is requested to move a joint, both the group of muscles which carry out the movement, and also the antagonistic muscles, are contracted, so that the limb is rigidly fixed. This phenomenon can easily be appreciated by palpation of the muscles, while the patient attempts to carry out the desired movement.

(d) *Radiograph*.—In long-standing cases disuse atrophy of bone occurs, as is shown by thinning of the compact layer, and loss of density of the cancellous tissue owing to absorption of calcium salts.

(e) *General Examination*.—Other hysterical manifestations will probably be found, such as globus hystericus, anaesthesia of the palate, and glove or stocking anaesthesia.

Treatment.—Psycho-analysis might be employed in order to correct the abnormal mental outlook. Symptomatic treatment directed to the joint includes encouragement regarding movements, or an anaesthetic is given, and the position of the joint altered.

CHRONIC ARTHRITIS

Chronic arthritis can be classified as follows: (1) Tuberculous. (2) Gummatous. (3) Rheumatoid. (4) Osteoarthritic.

Tuberculous arthritis originates either in the synovial membrane or in the bone. In the knee and elbow, the disease usually first attacks the synovial membrane, whereas in other joints, notably the hip, the initial focus of infection is generally in the adjacent bone. In children synovial disease is commoner than in adults.

Four types of tuberculous arthritis are recognised but only the first two are important:

(i) *Synovial*.—Characterised by oedema and infiltration of the synovial membrane and periarticular tissues, giving rise to the typical 'white



FIG. 340. —
Tuberculous dis-
ease of the ankle
joint.

swelling' (fig. 340). The synovial membrane becomes thickened and succulent, and subserous tubercles appear. Gradually the normal structure is lost, and the membrane becomes converted into granulation tissue. Bone destruction is not a prominent feature in synovial disease, and though abscesses may form they are not as common as in the 'osseous' type of focus.

(ii) *Osseous*.—The infection commences in the bone adjacent to the joint and extends into the joint by the continuous invasion of the intervening tissue. This type is more insidious than the synovial type, and symptoms are usually present before clinical signs are obvious. Thus, in a child, an osseous focus in the neck of the femur or in the roof of the acetabulum may cause a limp and mild discomfort, yet the child may have a full range of passive movement, without spasm, when examined. In the later stages, when the osseous focus has invaded

the joint, there is extensive bone destruction, abscess formation, and even pathological dislocation.

(iii) *Caries sicca* is seen most commonly in the shoulder joint (p. 295).

(iv) *Hydrops*.—Excess of fluid is a rare occurrence in tuberculous arthritis because the main bulk of a swollen tuberculous joint is the thickened synovia, but occasionally the knee joint becomes distended with fluid containing fibrin, which is later converted into flat, oval objects, referred to as 'melon-seed bodies' (fig. 368). Most commonly the 'hydrops' of the knee has negative histological features and the ætiology is obscure. It is rare and often symptomless and must be distinguished from Clutton's joint (p. 282).

Clinical Features.—*Symptoms*.—Tuberculous arthritis presents an insidious onset, aching after use and stiffness following rest being early symptoms. Pain is not severe, but sudden strains and twists are deliberately or subconsciously avoided. When erosion of cartilage has occurred, 'starting pains' are characteristic, and occur just when the patient is dropping off to sleep. They are due to relaxation of the muscles which guard the joint, so that slight movement between the articular surfaces causes friction between the exposed and sensitive bones. Swelling of the joint is sometimes noticed by the patient, and is due to œdema of the synovial membrane and periarticular structures.

Some deterioration of the general health is to be expected, and the temperament of a child may completely alter, so that happiness and contentment give way to peevishness and fretfulness.

Signs.—Following the routine which always should be adopted for the examination of joints, we find :

(a) *Inspection*.—Both extremities are exposed, and the position of the affected limb is observed. Deformity in the early stages is due to the position of ease which the joint automatically assumes ; later, more marked deformity results from disorganisation. Swelling of the joint is due to thickening of

the synovial membrane by the granulomatous tissue so characteristic of the tuberculous process. The swelling fades away above and below the joint and is characteristically spindle-shaped. This spindle-shaped or globular swelling (seen very well in the knee) is accentuated by the marked wasting of the associated muscles, which is an important and characteristic feature of a tuberculous arthritis (fig. 341). In a case suspected to be an early tuberculous arthritis of the knee, the presence of good thigh muscles would be strongly against the diagnosis. The whiteness of the overlying skin is due to pressure of the œdema emptying the cutaneous capillaries, and this 'white swelling' thus stands in marked contrast to the pink or red blush which is to be expected in pyogenic infections. It is to be noted that, though white in appearance, an active tuberculous joint is still warm to the touch. This should not be confused with the 'cold abscess' which often accompanies an acute tuberculous arthritis. A cold abscess is cold (i.e. ordinary body temperature, not warmer) to the touch and the overlying skin is not reddened.

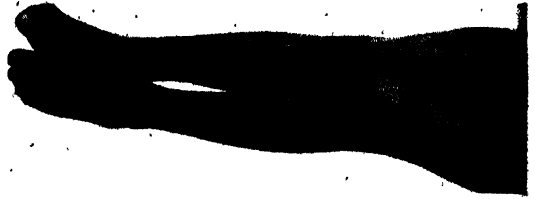


FIG. 341.—Tuberculosis of the left knee joint, showing flexion, swelling, and wasting of muscles.

In the later stages abscesses and sinus formation are apt to occur.

(b) *Palpation*.—An active joint will be unduly warm to palpation. A somewhat boggy or doughy thickening of the synovial membrane will be detected if this structure is sufficiently superficial. This doughy thickening of the synovia of the joint, together with the wasting of the muscles above and below it, may give the false impression that the bone-ends are actually enlarged. One characteristic of tuberculous disease is that all bony landmarks are usually somewhat obliterated by the synovial thickening.

(c) *Movement*.—The patient is requested to move the joint as far as possible in all directions, and it will be noted that active movements are limited in all directions.

The surgeon then puts the sound limb through its full range of movements, in order to gain the confidence of the patient, and also to ascertain the degree of mobility present in that individual patient. The passive movements of the affected limb are then tested, if possible while the patient's attention is diverted. In cases of tuberculous arthritis, active and passive movements are limited in all directions owing to protective spasm of muscles.

(d) *Measurement*.—The presence of muscular wasting is confirmed, and the amount of shortening, due to lack of growth or disorganisation of the joint, is estimated.

(e) *X-ray changes*.—Three types of change, singly or associated, are seen in tuberculous arthritis :

(i) *Generalised osteoporosis* due to disuse and hyperæmia. This can exist without any destructive changes, and is, in fact, the ordinary radiological appearance of a tuberculous synovitis.

(ii) *Loss of Bone Pattern*.—This is the so-called 'ground-glass' appearance in which the sharply defined outline of the cancellous trabeculae is lost, as though the film were seen through 'ground glass'. In the early cases the bones retain their normal outline so that true destructive changes are not obviously present. In more advanced cases the outline of the individual bones may be lost (fig. 342). This means that the individual bones are surrounded by granulation tissue which has eroded the thin shell of subchondral bone which normally gives them their clear-cut radiographic outline.



FIG. 342. — Tuberculosis of the wrist joint.

(iii) *Gross Destruction*.—Here large parts of the bone-ends are destroyed on both sides of the affected joint.

(f) *General Examination*.—Evidence of tuberculous trouble in other organs, either active, latent, or healed, may be discovered.

(g) *Special Tests*.—The erythrocyte sedimentation rate is increased. A negative tuberculin test is useful in excluding tuberculosis in children and sometimes it is helpful in young adults. Examination of pus aspirated from an associated abscess will often establish the diagnosis either by the direct staining of a smear, or by culture or guinea-pig inoculation.

Lymph-node and Synovial Biopsy.—Excision and microscopical examination of a regional lymph node shows invasion by *Mycobacterium tuberculosis* in a significant number of cases. Since the advent of streptomycin the danger of causing a sinus by direct biopsy of the synovial membrane of a tuberculous joint has become negligible, and lymph node biopsy is being replaced by the more certain method of direct synovial biopsy.

In suspicious cases the patient should be confined to bed as a therapeutic test and the joint immobilised. Mild toxic affections (such as may be associated with tonsillitis), or slight effusions due to injury, will rapidly respond, but a tuberculous lesion will do so less quickly. A tuberculous joint may improve quickly under rest, but it will relapse as soon as normal use is permitted.

Differential Diagnosis.—The diagnosis of a typical tuberculous joint causes no difficulty, but less typical cases may be confused with the following conditions: rheumatoid arthritis in a single joint, infective arthritis, hæmophilic joints (p. 76).

Diagnostic Triad.—When considering the possibility of hip symptoms being due to an early tuberculous arthritis, a useful diagnostic triad, taught for many years by Sir Harry Platt, concerns the most likely cause of hip disease at different age groups:

- 0–5 Congenital Dislocation
- 5–10 Perthes Disease
- 10–15 Slipped Epiphysis

Tuberculosis can start at any age, but these three diseases rarely occur outside these age groups. Perhaps the only exception to this rule is when Perthes disease starts in a congenital dislocation under treatment.

Natural History.—(i) *Resolution.*—Restoration of complete movement probably never occurs after a tuberculous arthritis, but after synovitis in children a considerable degree of painless movement is sometimes regained. This is particularly the case in the knee joint, where synovitis without destructive arthritis is common. Even here, in later life the damaged joint may become the site of osteoarthritis, and a late arthrodesis may then be necessary.

(ii) *Fibrous ankylosis* is the natural end-result of a healed tuberculous arthritis. If the fibrous ankylosis is very firm, it may function almost as well as a bony ankylosis—in which case it is called a 'sound' fibrous ankylosis. If considerable movement persists, it is likely to become painful at a later date and to show defective function, even in the absence of reactivation—in which case it is called an 'unsound' fibrous ankylosis and arthrodesis is to be advised.

(iii) *Bony ankylosis*, apart from surgical intervention, only occurs spontaneously in a case of tuberculous arthritis as a result of secondary infection, which gains entry to the joint along sinuses formed by abscesses bursting through the skin (fig. 343). There is one important exception to the rule that a tuberculous arthritis only heals spontaneously by fibrous ankylosis; in tuberculous caries of the spine the vertebral bodies commonly heal by osseous fusion.

(iv) *Cold Abscess.*—This almost invariably accompanies bone destruction by tuberculosis.

(v) *Pathological Dislocation.*—This frequently follows bone destruction and is rendered likely by muscular contractions with the joint in a position of deformity.

(vi) *General Dissemination.*—Acute miliary tuberculosis occasionally occurs as a result of blood-borne infection, particularly if the general resistance of the patient is undermined by any debilitating condition. This complication was in the past, prior to chemotherapy, a common cause of death in children, and tuberculous meningitis was often the final phase of the clinical picture.

(vii) *Toxæmia.*—If sinuses occur and prolonged suppuration follows, toxæmia is likely, and if of sufficiently long duration amyloid disease may develop. Amyloid disease is prone to occur if the hip is involved, for such a mutilating procedure as amputation through that joint is naturally dreaded and postponed by the patient.

Prognosis depends upon the following factors

(i) *Age of Patient.*—In general, though the cause of severe crippling, tuberculous arthritis is not fatal in childhood. In adult life the disease is much more serious and the prognosis is very bad when starting in the elderly.



FIG. 343.—Bony ankylosis of the hip joint with adduction of the femur, and a 'travelling acetabulum'. Result of secondary infection superimposed on a tuberculous arthritis. (London Hospital Museum.)

(ii) *Family History*.—Lack of resistance appears to be a familial characteristic, and when the disease occurs in a member of a tuberculous family the prognosis is less bright.

(iii) *The Stage of the Disease*.—Cases which are diagnosed in the early stages and which receive appropriate treatment are more likely to make a good recovery than those which have been neglected.

(iv) *The Presence of other Tuberculous Foci*.—If pulmonary or renal complications exist, the outlook is not good. A second osseous lesion is less serious than an osseous lesion combined with a visceral lesion.

Treatment.—GENERAL TREATMENT in a suitable centre is instituted without delay, and maintained until the condition is assumed to be cured. Fresh air, natural and artificial sunlight, suitable food, and cheerful surroundings are all of great importance. Of all these, the continuous exposure to fresh air is the most important. Children continue their education and adults are employed in occupational therapy. Streptomycin is a powerful agent in the treatment of osseous tuberculosis, combined with P.A.S. and I.N.A.H. (p. 23). Streptomycin undoubtedly allows the surgeon to operate successfully on many cases of tuberculous arthritis, which in the past would never have been suitable for surgical intervention or, if they had been operated on, would have broken down and developed tuberculous sinuses. Antibiotics have substantially reduced the annual mortality rate in sanatoria by clearing up the profusely discharging sinuses caused by secondary infection. Many cases of osseous tuberculosis died of miliary tuberculosis and meningitis, but these complications, thanks to streptomycin, have been substantially reduced in frequency.

The response to general treatment is judged by the fall of temperature and pulse-rate, absence of night sweats, increase in weight, rise in blood count, and the fall of the blood sedimentation rate. The fall of sedimentation rate does not run closely parallel with cure, and return to a normal level is often delayed.

LOCAL TREATMENT.—Three phases are distinguished in the classical treatment of tuberculous joints, but since the use of antibiotics these phases are not so rigidly adhered to as formerly.

Phase I.—Bed rest, with splintage, until quiescence is established (six to twelve months average—may be more).

Phase II.—Ambulant treatment in splints of leather and steel (may continue indefinitely, patient wearing splint the rest of his life).

Phase III.—Arthrodesis (here the fibrous ankylosis, resulting from the natural 'healing' of a tuberculous arthritis, is made permanently stable by being converted into an osseous ankylosis by operative intervention).

Phase I consists of immobilisation of the joint, with aspiration of abscesses to prevent the spontaneous discharge of a cold abscess, and thereby to forestall a sinus with the serious complication of secondary infection.

The limb is immobilised in the optimum position until some six months have elapsed after the last sign or symptom has disappeared. The actual length of time for which treatment should be continued, after the disease is apparently stationary, depends on the general condition of the patient, local response to treatment, including X-ray appearances, and the actual joint affected. The hip joint and spine in particular require prolonged fixation,

and one year's further treatment is usually advisable after the disease is apparently stationary.

If the limb is in an unsuitable position when first seen, e.g. considerable flexion of a knee joint, then the deformity is usually overcome by weight traction which is applied in the line of the bone below the affected joint. Cases with slight deformity often respond to a period of rest in bed, which results in disappearance of some of the muscular spasm, and permits the joint to be placed in the correct position. Occasionally a single forced manipulation under anaesthesia followed by plaster fixation is permissible.

Immobilisation is obtained by means of an appliance or apparatus specially suited to the part. For the *hip joint* a Jones's abduction frame, which fixes the spine and hip joints, is commonly applied. For the *knee joint* the Thomas's knee splint is used with skin traction. For the *ankle* a metal talipes shoe is usually preferred to plaster. The *spine* is treated either in a plaster bed with a turning case or on a Thomas's spinal frame. The *shoulder* is treated in a spica¹, and the *elbow* and *wrist* in either plaster or blocked leather supports.

Signs of Quiescence.—Quiescence is judged by local signs. The patient may look and feel very well even after only three months of convalescent therapy, but the tests for quiescence are made on local phenomena:

- (a) Absence of heat.
- (b) Absence of abscess formation.
- (c) Absence of muscle spasm.
- (d) Absence of swelling, often shown by reappearance of loose skin which can be picked up from the subjacent joint.
- (e) Absence of further destructive signs in the X-ray (i.e. not necessarily signs positively indicative of healing). The absence of further destruction after six months is presumptive evidence of the start of healing—which is what quiescence means.

Phase II.—Here the patient is allowed out of bed in an ambulant splint: back support for a spine, leather hip-spica for a hip, caliper splint for a knee, block leather or plaster splint for shoulder, elbow, and wrist.

Some patients may retain these appliances permanently—especially if they have other lesions, such as renal or pulmonary disease, which make them unsuitable for operative treatment.

Phase III.—Surgical intervention (arthrodesis) is indicated when the disease is quiescent.

Surgery since Antibiotics.—The introduction of streptomycin has radically altered the classical management of bone and joint tuberculosis. It is now possible to operate within three or four weeks of admitting a patient to hospital. The objective of treatment is no longer to cure the joint always by rendering it stiff. Synovectomy can often enable joint movement to be retained if bone destruction initially was absent in the radiograph. If the radiograph initially showed destruction, there may be no point in attempting to retain movement because osteoarthritis will inevitably develop after a number of years. In these cases antibiotics may permit arthrodesis to be performed within a few weeks of admission to hospital.

Amputation is now rarely needed in the treatment even of advanced tuberculous arthritis.

¹ Spica, Latin = an ear of corn, the individual grains of which overlap each other.

TUBERCULOSIS OF SPECIAL JOINTS

Sacro-iliac.—Owing to vagueness of the early symptoms and late appearances of definite signs, the correct diagnosis is often delayed. A local ache, vague sciatica, and sometimes a slight limp are the earliest slight symptoms. The condition should always be remembered in the differential diagnosis of the common complaint of sciatica. When the disease is established, pressure on the iliac crests causes pain. A radiograph confirms the diagnosis.

Treatment necessitates operation as soon as the condition is quiescent; obliteration of the joint causes no disability, and removal of diseased tissue and fixation of the joint favour a cure in the shortest possible time. Access is gained by removal of the posterior superior spine and adjacent iliac crest. Tuberculous granulation tissue and diseased bone are removed with a sharp spoon and gouge, and a bone graft or peg is driven through the bones so as to bridge the joint.

Hip.—The disease usually commences in bone, either in the under-surface of the neck of the femur or in the acetabulum.

Early symptoms include a limp, and pain commonly referred to the knee joint by the geniculate branch of the obturator nerve.

During the *first stage* of this condition the joint is held in the position of ease, i.e. flexed, abducted, and everted. The abduction gives rise to apparent lengthening of the limb. As the joint becomes more painful, the patient is increasingly inclined to assume a recumbent position, during which the patient lies on the sound and painless hip. Thus in the *second stage* the affected joint gradually becomes adducted and inverted, and flexion becomes more marked (fig. 344). Owing to adduction, the limb now is apparently shortened. The *third stage* corresponds with the start of articular disorganisation, including



FIG. 344.—Tuberculosis of the left hip, second stage. The arrow indicates a peri-articular abscess.

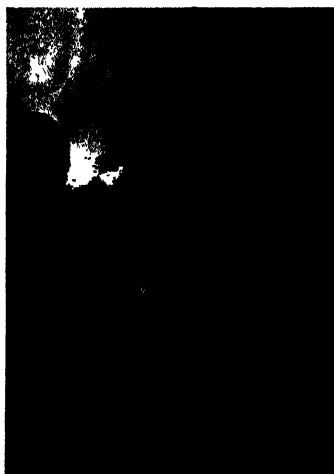


FIG. 345.—Active tuberculosis of hip in child — 'wandering acetabulum'.

absorption of the femoral head and acetabular cavity (fig. 345). The position of adduction, flexion, and inversion encourages the head of the femur to press more strongly against the dorsal lip of the acetabulum than into

the central depths of the socket as occurs in the normal or adducted position of the hip. Absorption of bone, combined with continuous pull of spastic muscles, results in 'travelling acetabulum' in which the dorsal lip of the acetabulum is eroded to allow the head of the femur to escape on to the dorsum ilii, i.e. 'pathological dislocation'. During the third stage disorganisation of the joint leads to true shortening of the limb, in addition to the apparent shortening due to adduction (fig. 346).



FIG. 346.—Tuberculosis of the left hip, third stage.

Differential Diagnosis.—Early acute slipped epiphysis with super-added spasm may cause confusion, but a week or so in bed results in disappearance of the spasm, after which the typical signs of unmasked coxa vara become evident (p. 337). Conditions as spasm of the psoas (from a chronic appendix abscess) and tuberculosis of the gluteal bursa also must be considered.

The Thomas Test for Fixed Flexion Deformity.—When a patient with a fixed flexion deformity of the hip stands erect, he is able to bring the knee of the affected limb to the side of the normal knee by tilting his pelvis and so producing a marked lumbar lordosis. This same concealment of a flexion deformity occurs when the patient lies in bed with both knees side by side. It is to reveal this concealed deformity in bed that H. O. Thomas first described his test, which is carried out in this way: place one hand under the lumbar spine to assess the amount of lordosis existing between the back and the surface of the bed or couch; take hold of the normal leg and flex the knee and hip as much as is necessary to flatten the lumbar lordosis. In this position the diseased hip will assume its true position, and if a flexion contracture is present the knee of the diseased side will rise from the bed, and the angle between the thigh and the horizontal will be the amount of fixed flexion deformity (fig. 347).

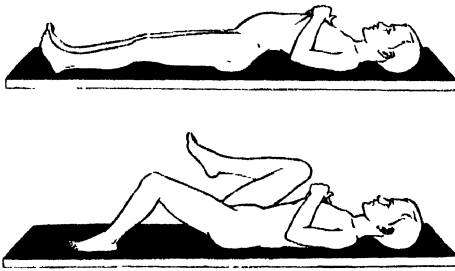


FIG. 347.—The Thomas Test for flexion contracture in the hip.

Treatment is conducted along the lines already prescribed. Deformity is corrected, usually by weight traction, and then a Robert Jones's frame fitted and later a plaster hip-spica. When the disease is quiescent, either arthrodesis is performed, or a blocked leather hip-spica fitted if for any reason operation is contraindicated.

Operative measures such as arthrectomy or excision of the head of the bone are obsolete, as excellent results are obtained by a fusion operation, e.g. extra-articular arthrodesis.

Extra-articular Arthrodesis.—Firm ankylosis in good position is the best result which can be expected, time is saved and this result obtained more certainly by arthrodesis. Extra-articular techniques are often preferred to intra-articular operations in tuberculosis, but experience with streptomycin now indicates that intra-articular procedures can be used with relative impunity.

The technique of hip fusion known as the ischio-femoral arthrodesis (Brittain) is probably the one most extensively used for tuberculosis of the hip. In children it is certainly a highly satisfactory procedure. It is an extra-articular method and combines a subtrochanteric osteotomy (which allows a fixed deformity to be corrected)

Hugh Owen Thomas, 1834–1891, who practised as a bone setter in Liverpool.
Sir Robert Jones, 1858–1933. Surgeon, Royal Southern Hospital, Liverpool.
J. A. Brittain, 1904–1964. Orthopaedic Surgeon, Norfolk and Norwich Hospital.

with a tibial bone graft which thus short-circuits the hip joint (fig. 348). Under the protection of the bone graft the diseased hip frequently undergoes spontaneous osseous fusion with complete disappearance of all tuberculous cavities.

In a very small number of cases where supuration persists and the patient's life is in danger a disarticulation of the hip may be indicated.

Knee.—The knee joint is commonly affected by tuberculous disease, and the diagnosis is, as a rule, not difficult. In neglected cases the position of 'triple deformity' will occur, which consists of flexion, external rotation, and backward subluxation of the tibia, the latter occurring when the cruciate ligaments have become softened and destroyed. Arthrodesis of the knee joint yields good results provided bony ankylosis is obtained.

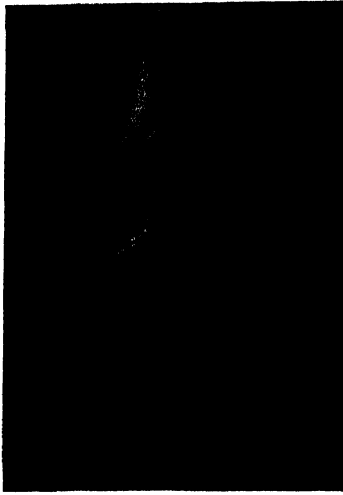


FIG. 348.—Brittain's ischio-femoral, extra-articular arthrodesis of the hip.

The technique most widely used for arthrodesis of the knee is probably that of compression arthrodesis. This is an intra-articular method in which the articular surfaces are sawn away to give flat surfaces of cancellous bone which are then pressed firmly against each other by transfixing the femur and tibia, above and below the joint level, by transverse stainless steel nails (fig. 355). The outer ends of these nails are approximated by means of special clamps. Under these conditions union occurs in six weeks and consolidation is complete in three months.

Ankle.—Tuberculous disease of this joint usually commences in the synovial membrane. Puffiness first appears under the extensor tendons, and later at the sides of the Achilles¹ tendon.

Arthrodesis is most commonly performed through an incision on the outer side of the joint, and a sliding bone graft from the tibia is inserted into the talus. In advanced cases with multiple sinuses, amputation is indicated, and should be performed at the modern site of election, i.e. about 5 inches (12.5 cm.) below the upper end of the tibia.

Shoulder.—On examination, wasting of the deltoid muscle is obvious (fig. 322), and the arm is held against the chest wall. Movements are all limited, and pain is produced when they are attempted. Treatment consists in fixation of the joint in 45° of abduction, and care must be taken to observe that true abduction is obtained as distinct from apparent abduction due to rotation of the scapula.

Following routine principles, arthrodesis is advisable when the disease is quiescent but, on the other hand, a sound fibrous ankylosis of the shoulder following the natural healing of tuberculosis in a sedentary worker need not always be arthrodesed because it has to take very little strain (unlike the weight-bearing joints). In women good results can sometimes be obtained by excision of the shoulder joint because a stiff shoulder makes it difficult for a woman to dress or arrange her hair.

¹ Achilles, the Greek hero. It is said that his mother held him by the ankle in order to dip him in the river Styx which, according to Greek mythology, separates this world from the next.

It is erroneously stated in most text-books that the commonest form of tuberculous disease of the shoulder is 'caries sicca'. This is not so; cold abscesses form round the tuberculous shoulder joint quite as often as round any other joint. The term 'caries sicca' is a relic of the era before X-ray examination, and the very common 'frozen shoulder' in those days was probably diagnosed as 'dry caries'. In modern times the error is quite the reverse: there is a danger of the occasional 'caries sicca' being diagnosed as a 'frozen shoulder' and treated by repeated manipulations and active exercise. X-ray examination will easily distinguish, because the term 'caries' means that destructive changes will be seen in the X-ray, whereas in 'frozen shoulder' the X-ray is normal.

Elbow.—In this joint the disease usually commences in the synovial membrane. The arm becomes weak and aches after use, and a doughy swelling appears on either side of the triceps tendon. Fixation is obtained by a plaster-of-Paris cast. Abscesses usually appear posteriorly. Arthrodesis is usually advised, though excision can give a satisfactory result with a mobile elbow if the patient is not faced with heavy work.

Wrist.—This joint is not uncommonly affected in elderly patients. Carpal bones are readily destroyed, and abscesses appear under the extensor tendons on the back of the wrist. In elderly patients the wrist frequently demonstrates the curious condition of 'senile tuberculosis', in which an appearance of gross destruction, getting neither better nor worse over a period of years, can be present without any signs of systemic ill-health. However, as a rule the onset of tuberculosis in the elderly is a serious condition; patients who develop tuberculosis of the spine or the hip after sixty have a very poor chance of survival.

RHEUMATOID ARTHRITIS

is seen in both sexes but most commonly in young females and commences in the small joints of the hands and feet. It gradually spreads to the large joints, thus causing progressive crippling, until the patient may become almost helpless. It is still of unknown ætiology and in the past was attributed to toxic absorption from septic foci. It is a systemic disease affecting the collagenous part of the locomotor system and it is thought to be one of the auto-immune group of diseases. During the earlier stages of the condition bouts of pyrexia occur periodically, associated with sweating, tachycardia, and exacerbations of pain and swelling in the affected joints. Fibrosis and contraction of ligaments gradually occur, and eventually the fingers become flexed and the hand fixed in a position of ulnar adduction (fig. 349). This disease is apt to run a painful course to more or less complete crippledom.

Rheumatoid arthritis starting late in life often has not the acute systemic



FIG. 349. — Typical rheumatoid arthritis of the hand.

disturbance associated with this polyarthritic condition in early adult life. In both cases the activity of the disease is clearly shown by the blood sedimentation rate, which is markedly elevated in this condition. A typical level of E.S.R. from active rheumatoid arthritis is 40 mm. in the first hour (Westergren).

Still's disease is a similar condition occurring in children, usually as they approach the second decade. In addition to joint changes, splenic and lymphatic enlargement and lymphocytosis are also associated.

Treatment.—Because the nature of rheumatoid arthritis is unknown, the treatment is empirical and symptomatic. Gold injections seem to improve a small but significant number of the cases who can tolerate the treatment. The great difficulty the patient experiences in carrying out the simplest household duties results from the pain and spasm associated with disorganised joints, and this can be controlled to a certain extent by very heavy doses of aspirin. Joints can be supported with bandages or plaster splints, and in some cases comfortable fibrous ankylosis can be brought about by prolonged splintage. Treatment is mainly directed to prevent deformity.

Rheumatoid arthritis offers a limited scope for surgery, but in a few cases the correction of fixed deformity by osteotomy, or capsulotomy of flexed joints, may be of value. Very painful arthritis of the knees can be relieved by arthrodesis, but arthrodesis is not of much value in other joints affected by rheumatoid arthritis.

ANKYLOSING SPONDYLITIS (*syn.* MARIE-STRÜMPPELL, SPONDYLOSE RHIZOMYELIQUE)

This is a crippling disease which seems to have some features in common with rheumatoid arthritis, but which forms a distinct clinical entity.

The disease is essentially a process of ossification of the ligaments and capsular ligaments of joints, which results in complete bony ankylosis of the great central articulations of the body (unlike rheumatoid arthritis which is not specially an ankylosing state and which affects the small joints of the distal extremities first). Whereas rheumatoid arthritis affects mainly females, ankylosing spondylitis is more common in men, starting in early middle life.

The articulations most affected, in order of frequency, are sacro-iliac joints, spine (from below upwards), hips, costal joints, and shoulders.

In addition to bony fusion of these joints, the spine becomes flexed so that eventually the unfortunate patient is literally bent double and is forced to seek help when he can no longer see in front of himself no matter how much he may try to look upwards.

In the early stages of the disease, attacks of pain may simulate 'fibrositis', but suspicion will be aroused if the stiffness of the back persists when the exacerbations of pain have subsided. Frequently, continuous bone ache is a very disturbing symptom.

X-ray will establish the diagnosis by the complete bony obliteration of the sacro-iliac joints and by the presence of ossification of the spinal ligaments ('bamboo spine', figs. 350 and 351).

Alf Westergren, Contemporary. Professor of Medicine, Stockholm.
Sir Frederick Still, 1868–1941. Physician, King's College Hospital and Hospital for Sick Children, London.
Pierre Marie, 1863–1940. Professor of Neurology, Hospice de Bicêtre, Paris.
Ernst Adolf Gustav Gottfried Strümpell, 1853–1925. Professor of Medicine, Leipzig.

The patient often looks ill and is usually ill-nourished. In active disease the E.S.R. is usually elevated to 40 mm. (Westergren) in the first hour.

Treatment.—There is no specific treatment for this distressing condition. Fortunately the less severe cases often ‘burn themselves out’ before the full



FIG. 350.—Ossification of the lumbar spine ('bamboo' spine).



FIG. 351.—Ankylosing spondylitis: fusion of sacro-iliac and hip joints.

picture of spinal ankylosis in gross deformity is reached. Severe cases often succumb to chest complications as a result of the ankylosis of the thoracic cage.

Deep X-ray therapy to the spine seems to be of real value in a significant number of cases and is worth while as far as the relief of spinal pain is concerned. Beyond this, surgery can only offer splintage to prevent further deformity, or osteotomies to correct established deformity, and arthroplasty to release ankylosed hip joints.

Spinal osteotomy is occasionally useful in order to correct gross cases of rigid flexion deformity of the spine, thereby improving the patient's unfortunate lot.

OSTEOARTHRITIS

Osteoarthritis is quite unlike rheumatoid arthritis both clinically and pathologically. It seems to be mainly a mechanical ‘wearing out’ of a joint and is not associated with any systemic disturbance. It is generally a monarticular lesion (unlike rheumatoid arthritis) and occurs mainly in the weight-bearing joints. It affects patients in late life, and more commonly the hips or knees than the small joints of the distal extremities. Almost all elderly subjects have some degree of this condition in hips, spine, or knees even in the absence of symptoms and merely as the natural result of the ageing process.

Evidence to suggest that local mechanical causes (as opposed to toxic causes) may be the ætiological factor in osteoarthritis is seen in the condition of ‘traumatic osteoarthritis’. Traumatic osteoarthritis follows injuries to joints, especially fractures, which have disturbed the perfect mechanical ‘fit’ of the articular surfaces, and thus all the changes of osteoarthritis eventually

develop in a joint which prior to the injury was known to be quite healthy. Examples are those of Pott's fracture of the ankle ; traumatic dislocation of the hip, followed years later by osteoarthritis ; arthritis of the wrist following non-union of the carpal scaphoid.



FIG. 352. — Osteoarthritis of lateral compartment of knee superimposed on genu valgum.

Even in the absence of injury which has disturbed the anatomy of the joint, it may be possible to detect other anatomical errors (or excessive use) which have made the joint 'wear out' more quickly than normal (fig. 352). Thus in the hip a slight congenital subluxation may exist for forty years without causing symptoms and then give rise to osteoarthritis because the weight of the body is being taken on one part of the femoral head instead of being evenly distributed over a larger area. Similarly, a unilateral slipped femoral epiphysis or Perthes' disease may leave a femoral head which is no longer spherical and this will wear out before the normal hip on the other side.

Joints which have been damaged by inflammatory disease but which have made a reasonable functional recovery will also later wear out prematurely and become osteoarthritic. Frequently the knee joints in rheumatoid arthritis will later take on some of the characteristics of osteoarthritis due to the mechanical effects of attrition.

It is sometimes useful to call these types of osteoarthritis 'secondary osteoarthritis' to imply that a pre-existing cause is known. In cases where no known cause pre-existed, the disease is often called 'primary osteoarthritis'.

Pathological Changes.—Thickening of the synovial membrane occurs, and as this progresses a villiform process may develop which on movement is nipped between the bones, and when unduly enlarged is termed a 'lipoma arborescens'. The thickened synovial membrane overlaps the edge of the articular cartilage, and under this fringe cartilage cells proliferate. Thus lipping or osteophytic growth occurs, as the cartilage cells subsequently ossify. Owing to the degeneration of the cartilage it is worn away on pressure-bearing surfaces, with resulting erosion and grating on movement. The underlying bone thus exposed reacts to pressure, and becomes dense and smooth i.e. 'eburnation' (fig. 353).



FIG. 353.—Osteoarthritis of the knee joint, showing thickened synovial fringes, lipping of the cartilaginous margins, and eburnation of bone on surfaces exposed to friction. (R.C.S. Museum.)

Not infrequently exacerbations occur with synovial effusion, and as a result a bursa communicating with the joint (e.g. a psoas bursa), is apt to become distended. In the knee an effusion distending the semimembranosus bursa resembles a Baker's cyst (p. 318) but in actual fact this is a rare condition while the ordinary 'semimembranosus bursa' is a very common condition. This latter occurs in young patients with normal knee joints, and is probably nothing more than a simple ganglion which does not communicate with the joint cavity (p. 318).

Occasionally chondrification or ossification occurs in the synovia, and in the case of the knee joint a solid mass sometimes appears due to chondrification of the synovial membrane lining the suprapatellar pouch. Ossified synovial villi or osteophytes sometimes become detached and form loose bodies in the joint.

Clinical Features.—The early symptoms of osteoarthritis are pain and stiffness. Pain is characteristically aggravated by changes in the weather, so that the appropriate term 'barometric joint' is sometimes used. Stiffness is intensified by rest, and the joint must be 'worked loose' before the range of movement is recovered. In advanced cases creaking, or locking of the joint by osteophytes, is a tiresome feature.

The signs depend upon the extent to which the disease has progressed. In the early stages some effusion, thickening of synovial membrane, and perhaps a little wasting of muscles are the only features. At a later stage lipping or osteophytes are sometimes palpable, while grating or creaking is detected on movement. The range of movement eventually diminishes and a painful ankylosis in a deformed position is the rule.

Radiologically osteoarthritis is characterised by loss of joint space at the site of maximum pressure, sclerosis of the adjacent bone surfaces, osteophytic lipping of the joint margins and cyst formation (fig. 354).

Treatment.—In the early stages, the joint should be protected from exposure and cold (e.g. knitted woollen knee caps). Rest to the joint should only be permitted during painful exacerbations, and movements and massage are resumed as soon as they can be tolerated. Radiant-heat baths, diathermy, and rubefacients are useful.

The surgical treatment of osteoarthritis is not to be considered until the patient is driven to it by pain or severe reduction of activity. The operative procedures available are the two extremes, i.e. arthrodesis or arthroplasty. Often osteotomy can be used if deformity is the primary disability. Considerable experience is necessary in choosing the most suitable surgical procedure for the requirements of the particular patient. The problems

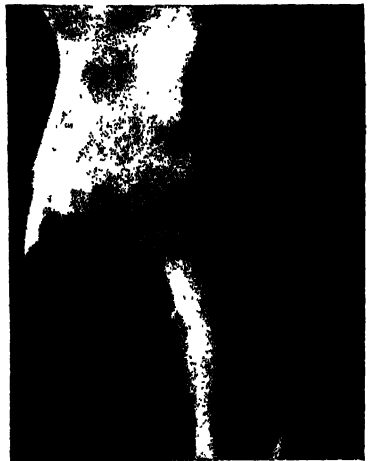


FIG. 354.—Osteoarthritis of hip.

differ in different joints, but the two most commonly requiring attention are (1) the knee, and (2) the hip.

Osteoarthritis of the Knee.—Arthroplasty of the knee is an unsatisfactory procedure but continuous efforts are being made to develop a method. Sometimes it is possible to relieve pain, and avoid stiffening of the knee, by removing the patella and excising the suprapatellar pouch. Osteotomy of the upper end of the tibia can sometimes benefit osteoarthritis of the knee, either by correcting deformity or influencing the blood-supply. The results of

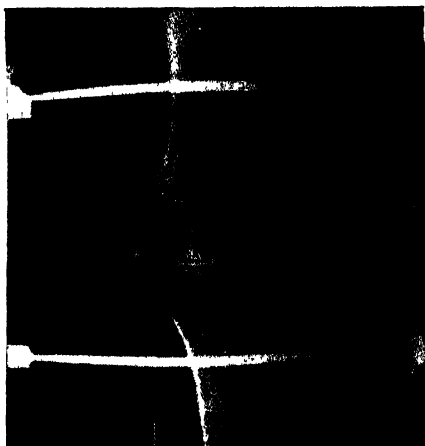


FIG. 355.—Charnley compression arthrodesis of the knee.

arthrodesis of the knee can be excellent, and the benefit so quickly appreciated without protracted rehabilitation (fig. 355), that arthroplasty of the knee need only be considered in bilateral cases. The complete freedom from pain, and the ability to walk without a stick, are so much appreciated that the nuisance of the knee being stiff is easily tolerated by male patients and, in severe cases, even by female patients. Bilateral osteoarthritis offers serious problems, but even here there are many cases, provided that the patient is intelligent and co-operative, which are enormously benefited by bilateral knee arthrodesis.

Osteoarthritis of the Hip.—The choice of the best surgical method is still a matter of discussion; the status of the two rival methods (arthroplasty and arthrodesis) tends to vary in each decade as new technical improvements are made in one or other method. Obviously, arthroplasty is indicated if osteoarthritis of the hips is bilateral.

Arthrodesis.—Unilateral osteoarthritis in patients under fifty years of age is probably best treated by arthrodesis, because it is still doubtful whether many arthroplasties will remain painless much longer than ten years. Sound bony ankylosis of the hip, in good position, with a fully mobile knee gives a very excellent result in patients of active and agile temperament. Patients who have an osteoarthritic hip which is already ankylosed, though very painful, are very suitable for arthrodesis because they are already accustomed to a stiff hip.

Arthroplasty of the hip, though often imperfect from a mechanical point of view (necessitating a stick and not permitting long distances to be walked or weights to be carried) is often preferable to arthrodesis in elderly women, especially if they still have a good range of movement in the painful hip.

The actual techniques of arthroplasty are continuously changing as experience accumulates. In the 'cup' arthroplasty of Smith-Petersen a stainless steel cup is placed between the acetabulum and the head of the femur, but this method is diminishing in popularity. Replacement of the femoral head by the metal Austin Moore prosthesis occasionally gives valuable relief. In very disabled patients the old operation of resection of the head and neck of the femur (the pseudarthrosis of the hip) has a definite place, but it has the disadvantage of shortening the limb and necessitating the use of a stick. Research is in progress on methods of replacing both the acetabulum and the femoral head, and the early results of this technique are spectacular.

Osteotomy.—Osteotomy was introduced by MacMurray for the treatment of osteo-

arthritis even when the hip is not ankylosed. The principle is to divide the femur just above the lesser trochanter, and usually internal fixation is used to hold the fragments and enable external fixation to be avoided (fig. 356). The rationale of this method is obscure and at the moment no satisfactory explanation of the success of pain relief has been put forward. In some cases the joint-space will return, indicating that new articular cartilage has developed. Not all cases are successful.

Loose bodies (a definition which excludes foreign bodies introduced from without) arise from the various constituents of the joint :

SYNOVIAL FLUID.—A single fibrinous body sometimes results from a hæmorrhagic or inflammatory effusion, or less commonly the joint contains many loose bodies (fig. 357). In tuberculous disease 'melon-seed' bodies may be present in large numbers (fig. 368). A sesamoid bone in the neighbourhood of a joint must be distinguished from a loose body (fig. 358).

SYNOVIAL MEMBRANE.—Villous processes, such as those which occur in osteoarthritis, become detached, especially if bone or cartilage develops within them (synovial chondromatosis).



FIG. 356.—Osteoarthritis of hip treated by osteotomy with internal fixation. Joint space has not returned but symptomatic cure has occurred.

BONE.—Injury may cause separation of a small portion of bone, e.g. a tibial spine. Osteophytes are occasionally detached in cases



FIG. 357.—Loose bodies in knee joint.



FIG. 358.—Sesamoid bone in the outer head of the gastrocnemius muscle (fabella). Oval in shape and behind the condyles.

of osteoarthritis. If the loose body contains living cells, growth may continue, nutriment being obtained from the synovial fluid.

*Osteochondritis dissecans*¹ (*syn.* Paget's quiet necrosis) is the commonest cause of a single loose body in young people. It most commonly occurs in the knee joint (fig. 359), and the loose body arises most frequently from the lateral aspect of the medial condyle adjacent to the intercondyloid notch. The spontaneous separation of the fragment of bone, together with overlying cartilage, about the size of a sixpence or a shilling, seems to result from the sequestration of the underlying bone, and it has been suggested that this is caused by the thrombosis of an end artery supplying an area of bone of this size. Possibly direct trauma caused by the impact of the tibial spine against the condyle determines the thrombosis. The condition can, however, occur on the posterior surfaces of the femoral condyles which is against this traumatic theory. A condyle of the humerus is the second commonest site.

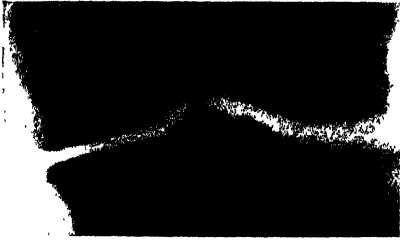


FIG. 359. — *Osteochondritis dissecans* affecting the internal condyle. (Professor Carl Krebs, Aarhus, Denmark.)

Osteochondritis dissecans often starts without any definite history of trauma at about the age of eighteen. It presents as a mild traumatic effusion. Symptoms of a vague character may persist for many months and then settle completely. The separation of the fragment may not take place till some years later, and the joint may then suddenly lock 'out of the blue', all past history of knee trouble having been forgotten.

Symptoms.—The commonest symptom caused by a loose body is locking of the joint. This causes severe pain and is followed by synovial effusion. Locking is often only momentary, and some slight manoeuvre of the joint by the patient causes the body to slip out from between the bones and so unlock the joint. Repeated attacks of locking and synovitis eventually result in degeneration of the joint and osteoarthritis. Occasionally the patient learns to manipulate the joint so that the loose body becomes palpable, in which case it is felt to slip away from under the examining finger when the position of the joint is altered. This free mobility explains why loose bodies are sometimes referred to as 'joint mice'. A radiograph usually reveals the presence of the body.

Treatment.—Unless the joint is already disorganised, the loose body should be removed in order to relieve symptoms and prevent the onset of osteoarthritic changes. In the case of the knee joint, the body should be manipulated, if possible, so that it comes to lie in the suprapatellar pouch. It is then imprisoned in that situation by a firm, sterile, elastic bandage passed around the extended joint, so that a small vertical suprapatellar incision is sufficient to allow removal. If the loose body cannot be found, a full arthrotomy and exploration may be needed and the joint put through a range of movement till the loose body presents in the wound.

Hæmophilic Joints are described on p. 76.

¹ Note spelling: this implies dissecting, *not* dessicating.

CLICKING JOINTS

Many people are able to produce loud clicks from what would appear to be otherwise normal joints. There are two main sources of these clicks, intra-articular menisci in joints which possess these structures, and the movement of tendons over bony anatomical prominences near the joint.

Sometimes these clicks may be sufficiently disturbing to warrant surgical treatment, but more often than not the trouble is that the patient is rather neurotic, finding a trivial discomfort intolerable and persistently evoking the click by a 'self-inflicted' habit. In these cases reassurance must be tried because the results of operative treatment are often rather disappointing. The condition is to be distinguished from the 'trigger' phenomenon of stenosing tenosynovitis.

Common examples of snapping joints due to intra-articular menisci occur in the jaw and the knee. Examples due to snapping (or 'clicking') tendons are found in the hip, shoulder, and peroneal tendons.

Clicking jaw is generally caused by derangement of the articular meniscus. In patients where the jaw locks or a palpable click is elicited, removal of the cartilage is often curative, though it should only be done after six months of reassurance and after any errors of dental occlusion have been corrected by appropriate orthodontic measures. Patients without any obvious click, and complaining mainly of pain, are usually cured by reassurance.

Patients insisting on the operation should be warned that some drooping of the associated eyebrow might occur as a result of damage to the uppermost branch of the facial nerve and that transient deafness due to œdema of the external auditory meatus may last some weeks.

Snapping knee is due to a congenital abnormality of the lateral meniscus (congenital discoid cartilage). There is no neurotic element in these cases. Instead of the cartilage developing in its normal shape as a disc with a central aperture, it is present as a solid disc, thicker in the centre than at the periphery, which slips about causing a click. The click is often of surprising magnitude, being clearly audible even at some distance. The knee will jump as though it is dislocating. The condition is usually first noticed between late childhood and puberty. Operative removal completely cures this rather uncommon condition.

Snapping hip is caused by the tendon of tensor fasciæ femoris (i.e. the ilio-tibial band) slipping backwards and forwards over the prominence of the great trochanter. There is usually a strong neurotic element in these cases. If the click is very severe, the fascia can be divided at the level of the trochanter.

Snapping Shoulder.—Patients with indefinite pain in the shoulder, probably originating in the supraspinatus tendon or the subacromial bursa, often insist that it must be related to 'clicks' which they can elicit from their shoulder. In the majority of cases they are unrelated and the patient can be reassured.

GOUT

Gout is a disturbance of purine metabolism associated with an excess of uric acid in the blood. There is often a familial tendency with a ratio of men to women of 10:1. In the past gout was usually associated with high living and the excessive intake of meat and alcohol, but in modern times this does not seem to apply so clearly.

Acute gout can occur without radiological signs in the bones of the joint and it may recur repeatedly as acute attacks with the patient apparently reverting completely to normal between attacks. The af-



FIG. 360.—Gouty tophi.

fectured joint, usually in the foot, can be extremely painful, swollen, and red. First attacks may baffle the diagnostician who does not think of the condition; acute suppurative arthritis may be seriously considered, but the patient with acute gout appears too well and the temperature, pulse rate, and white cell count are normal. A raised blood uric acid during the attack (normal 2 to 4 mg. per 100 ml.) confirms the diagnosis. Areas of inflammation in acute gout can appear over tendon sheaths, again usually in the foot, and diagnosis may be difficult.

Chronic gout can occur without acute attacks of pain, and without a raised blood uric acid. The picture is that of a chronic arthritis. Radiological signs in the skeleton are here definite, and deposits of urates appear as 'punched-out' areas in the bones near a joint as these deposits are radio-translucent. Tophi, which are localised deposits of sodium biurate, may occur and, if subcutaneous, they may ulcerate through the skin to appear as extruding chalky masses. The hands (fig. 360) olecranon bursae, and lobes of the ear are favourite sites for tophi. Colchicine 0.5 mg. is an ancient and specific remedy for an acute attack and now Indocid (Indomethacin 25 mg.) is a useful anti-inflammatory substance. However, the treatment of the metabolic disease is essentially medical.

CHAPTER 15

MUSCLES, TENDONS, AND BURSÆ

JOHN CHARNLEY

INJURIES OF MUSCLES AND TENDONS

Contusion of a muscle results from direct injury. Localised pain follows attempts at contraction, and an extravasation of blood occurs within the muscle sheath. This extravasation often appears at a considerable distance from the actual site of injury, e.g. hæmorrhage from a torn rectus femoris in its upper part usually appears near the patella.

Rest and pressure dressings are required for the first two days, and, when risk of further extravasation has passed, gentle active movements will expedite absorption, and prevent, or limit, subsequent stiffness.

The rupture of some deep fibres of the quadriceps with a hæmatoma on the anterior surface of the femur is a not uncommon spontaneous injury in sport, but it may also result from a direct contusion. If too vigorously managed and passively stretched, this is a notorious site for 'myositis ossificans' (fig. 361).

Rupture of a muscle usually occurs at the junction of tendon and muscle itself. Thus the *quadriceps extensor* ruptures immediately above the patella. It is not a common injury and usually occurs in elderly patients, often after a trivial indirect injury such as stumbling. The gap is easily visible and palpable when the patient contracts the quadriceps muscle (fig. 362). The patient is unable actively to extend the knee. Repair with mattress stitches is necessary.

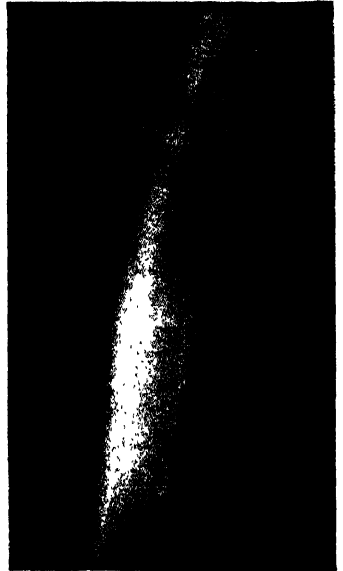


FIG. 361.—Myositis ossificans of the thigh. Late appearance of bone on anterior aspect of the femoral shaft.



FIG. 362.—Torn quadriceps femoris muscle.

Rupture of the muscle belly of *rectus femoris* occurs at about mid-thigh level. This occurs not uncommonly in young athletic persons and is caused by indirect violence during very great effort. Extension of the

knee is still possible after the pain of the rupture has subsided. When all local œdema has disappeared, the proximal end of the muscle will become

prominent and stand out as a visible lump when the patient contracts the thigh. No surgical treatment is possible (since it will not hold stitches), nor is it necessary because it causes no permanent disability.

Rupture of Tendons.—Normal tendons rarely rupture. With the exception of 'mallet finger' (p. 307), tendon ruptures occur in or after middle-age and often are associated with local attrition by friction against bone.

The Achilles tendon is the commonest tendon to rupture, and as it is frequently diagnosed incorrectly, special note should be made of its clinical features. It is an injury most common in males in later life and, though it can happen during games and severe exertion, it usually happens as a result of a stumble when the violent contraction necessary to regain balance causes the rupture. The sensation of rupture is often mistaken for a blow on the back of the leg and the patient will often turn round to see who has hit him. The rupture usually occurs about 1 inch to $1\frac{1}{2}$ inches (2.5 cm. to 3.75 cm.) above the insertion of the tendon into the os calcis.

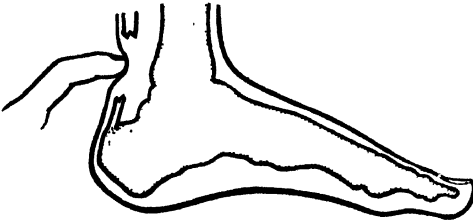


FIG. 363.—Palpable gap in diagnosis of rupture of Achilles tendon.

Clinically, a gap is easily palpable at the site of the rupture, and in early cases this is sometimes even visible (fig. 363). The foot can be dorsiflexed to a greater extent than the normal foot. The power of plantar flexion is markedly reduced, but it is to be noted that it is never com-

pletely absent. This is a most important point, because one can be led to imagine that the tear must be incomplete by the fact that some power of plantar flexion exists. This power is, of course, exerted by the long flexors of the toes, tibialis posterior, and peroneus longus. Very few cases of suspected 'partial' rupture of the tendo Achillis are in fact incomplete; it is dangerous to make this diagnosis without an expert opinion, because suture of the tendon is much more difficult if the operation is delayed.

The diagnosis of rupture of the Achilles tendon has been obscured in the past by the hypothetical diagnosis of 'rupture of the plantaris tendon'. It is probable that plantaris rupture is an imaginary condition, and that the clinical picture is that of a partial tear of the calf muscle fibres at a much higher level than the true Achilles rupture. The so-called 'plantaris rupture' was diagnosed in retrospect by complete recovery without operation. The clinical picture is seen in a younger age group than that of the classical Achilles tendon rupture.

Treatment.—Early suture of the Achilles tendon gives excellent results, but late suture is usually disappointing because the proximal end has retracted and lost its elasticity, so that the gap cannot be closed by plantar flexion of the foot and flexion of the knee (in which position the gastrocnemius and soleus muscles are relaxed).

The tendon of the long head of biceps is sometimes torn where the tendon is frayed in the bicipital groove in the case of osteoarthritis of the shoulder joint (fig. 364). Recognition is easy, as on flexing the forearm the soft muscular belly is drawn downwards towards the elbow. Some hyper-

rophy of the short head partially compensates for the resulting deficiency. Efforts at repair are not always successful, and are unnecessary in elderly patients.

Rupture of the tendon of the supraspinatus is a not uncommon accident in middle-aged patients, and may occur after a trivial injury (p. 310).

The pubic attachment of the **adductor longus** muscle is sometimes partially avulsed when riding a frisky horse. Myositis ossificans may subsequently supervene. Tetanus or strychnine poisoning may cause such violent contractions that rupture of the **rectus abdominis** muscle has resulted (fig. 9).



FIG. 364.—Spontaneous ruptured long head of the biceps tendon and 'bunching' of the muscle nearer the elbow than normal.

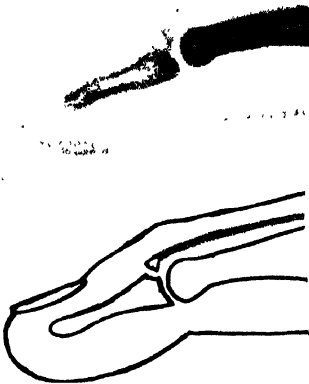


FIG. 365.—Avulsion of a flake of bone to which the long extensor tendon is attached, resulting in a 'mallet' finger.

Rupture of the **extensor longus pollicis** tendon is an occasional sequela of a Colles fracture (p. 205).

A '**mallet**' finger (syn. baseball finger) is due to avulsion of an extensor tendon of the finger, which occasionally includes a small flake of bone from the base of the distal phalanx (fig. 365). In treatment the finger is splinted in a position of right-angled flexion at the proximal interphalangeal joint, with hyperextension of the terminal interphalangeal joint, so that the central slip of the extensor tendon is relaxed. For this purpose the patient is shown how to press the

thumb against the finger-tip so that the correct position is maintained. A dry tube of plaster is then slipped over the finger and the hand dipped in warm water. The wet plaster is then moulded by the surgeon, and the finger is retained in the correct position until the plaster dries (fig. 366).

The treatment of mallet finger causes much dissatisfaction to surgeon and patient alike. From the point of view of the surgeon, the failure is to prevent recurrence of the flexion deformity of the distal phalanx which is the essential deformity. Even in recent cases and when the phalanx is held in its correct position for six weeks, the avulsed tendon often fails to unite to the bone and the deformity recurs. From the patient's point of view, the trouble is often not so much the disfigurement but the



FIG. 366.—Treatment of 'mallet' finger.

persistent pain in the affected joint. The pain disappears in a few weeks with reassurance and use and, fortunately, the deformity usually improves spontaneously after a few months. Despite this, the orthodox treatment of plaster fixation for six weeks in the fully corrected position is advised lest the patient alleges neglect. An alternative is the use of adhesive strapping renewed daily by the patient.

Long Extensor of Thumb.—This occurs most commonly in women some months, or years, after a Colles' fracture. There is a sudden inability to extend the terminal joint of the thumb. The extensor tendon is frayed in the distorted canal on the dorsal surface of the radius. The condition is well treated by transferring into it the tendon of extensor indicis proprius.

'Tennis Elbow'.—This is a very common minor malady of which the underlying pathology is still obscure, despite the fact that the condition has been recognised for many years and has often been explored at operation without any recognisable abnormality being visible. It seems to be a painful disorder of the aponeurotic fibres through which the common extensor origin is attached to the external epicondyle. No recognisable histological abnormality is present. The condition sometimes starts spontaneously, and though it takes its name from the strain commonly experienced by players, few of the numerous sufferers ever play tennis.

Clinically, the patient often complains of pain on the outer aspect of the elbow when lifting small objects by dorsiflexion of the wrist; the patient is often surprised at this, pointing out that it does not hurt to lift heavy objects with the elbow straight, e.g. buckets of coal. On physical examination there will usually be a point of localised tenderness over the external epicondyle, though sometimes the site of maximal tenderness may be more distal, lying over the radio-humeral joint-line or even over the head of the radius.

Vague pain in the arm from tennis elbow is sometimes mistaken by physicians for root pain and attributed to root pressure by a cervical disc. Local tenderness in the classical site clinches the diagnosis very simply.

Many methods of treatment have been advocated with varying degrees of success. Short-wave diathermy, repeated injections of local anæsthetic, manipulation, and even local excision of the tender area. All methods have their successes and all their failures, but in the end, after six to twelve months, one can be fairly certain that a spontaneous cure will result under any form of therapy.

The local injection of 25 mg. of hydrocortisone frequently produces a cure within two or three days. This suggests that 'tennis elbow' is one of the manifestations of 'collagen degeneration' (p. 309).

Hernia of a muscle sometimes follows a tear of the muscle sheath, the adductors and biceps brachii being the muscles most commonly affected. On contraction, muscular fibres protrude through the aperture in the sheath. This feature distinguishes a torn sheath from a torn muscle, as in the latter case a gap appears between the two portions on contraction. If disability ensues, the sheath is sutured.

A small rupture of the sheath of the *tibialis anticus* is not uncommon in athletes. The swelling appears over the belly of the muscle on the outer side of the tibia. Unless the clinician is aware of the condition, he may be puzzled by its appearance. No disability results, and the swelling disappears as life becomes less strenuous.

Displacement of tendons occasionally occurs where these structures traverse fibro-osseous canals, e.g. in the region of the wrist or ankle, or the long head of the biceps in the arm. Sudden pain occurs, followed by a sensation of weakness and further pain on attempted movements. The displaced tendon, if superficial, e.g. the peroneus longus, can be palpated in its abnormal position. The replaced tendon should be immobilised for at least eight weeks. The condition sometimes recurs, and if disability persists, the tendon is fixed in position, e.g. a flap of periosteum is raised from the bone in order to form a tunnel for the tendon. 'Snapping hip' is discussed elsewhere (p. 303).

COLLAGEN DEGENERATION IN TENDON

A new epoch in the study of pain arising in tendinous or aponeurotic structures is marked by the concept of 'collagen degeneration'. It is now recognised that tendon is composed of the chemical substance 'collagen', which possesses a specific molecular structure and that the very large and complex molecules arrange themselves in parallel fibres. These fibres can be demonstrated by electron microscopes, and they can be dissolved into a homogenous solution by chemical means and re-precipitated to re-constitute the original fibre pattern. It seems likely that localised areas of 'degeneration' of collagen can occur in tendon—especially after middle-age and at sites where tendon is exposed to great strain. This may well be the explanation of the so-called condition of 'tendinitis' which has been recognised clinically for many years in connection with the supraspinatus tendon of the shoulder. On the basis of collagen disease a number of painful conditions affecting the shoulder can be conveniently considered at this point, i.e. periarthrititis of the shoulder, supraspinatus tendinitis, spontaneous rupture of the supraspinatus tendon, calcification of the supraspinatus, and 'subacromial bursitis'.

Periarthrititis of the Shoulder (syn. 'Frozen Shoulder').—This is a common shoulder complaint in persons beyond middle-age, affecting women more often than men. It starts insidiously, or suddenly, as pain in the shoulder which is frequently worse at night. In the early stages a full passive range of movement will be present if the patient permits examination, but later the shoulder joint may become so stiff as to suggest an ankylosis. Though movement is restricted in all directions, the first to be lost is external rotation, which is soon followed by loss of abduction. Unless movement of the scapula is controlled during examination, the degree of ankylosis of the shoulder will appear less than it really is.

It seems likely that this condition starts as a patch of 'collagen degeneration' in one of the tendons of the short rotator muscles which blend intimately with the capsule of the shoulder joint. As part of the process of removal of the necrosed collagen fibres, a round-cell infiltration develops and the whole joint capsule becomes thickened and indurated. The capsule adheres to the articular cartilage of the head of the humerus like a postage stamp. The condition is quite localised, the patient in good health, and there are no systemic changes of a 'rheumatoid' nature.

The diagnosis is made by the normality of the X-ray. When he encounters his first example of this condition the student will be amazed to see a normal X-ray, expecting to see the advanced bone changes associated with osseous ankylosis. Many of these apparently ankylosed shoulders must in the past have been mistaken for tuberculosis (so-called 'caries sicca'), but nowadays the absence of destructive changes in the X-ray will immediately establish the diagnosis.

all forms of tuberculous disease of bone, joint, or tendon, obvious wasting of adjacent muscles is present. Treatment consists in general measures and the application of a

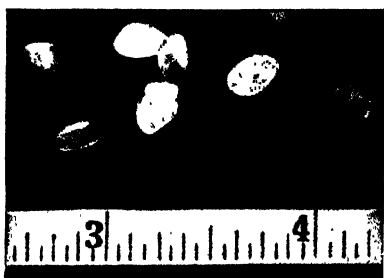


FIG. 368.—'Melon-seed' bodies from a case of compound palmar ganglion.

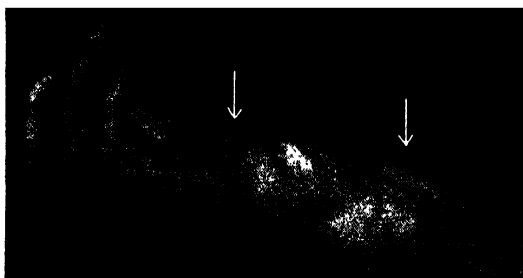


FIG. 369.—Compound palmar ganglion. The swelling in the palm communicates with the swelling above the wrist.

plaster cast, but if the condition progresses careful dissection and removal of the diseased tendon sheaths is indicated.

Stenosing teno-synovitis (or *tendo-vaginitis*) (*syn.* de Quervain's disease) is a fibrous thickening of the sheath of a tendon, and at the wrist is characterised by thickening and tenderness immediately above the radial styloid process. The abductor pollicis longus and extensor pollicis brevis, as they lie on the lower and outer aspect of the radius, are most commonly affected, especially in workers who use their thumbs excessively (e.g. charwomen wringing cloths). The condition is cured by incision of the thickened sheath under local anæsthesia, after which suture only of the skin is sufficient.

Trigger Finger.—This is a condition affecting the flexor tendons of the fingers or thumb. In adults it usually affects a single digit, but it is occasionally seen in infants, when it often affects several digits of both hands.

Most commonly it is due to a constriction of the entrance to the fibro-osseous tunnel at the level of the metacarpophalangeal joint. This constriction impresses a groove in the enclosed tendon which 'snaps' as it passes through the constriction. While the flexor muscles are strong enough to flex the finger against the trigger mechanism, the extensors are not strong enough to extend and the finger has to be extended passively with a 'click'. Sometimes a small, tense ganglion may arise in the tendon sheath to cause the trigger phenomenon by encroachment on the width of the tunnel.

The condition is easily cured by slitting the fibro-osseous tunnel at the level of the constriction.

Carpal Tunnel Compression.—It is now recognised that many of the symptoms which in the past were ascribed to the cervical rib, and the 'costoclavicular syndrome' (p. 442), are in reality the result of compression of the median nerve in the wrist at the site of the carpal tunnel. The carpal tunnel, which contains the flexor tendons and the median nerve, is formed by the concavity of the carpus being closed on the palmar surface

by the flexor retinaculum which is attached on the radial side to the trapezium and tuberosity of the scaphoid, and on the ulnar side to the pisiform and hook of the hamate (fig. 370). The clinical features of this condition are worthy of careful note because it is easily diagnosed and, what is more, is easily cured by a relatively minor surgical operation.

The patient is usually a middle-aged female and frequently has bilateral symptoms though one side may predominate. She complains of severe 'burning' pain, or severe 'pins and needles', in the hand and fingers. Characteristically this is much worse at night, while in the day it may be tolerable. The fingers on waking sometimes may feel stiff and the patient may have difficulty in tying knots or buttoning clothes while dressing first thing in the morning, but this wears off in the course of half an hour. In a minority of cases objective impairment of sensation can be detected in the three digits supplied by the median nerve. The patient may sometimes describe abnormal sensation in all the digits (i.e. including the little finger) which might suggest a more proximal lesion because the ulnar nerve lies outside the carpal tunnel. The strict localisation of *acroparæsthesiæ* to the thumb, index, middle and ring fingers, as might be logically expected from involvement of the median nerve on anatomical grounds, is not absolutely essential in the diagnosis of carpal tunnel compression, but the digit most severely affected is invariably one of those supplied entirely by the median nerve (i.e. thumb, index, or middle finger).

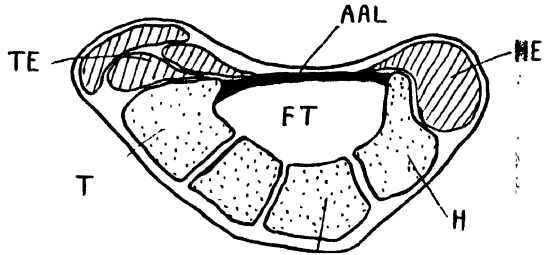


FIG. 370.—Carpal tunnel formed in the concavity of the carpus by the anterior annular ligament on its volar aspect. T, os multangulum major; C, capitate; H, hamate; FT, flexor tendons; AAL, anterior annular ligament; TE, thenar eminence; HE, hypothemnar eminence.

If the carpal tunnel is explored through a longitudinal incision on the palmar surface of the wrist, the median nerve only rarely shows obvious visible evidence of local constriction. What is commonly seen is a soft swelling of the median nerve proximal to the carpal tunnel which terminates sharply at the point where it passes under the carpal ligament. Nissen believes that in this condition the para-tenon of the flexor tendons has become bulky and thickened, and that this is the space-occupying agent which causes compression.

Because the condition is so much commoner in middle-aged women than in men one is tempted to think of an endocrine explanation, but trials of endocrine therapy have not encouraged this idea. This syndrome is quite common in young women as a temporary phase in pregnancy.

Treatment.—Whatever may be the pathology, the fact is quite definite that simple decompression of the tunnel by a longitudinal ventral incision cures the condition in an almost dramatic fashion. In a severe case the patient may be practically symptom-free in twelve hours after having had the discomfort for perhaps five years. The operation can be done in the out-patient department because no deep sutures are inserted.

A **simple ganglion** appears as a localised, tense swelling often in connection with a tendon sheath or near the capsule of a joint, and contains clear gelatinous fluid. It is a mucoid degeneration of connective tissue, and is predisposed to by injury. It is a manifestation of 'collagen degeneration'. Simple ganglia are most commonly found on the dorsum of the wrist and foot (fig. 371). They occur more commonly in females than in males and much less commonly in children or old people. Rupture of the ganglion can be accomplished by pressure, or a blow, and although recurrence is likely this simple method is worth a trial. Some cases are cured by aspiration with a wide-bore needle and injection with a sclerosing agent, followed by firm pressure for a few days. If simple measures fail, excision may be deemed necessary, but even then, to the patient's annoyance, there can still be a recurrence. Not infrequently a ganglion disappears spontaneously, so unseemly haste on the part of the surgeon is unwise.



FIG. 371.—Simple ganglion on the tendon of the peroneus brevis muscle.

Tumours of tendon sheaths occasionally occur, in the form of *synoviomias*. These tumours arise from the synovial membrane lining the tendon sheath, and are either innocent or malignant. In the past the benign synovioma was called myeloma or giant-cell tumour. In both the innocent and malignant types 'foam' cells are characteristic, which are large and spheroidal and contain numerous fat droplets.

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CUT TENDONS¹

Cut tendons are a common cause of disability after open wounds of the hand and fingers, and only too frequently receive inappropriate treatment by surgeons who have not received adequate training in this often difficult problem.

Flexor tendons are provided with a sheath, and consequently considerable retraction of the cut ends occurs when such a tendon is divided; union is therefore unlikely without surgical intervention. Extensor tendons are enclosed in loose elastic and connective tissue—the paratenon—and the severance of such tendons results in a minor degree of separation; union of extensor tendons may therefore occur if the tendon is relaxed for an adequate period, though of course direct suture is to be preferred.

Immediate repair of tendons is only indicated under the following circumstances:

(1) A competent surgeon is available, and suitable instruments and sutures are to hand.

(2) The wound must be uncontaminated, and not more than six hours should have elapsed since the accident occurred.

¹ In a book of this nature space precludes other than an abbreviation of general principles, with a short account of some common injuries.

(3) There is no loss of skin or serious damage to bones or joints.

Unless these conditions are fulfilled it is only permissible to attend to wound toilet, suture the skin, and administer penicillin in order to obviate infection. A formal operation and suture is performed when the wound has healed, usually within four weeks. A further lapse of time results in excessive retraction of the ends so that approximation is impracticable, and a tendon graft may then be required.

Flexor Tendons.—If conditions are suitable, immediate suture sometimes yields satisfactory results. If both the sublimis and profundus are divided within the digital sheath, the profundus alone is sutured, and the superficial tendon removed. Suture of both tendons inevitably results in the formation of adhesions and impairment of function. Even in expert hands the suture of a tendon is likely to fail owing to adhesions if it is performed anywhere inside the flexor sheath, i.e. anywhere between the level of the distal interphalangeal crease and the transverse palmar crease (the so-called 'danger area'). Under ideal conditions the best results are obtained by using a tendon graft (complete with its natural paratenon) attaching this distally to the distal phalanx and proximally to the cut end of the profundus tendon in the palm. In this way the flexor sheath contains no suture line.

Excepting when the flexor tendons of the fingers are divided in the 'danger area', the suture of all other tendons presents no special difficulty if ordinary surgical principles are followed.

TENDON SUTURE

Some of the important points in technique are as follows:

A bloodless field is obtained by means of a sphygmomanometer. Incisions follow the natural creases of the hand, or in the case of a digit pass longitudinally just posterior to the digital vessels and nerve. The most suitable suture material is 40-gauge stainless steel wire, which should be threaded into an atraumatic needle. If these needles are not available, the tendon can be transfixed with a hypodermic needle along the lumen of which the wire is threaded, and the needle is then withdrawn. Various stitches are used to approximate the ends of the tendon (fig. 372). Measures to prevent adhesions by insulating tendons with flaps of fascia or artificial preparations are not recommended, and reliance is placed on covering the suture line with any local tissue available.

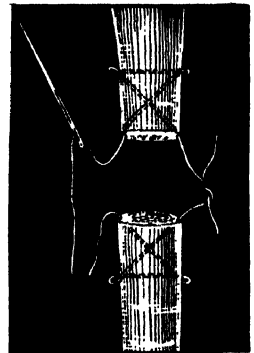


FIG. 372. — Tendon suture.

TENDON GRAFTING

This procedure is necessary if excessive retraction has occurred, if part of the tendon has been destroyed or if suture of a flexor tendon of the finger is needed in the 'danger area'. Considerable skill and experience are necessary to obtain a good result. The palmaris longus or extensor indicis are most suitable in the hand, or grafts may be taken from the long extensor tendons of the toes. The paratenon is included with the graft. Free exposure is necessary. Preservation or reconstruction of the fibrous sheaths over the interphalangeal joints is important so that there is no 'bow-stringing' of the tendon in flexion.

TENOTOMY AND TENDON LENGTHENING

Tenotomy is needed to correct deformities caused by spastic conditions of muscle or by rigid contractures. It is one of the oldest orthopaedic operations and was

originally done almost exclusively by subcutaneous puncture in order to reduce the possibility of sepsis. Subcutaneous tenotomy is used where the exact amount of elongation is not important—i.e. where the total abolition of the action of that tendon is needed.

The most important examples of tenotomy concern (1) the tendo Achillis, (2) the sternomastoid, and (3) the adductor tendons of the thigh.

The tendo Achillis needs elongation in spastic paralysis and clubfoot to correct fixed equinus deformity. This is usually done by open operation and 'Z' method of lengthening enables an exact elongation to be obtained (fig. 373) so that the 'equinus' deformity is not made into a 'calcaneus' deformity.

The sternomastoid muscle and tendon need division in some cases of torticollis. Here simple division is necessary and elongation by a predetermined amount is not

wanted. The operation can be done by subcutaneous puncture, but open operation is strongly to be advised because of dangerous structures nearby, and because tight fascial bands may need division as well as the muscle and tendon.

The adductor tendons are usually tenotomised near their attachment to the pubic bone, which is done by subcutaneous puncture with a narrow-bladed 'tenotomy' knife.

SHORTENING of a tendon is accomplished by the 'Z' method (fig. 374). The two halves of

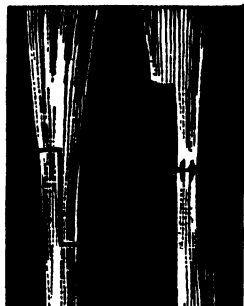


FIG. 373. — Tendon lengthening.



FIG. 374. — Tendon shortening.

the tendon, after separation, are shortened as required, and the tendon is then sutured.

TENDON TRANSPLANTATION is performed in order to restore muscular balance, or to supplement the action of ligaments (p. 350).

INFLAMMATION OF MUSCLES

Acute suppurative myositis as a primary lesion is an almost unknown condition. Collections of pus form in muscles in the course of pyæmia, but these are silent collections often mimicking a tuberculous 'cold' abscess.

'**Rheumatic Myositis**' is a misnomer but a term not easily abandoned. This is particularly the case when pain and stiffness of muscle, as in 'stiff-neck' or in 'lumbago', seems to follow exposure to draughts and dampness. Though it has for years been customary to use the term 'rheumatic myositis or fibrositis,' and even to palpate 'fibrositic nodules' in muscles so afflicted, the fact is now undisputed that no histological abnormality can be discovered in these tissues. The present tendency is swinging to the belief that most of these acute muscle pains with spasm and tenderness are probably reflex guarding mechanisms evoked by a sudden derangement of an underlying disc which has the same dermatome innervation. This would certainly seem to be the case in acute lumbago, because so often a person suffering from this condition will later develop an unequivocal disc protrusion and sciatica. Acute lumbago often occurs in young men of athletic build who after three weeks recover completely; there is no suspicion of 'rheumatism' in these cases, and the whole picture is suggestive of a mechanical derangement parallel to internal derangement of the knee.

Whatever may be the theories of causation of the 'lumbago-fibrositis-

'fibromyositis-sciatica' group of minor maladies, the best treatment still remains the same as if they were based on the older conception of 'rheumatic inflammation' locally in the muscles. Radiant heat and short-wave diathermy, counter-irritation with embrocation, aspirin and anti-rheumatic mixtures, and rest are still the most effective remedies. Widespread searches for septic foci are no longer encouraged.

Traumatic myositis ossificans occurs principally in the brachialis muscle after dislocation of the elbow, and in the quadriceps of the thigh after direct contusion (fig. 361). A much less common site is in the attachment of the adductor muscles to the inferior ramus of the pubis—'rider's bone'. When ectopic ossification occurs in other sites, e.g. round the hip-joint, it is not truly a 'myositis', but more often in the joint capsule.

This condition is much less common today than formerly, probably because the evil effects of passive stretching exercises applied to the stiff elbow after dislocation are widely known. If myositis is developing, the elbow becomes stiff and a palpable deep thickening can be detected in front of the elbow joint. X-ray will reveal a haze of new bone formation in the lateral view three to four weeks after the injury. Treatment is to apply a sling and to avoid all strain such as weight-lifting or 'deep-friction' massage. Recovery may take many months.

Generalised myositis ossificans is a rare condition, commencing in young adults, in which muscles are gradually transformed into bone. The condition usually commences in the flat muscles of the back (fig. 375), and spreads to the spinal and thoracic muscles. The condition steadily progresses, and the patient gradually becomes more rigid ('poker' man), until fatal respiratory complications supervene. Congenital absence of the last phalanx of the big toes and inability to flex the thumbs are sometimes associated conditions.

Trichiniasis is an uncommon cause of myositis in this country. Nematode worms obtain access to the alimentary canal in infected pork. After a few days the embryos find their way to striated muscles via the lymphatics. The muscles become painful and indurated, and the migration of the embryos continues for from two to four weeks, during which period eosinophilia is present. The embryos become encysted, or eventually perish and calcify.¹ The treatment is symptomatic.



FIG. 376.—Gumma of the left sternomastoid muscle. The patient also exhibited leucoplakia and fissuring of the tongue.

Syphilitic myositis may occur as a localised gumma, particularly in the sternomastoid muscle (fig. 376) or tongue. An indurated swelling appears, which gradually involves the overlying skin or mucous membrane. More rarely a diffuse myositis occurs, e.g. parenchymatous glositis, the tongue eventually becoming fibrotic.



FIG. 375.—Ossification of the flat muscles of the back. The child had characteristic deformities of the thumbs and toes. (G. D. F. McFadden, F.R.C.S., Belfast.)

TUMOURS OF MUSCLES

Innocent.—Lipomas and fibromas occasionally occur (Chapter 4).

Malignant.—Primary fibrosarcoma of a muscle sheath is not common. This tumour was formerly considered to be a simple fibroma, and it is some-

¹ The condition was discovered by Sir James Paget, 1814–1899, when he was a medical student, in the dissecting room at St. Bartholomew's Hospital.

times difficult to distinguish a fibroma from a slowly growing fibrosarcoma. Failure to realise the sarcomatous nature of these tumours accounts for Paget's description of them as 'recurrent fibroids' (fig. 60). A slowly growing, swelling appears, which is firm, circumscribed, and connected with the muscle. Exploration is necessary, and on confirmation the tumour is removed with a wide margin of surrounding muscle.



FIG. 377.—Housemaid's knee.

Even then local recurrence is all too common, and dissemination by the blood-stream, especially to the lungs or other muscles, will already have occurred in the majority of cases.

Secondary invasion of muscles sometimes occurs, e.g. the pectoralis major, following carcinoma of the breast.

DISEASES OF BURSAE

Injury.—Acute traumatic bursitis follows injury and may also follow unaccustomed exercise, e.g. inflammation of the bursa under the tendo Achillis after a cross-country run.

Chronic bursitis is the result of repeated slight injuries, or constant pressure, to *anatomical bursæ*, e.g. housemaid's knee (distal part of patella) (fig. 377), student's or miner's elbow (olecranon) (fig. 378),

weaver's bottom (tuber ischii).

Enlargement of the *semimembranosus bursa* is common in children (fig. 379). If disability results, aspiration is performed, but most cases disappear spontaneously.



FIG. 379.—A semimembranosus bursa, tense in extension and flaccid in flexion.

Semimembranosus bursæ, if they communicate with the knee joint sometimes enlarge as a result of such conditions as osteoarthritis of the joint.

A *Baker's cyst* is an overflow of synovial fluid from an osteo-

arthritic knee joint through a herniation of the synovial membrane through the capsule, which occurs along the vessels which penetrate the posterior ligament of the knee joint.



FIG. 378.—Chronic olecranon bursitis.

Adventitious bursæ form as a result of prolonged pressure over bony prominences, e.g. Billingsgate¹ hump, due to pressure of a fish-basket over the seventh cervical spinous process. The term 'adventitious' means that no anatomical bursa was present at the site of the newly

formed cyst, and that it was generated in connective tissue as a result of

¹ Billingsgate—London's fish market.

repeated friction. One of the commonest of these is the 'bunion' on the big toe.

Infection.—ACUTE SUPPURATIVE BURSTITIS is due to direct infection by penetrating wounds, or from local subcutaneous infection and involvement of the prepatellar bursa. In the case of prepatellar bursitis, a 'sympathetic' effusion into the knee joint sometimes follows, but confusion with infective arthritis should be avoided, as in the latter condition any attempt to move the joint is painful, and pain is elicited by pressure in the popliteal space. The infection usually responds to chemotherapy, but if pus is already present drainage will be necessary.

CHRONIC INFECTIVE BURSTITIS can be pyogenic, tuberculous, or, rarely, syphilitic. One very common type of chronic bursitis needs special mention because it has been given the special name of a 'bunion.' This is an 'adventitious' bursa produced by the friction of a shoe against the prominent head of the first metatarsal bone in 'hallux valgus' (p. 346). At first sterile, this bursa later becomes infected, and thereafter runs a recurrent course in which it flares up, discharges pus, settles down, and again flares up. Treatment in an acute attack is by local heat, rest, and antibiotics, but the prevention of further attacks can only be by the radical cure of the deformity by operation in a quiescent phase.

Tuberculous bursitis resembles tuberculous tenosynovitis, in that two varieties occur: in one the effusion contains 'melon-seed' bodies; in the other the bursa becomes lined with granulation tissue, may progress to abscess formation, and eventually fistulæ. The *gluteal bursa* between the insertion of the gluteus maximus and the great trochanter is particularly prone to tuberculous infection. In some cases as time passes a tuberculous focus declares itself in the great trochanter. Tuberculous *subacromial bursitis*, containing melon-seed bodies, may indicate disease of the humeral head, and a tuberculous arthritis of the shoulder may occur later. A tuberculous bursa should be completely excised, an operation which often requires considerable patience on account of ramifications and loculations of the bursa.

Syphilitic bursitis occurs during the secondary stage, and gives rise to transitory, and often symmetrical, effusion. During the tertiary stage, a local gumma or a diffuse gummatous bursitis are uncommon manifestations.

New-growths of Bursa.—An endothelioma from the lining membrane, or fibrosarcoma from the wall, occur as curiosities.

CHAPTER 16

DEFORMITIES

JOHN CHARNLEY

DEFORMITIES can be *congenital* or *acquired* in origin, and *fixed* or *mobile* in type. *Fixed* deformities are those which cannot be corrected by gentle persuasion exerted by the examining hands, whereas *mobile* deformities can be so corrected.

The wide range of deformities can be reviewed by considering the pathological changes in the anatomical structures responsible for these deformities :

(1) **Skin and Subcutaneous Tissues.**—*Burns* are common causes of fixed deformities when they produce scars across the flexor aspects of joints. *Dupuytren's contracture* is a spontaneous contracture affecting the fibrous tissue of the palmar fascia.

(2) **Muscles and Tendons.**—*Ischæmic contracture* of muscle following trauma or embolism results from the contraction of scar tissue left after necrosis of the contractile substance of the muscle belly. *Infantile paralysis* (poliomyelitis) is often followed, in untreated cases, by contracture of the muscle groups no longer opposed by normal muscles.

(3) **Joints.**—(a) Congenital deformities, such as congenital dislocation of the hip, club-foot, congenital absence of limbs or parts of limbs.

(b) Ankylosis following arthritis (i.e. septic, tuberculous, and rheumatoid arthritis).

(c) Traumatic dislocations if unreduced.

(d) Hysterical deformities are produced by the patient holding the joint in an abnormal position for many months.

(4) **Bones.**—(a) Congenital and familial errors of growth (i.e. achondroplasia, fragilitas ossium, absence of bones, etc.).

(b) Metabolic diseases of bone (i.e. rickets, renal rickets).

(c) Fibrous dysplasias of bone (Paget's disease, osteitis fibrosa cystica).

(d) Irregular growth at epiphyses due to trauma or disease.

(e) Malunited fractures.

(5) **Nervous Lesions.**—Spastic deformities due to Little's disease or hemiplegia in later life. Flaccid deformities due to poliomyelitis or peripheral injury of nerves (both followed later by contracture of unopposed groups, i.e. claw-hand after injury of both median and ulnar nerves).

Consideration of the above causes will suggest that deformities are often preventable, and that in some cases operations would be rendered unnecessary if care and foresight were used in the early treatment ; this is especially the case in acute anterior poliomyelitis. The following summary indicates the surgical procedures which may be adopted for the correction of deformities :

(i) Manipulation with retention, such as the daily correction of a club-foot by moulding it into the over-corrected position assisted by a splint.

(ii) Operations on soft parts, such as fasciotomy, tenotomy, or tendon transplantation.

(iii) Operations on joints, as by performing arthrodesis of the tarsus for a flail ankle joint.

(iv) Operations on bones, either osteotomy, excision, or amputation.

Baron Guillaume Dupuytren, 1777-1836. Surgeon, Hôtel Dieu, Paris.
Sir James Paget, 1814-1899. Surgeon to St. Bartholomew's Hospital, London.
William J. Little, 1810-1896. Physician to the London Hospital.

The following are some of the factors which require consideration in deciding whether intervention is advisable in any given deformity. Age is of obvious importance and, as a general rule, the older the patient the less need arises for correction. Patients often adapt themselves to a long-standing deformity in a remarkable manner, and in such cases anatomical correction may interfere with function. A well-known example of this is seen in the fixed equinus deformity of the foot often encountered in extensive paralysis of the lower limb by poliomyelitis; not only does this equinus make up for loss of leg length, but it may mechanically assist the action of a weak quadriceps muscle to hold the knee in extension and help a patient to walk without the need of a caliper splint.

TORTICOLLIS (*syn.* WRY NECK)

It is customary to classify torticollis under the headings of congenital or acquired, but the condition commonly called 'congenital' torticollis is now known to be acquired as a result of trauma during childbirth. True congenital deformities of the bones of the cervical spine are commonly encountered (hemivertebrae, congenital fusions, congenital short-neck, etc.), but these rarely produce a torticollis as the main feature of the deformity.

Torticollis results from the following causes:

(1) **Acute Rheumatic.**—This is due to fibrositis following exposure to cold or a draught, e.g. a chilly drive in a draughty car. The onset is sudden, and muscles are tender on pressure. Radiant heat and salicylates hasten recovery. It is probable that the so-called 'acute rheumatic torticollis' is protective spasm covering an acute protrusion of a cervical intervertebral disc, but this does not mean that 'old-fashioned' treatment on the basis of 'fibrositis' is in question, nor that the patient need be alarmed by any mention of disc protrusion unless simple remedies have failed to produce a cure after three or four weeks. It is very easy to convert a patient into a confirmed neurotic by talking glibly about 'discs out of place' in the neck.

(2) **Spasmodic.**—This distressing condition, fortunately uncommon, occurs chiefly in middle-aged neurotic females. It is characterised by clonic spasms of the sternomastoid and trapezius muscles on one side of the neck, and later the deep cervical muscles on the opposite side may be affected. The head is continually jerked downwards towards one shoulder, particularly during excitement, and mental instability may be an associated condition. Treatment is unsatisfactory, but every effort is made to discover any functional cause or source of peripheral irritation. Division of the spinal accessory nerve on one side, and of the opposite posterior primary divisions as they lie in the semispinalis cervicis, is sometimes necessary, but even this procedure is not always successful, as the lesion, whatever it may be, would seem to be at a high level in the cerebrum.

(3) **Inflammatory.**—This may be due to parotitis, acute adenitis of the cervical lymph nodes, or Pott's disease of the cervical spine.

(4) **Hysterical.**—Torticollis is an occasional manifestation of hysteria, and there will usually be other bizarre features of the cases suggesting this diagnosis. Some clinicians consider 'spasmodic torticollis,' (2) above, as an hysterical condition.

(5) **'Congenital.'**—This is a condition which only renders itself noticeable in children between the ages of five and seven years. It is now believed that these children have all demonstrated a sternomastoid 'tumour' during the early weeks immediately following birth. This is an oval swelling the size and shape of a hazel nut, lying with its long axis in the length of the affected sternomastoid muscle (fig. 380). It is the result of stretching of the muscle fibres during birth, and has the same pathology as the Volkmann's ischæmic contracture in which necrotic muscle fibres are replaced by granulation tissue, which on bisection of the lump is seen as white fibrous

tissue which later contracts and abolishes any normal elasticity of the muscle belly. In torticollis the latent interval between the injury at birth and the first appearance of the deformity is explained by the fact that between five to seven years of age the child's neck starts to elongate during growth, so that from being an infant with an almost imperceptible neck it starts to take on the adult length. The inextensibility of the affected sternomastoid muscle thus causes the head to be drawn to the affected side. In the untreated case the deformity can become extreme, so that in adult life the ear on the affected side may be in contact with the tip of the shoulder. Even



FIG. 380.—Sternomastoid 'tumour'.

if left untreated for only a few years, asymmetrical growth changes will start in the face, and these will interfere with a perfect cosmetic result. The asymmetrical changes in the face are probably due to the head adapting its position so that the eyes will both work on the same horizontal plane. In later stages the cervical vertebræ will take on permanent wedge-shaped deformities.

Treatment.—If a sternomastoid 'tumour' has been recognised soon after birth, the parents should be instructed to have the child kept under careful supervision during subsequent years and to see that the child can stretch the affected muscle fully (i.e. the child should be able to look upwards and over the shoulder on the affected side—rotation of the chin to the affected side combined with extension of the neck).

If the contracture develops, treatment by tenotomy gives highly gratifying results.

After operation it is advisable to fix the head and neck in an over-corrected position in plaster for three or four weeks (fig. 381). It is usually necessary to attend a physiotherapy department for a few weeks, in order to get the little patient's confidence in active exercises designed to keep the affected muscle stretched.

It has been suggested that a sternomastoid tumour should be excised, as soon as it is recognised, to prevent a contracture developing later, but a final decision on this policy cannot yet be given.



FIG. 381.—Operative treatment of congenital torticollis.

SPINAL DEFORMITIES

Scoliosis is defined as lateral curvature of the spine. In many cases the lateral curvature is complicated by a rotational deformity which, in the thoracic region, is transmitted to the ribs, producing asymmetry of the thorax. Frequently the deformity of the ribs is one of the most striking aspects, producing, in addition to the scoliosis, an appearance of kyphosis (kyphoscoliosis). In very severe cases the vital capacity of the chest is so seriously diminished that the patient usually succumbs to intercurrent infection before

adult life. Sometimes the scoliosis may compress nerve roots and cause severe neuralgic pain or press on the cord on the concave side and produce spastic hemiparesis.

The causes of scoliosis may be classified:

(1) *Congenital*.—This is the result of a congenital hemivertebra, which in the thoracic region is confirmed by co-existing congenital anomalies of the associated ribs (fig. 382).

(2) *Postural*.—This is a type of lateral curvature which affects children of school age and is characterised by being a simple C-curve without rotation of the vertebræ and with the deformity fully mobile and correctable. It is seen now much less commonly than formerly, probably because of school gymnastics and the avoidance of bad postures when sitting at school desks. The treatment of this type of case is obviously by gymnastics and lessons in deportment standing in front of a mirror.

(3) *Paralytic*.—This is the scoliosis which accompanies extensive paralysis from acute anterior poliomyelitis. Some of the most severe cases of scoliosis ever to be encountered are in this group. Generally the paralysis is so widespread that, in addition to the scoliosis, the patient will have partial paralysis of both legs and walk only with the aid of crutches and double leg calipers.



FIG. 382. — Lumbar scoliosis: hemivertebra of L.5 with spina bifida occulta.

The treatment of paralytic scoliosis presents the orthopædic surgeon with what is sometimes an insuperable problem. Prolonged bed rest and avoidance of early weight bearing is a preventive measure when the weakness of the spinal muscles has been detected, but obviously a time will come when the young person must be allowed to be up and about. The fitting of a spinal brace may help considerably to minimise the development of deformity. In cases where the erector spinæ has been completely and permanently paralysed, it is

doubtful if anything can be done to prevent considerable deformity even while under treatment. So great are the bending forces exerted by the superincumbent body weight that even spinal fusions over large areas of the spine will bend or crack under the strain.

(4) *Spastic*.—Following unilateral spasm of the spinal muscles, e.g. syringomyelia (p. 284).

(5) *Pulmonary* conditions will produce a scoliosis when disease of one lung results in contraction of the chest wall on that side. In the past the worst examples followed from the treatment of empyema by prolonged drainage through tubes in the chest wall, but the modern treatment of these suppurative conditions of the pleura has made this complication one of the rarer causes of scoliosis. Extensive thoracoplasties with complete collapse of one half of the

chest may cause a slight deviation of the spine, but not usually to such a degree as to produce a clinically detectable deformity of the spine.

(6) *Idiopathic Scoliosis*.—Though of unknown origin, this is the commonest scoliosis, and next to the paralytic the most severe. In this serious condition the experienced orthopædic surgeon can prevent a patient becoming a helpless cripple and 'hunchback' and maintain a condition of the spine which will appear comparatively normal to the lay observer. It is to be noted that the emphasis here is laid on the maintenance of an acceptable condition of the spine; in other words, early diagnosis and early treatment are the fundamental aspects of this problem. The orthopædic surgeon presented with a case of advanced deformity can do nothing to restore the cripple to normal, but his knowledge of the natural history of the development of these gross deformities may enable him to check the deformity in early cases.

Idiopathic scoliosis is commoner in girls than in boys. It does not usually present itself until the child is about ten years of age, but from then until puberty a rapid and sometimes appalling deterioration takes place. The deterioration may thereafter continue at a slower rate until the maximum deformity is achieved at about the age of adolescence. Much can be done for these unfortunate patients if the condition is diagnosed between ten years of age and early puberty, but if missed at this crucial time the orthopædic surgeon can alleviate only a limited number of cases.

The fundamental nature of idiopathic scoliosis is still obscure. It has often been attributed to a paralysis of the intrinsic muscles of the spine by a condition such as poliomyelitis, but the preponderance of girls (poliomyelitis affects both sexes equally) and the absence of paralysis in the extremities are against this. It is, in fact, the complete normality of the rest of the body which distinguishes this condition from the scoliosis of poliomyelitis. There is a possibility that there may be some obscure neurological cause underlying it, as it is sometimes seen in Friedreich's ataxia and in von Recklinghausen's disease (neurofibromatosis).

Deformity.—In order that the body may remain in balance, the *primary* curve of the scoliosis is balanced by secondary curves above and below it. These compensatory curves bring the shoulders over the pelvis and, when the patient is fully clothed, no particular abnormality will be visible. The main effect of a severe scoliosis is (a) loss of height due to the spine becoming 'concertinaed'; this may account for a loss of three inches to six inches (7·5–15 cm.) of height, according to the severity of the deformity, and (b) the presence of a 'rib hump', which gives the patient the appearance of a 'hunchback' in addition to being a little dwarfed. This rib hump is very characteristic of idiopathic scoliosis, and is best demonstrated in an early case by standing behind the unclothed patient and asking her to touch her toes. Even in the absence of any very marked clinical deviation of the spine, this test will show the asymmetry of the chest (fig. 383). This simple test is of great importance because the deformity of the spine discovered by X-ray is often much greater than other clinical tests would lead one to suppose. The reason is that the rotation of the vertebral bodies, which always accompanies the lateral curvature in idiopathic scoliosis, is such that the tips of the spinous processes turn towards the midline (so diminishing the apparent

curve), and the bodies turn away from the midline, so increasing the curvature (and these, being intrathoracic, are not visible until X-rayed). The presence of a rib hump, rendered more clearly manifest in flexion, will therefore lead the surgeon to suspect rotation, and that the clinical deformity, as demonstrated by marking out the tips of the spinous processes, is concealing a more serious radiological appearance.

Treatment.—The ideal treatment of idiopathic scoliosis is early diagnosis and prevention. So rapidly can the deformity progress once it has started, that the aim of treatment should be frequent surveillance during the year after the deformity has first been noticed. Comparable anteroposterior X-rays of the spine should be taken every three months during the first year, and careful measurements of these curves will show whether the deformity is increasing. It is valueless to depend on clinical impressions of what the spine looked like. Without this careful supervision it may sometimes happen that, within a year of the curve being noticed, a deformity has developed which may be too severe for correction.

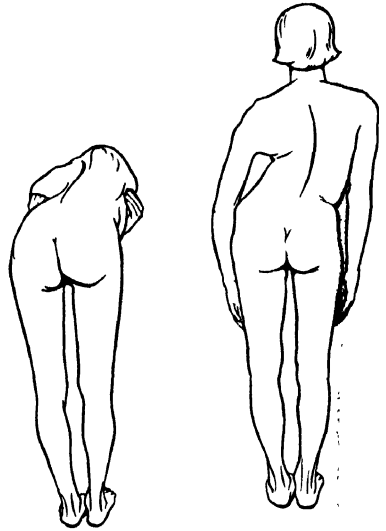


FIG. 383. — Scoliosis showing 'rib-hump' (rotatory deformity) revealed in flexion.

It is true that all cases of idiopathic scoliosis do not behave alike. There are certain types which are benign in their untreated natural history (many of these occurring in the lumbar region). On the other hand, there are others of a most sinister character (especially those in the thoracic region), and until more is known of how these can be distinguished the routine of careful surveillance by X-ray is to be advised in all cases. This aspect of the problem is one which favours the concentration of these difficult cases in centres which specialise in the treatment of idiopathic scoliosis.

In most of the severe cases a rapid deterioration of the shape of the spine takes place in the two or three years prior to puberty, but thereafter no further deterioration is likely. Thus a child presented for examination at puberty with only a mild curvature can be reassured, but she should be checked by comparable anteroposterior X-rays on several occasions.

In all these mild cases the importance of exercises while under surveillance will be manifest : exercises to encourage bending to the side of the convexity and rotation exercises in the appropriate direction to de-rotate the spine. The child should be instructed to sleep on the side of the concavity and not to carry loads on this side.

If the deformity has reached a degree to be easily visible to the lay person, and if there is a strong suspicion that the curvature is of the serious kind which will increase before puberty is reached, there is no point in adopting inefficient conservative methods which may waste time and allow a

deformity to develop. These cases are best corrected by orthopædic apparatus, after which the corrected position should be maintained by spinal fusion.

Technique.—The most widely used method of obtaining correction is by the use of the Risser jacket (fig. 384). This is a plaster body cast, including the head and one thigh, applied with the patient bent to the side of maximum correction which can be tolerated. The cast is heavily padded with sorbo rubber at the essential points where experience has shown that pressure is likely to be encountered. When the cast is dry it is sawn across at the level of the apex of the primary curve and angulated (taking out wedges if necessary). The new position is held by means of metal hinges incorporated with the plaster to hold the two halves together. A very slow correction can then be obtained from week to week, and checked by X-ray. When the primary curve has been corrected as far as the surgeon thinks desirable (and this is often not a complete straightening of the curve), a hole is cut through the back of the plaster



FIG. 384.—Risser jacket.

over the region of the scoliosis and the operation of spinal fusion is performed through this window. The bone used is generally taken as small chips from the patient's own tibia, and the volume can be increased by adding 'banked bone'. Sometimes, in a severe case, the operation is performed in several stages so that a long length of spine

is fused on both sides of the spinous processes. After operation, six months are necessary for consolidation of the fusion, and another year of ordinary activities in carefully moulded corset is advisable so that the soft new graft is not subjected to severe bending strains.

Milwaukee Brace.—This is an ambulatory splint applied to the trunk which carries an extension device to the head. It is used as a prophylactic method in mild cases, or as the last splint after a spinal fusion.

Internal Jacking Systems.—Attempts have been made to bury metallic screw-jacks, or compression springs, to exert corrective forces directly on the affected spinal segments. These are still under trial.

Unilateral Epiphysectomy.—Attempts have been made, with variable success, to destroy the growing epiphyses at the affected segments of the spine, on the convex border of the curve.

✓ Kyphosis

Kyphosis can be defined as an increased dorsal convexity of the spine. The deformity of kyphosis lies in the anteroposterior plane (unlike scoliosis, which is in a lateral plane), and it produces a flexion deformity of the spine whether it occurs in a region which is normally convex (i.e. the thoracic spine) or in a region which is normally concave (i.e. the lumbar or cervical spine).

Three clinical types of kyphosis are encountered :

(1) '**Knuckle**'.—This is nothing more than the undue prominence of a single spinous process. The general curvature of the spine as a whole is normal, and is merely interrupted by this prominent knuckle (fig. 385 (a)). Sometimes such a prominence may be of no significance, being only a congenital irregularity of the spinous processes. When due to disease, it indicates the collapse of a single vertebral body.

(2) '**Angular**'.—This is a sharp angular break in the line of the spine, so that the parts above and below no longer form part of a simple curve (fig. 385 (b)). This type of kyphosis always implies severe collapse of at least two, and often three, vertebral bodies.

(3) **Round.**—This is usually seen in the thoracic region as an increase in the normal convexity of this region, and it indicates that the increased wedging of the vertebræ responsible for it is distributed more or less equally over five to eight thoracic vertebræ.

The causes of kyphosis can be classified as :

(1) *Traumatic.*—Severe fracture-dislocations of the spine will produce a marked *angular* kyphosis ; mild compression fractures may be detectable as a knuckle kyphos.

(2) *Inflammatory.*—Of these, only Pott's disease produces kyphosis. Tuberculous caries of the spine in its earliest stages will be suspected if there is rigidity of the spine on attempted forward flexion, and especially if there is a knuckle kyphos. The later stages of the disease may develop angular kyphosis if two or three bodies are destroyed. In late healed cases the presence of old sinus scars may clinch the diagnosis of Pott's disease.

(3) *Neoplastic.*—Secondary carcinomatous deposits occur with great frequency in the red marrow of the vertebral bodies, but it is unusual for a kyphos to develop. More often the patient will succumb to paraplegia or from the effects of other secondary deposits elsewhere before sufficient collapse takes place to cause a marked deformity.

(4) *Osteochondritis (syn. Scheuermann's Disease).*—This is a disorder of the epiphyses in rapidly growing children (cf. p. 250—Perthes disease, Osgood-Schlatter, etc.) which affects the vertebræ in the thoracic region. It is the commonest cause of 'round shoulders' in otherwise healthy young people. It produces a marked increase in the normal convexity of the thoracic spine which is compensated by an increase in the concavity (lordosis) of the lumbar spine below it.

The disease often starts with pain in the back at puberty, and the condition can progress until about the age of eighteen. Lateral X-rays of the thoracic spine show some irregularity of the anterosuperior and antero-inferior angles of the vertebral bodies at the site where the normal epiphyses are present. Eventually the vertebral bodies from about the second to the tenth will show excessive wedging.

If recognised in the early stages, a period of three months on a plaster bed with a hinge to correct the deformity, followed by a posterior spinal support to brace the spine, is a logical order of treatment. In practice, such a drastic curtailment of school activities is rarely necessary, and if the patient concentrates on hyperextension exercises, has a daily regimen which increases the number of hours he lies flat on his back, and reduces the spare time left for 'lounging about', this is as much as is usually necessary to control the condition. A well-fitting back-brace is permissible, provided that it does not take the place of the exercise. A large part of the external deformity of 'round back' is attributable to a bad position of the shoulders, and many young people can conceal the fixed deformity of the spine by developing a good

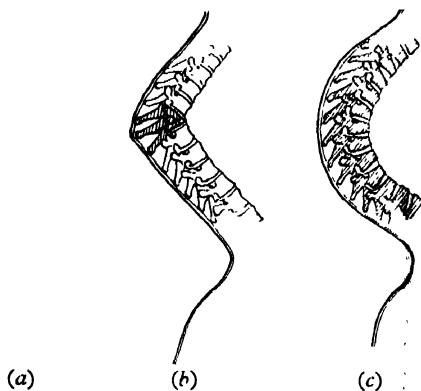


FIG. 385.—Types of kyphosis.
(a) Knuckle. (b) Angular. (c) Round.

carriage with shoulders well braced back. It must be confessed that the most elaborate and time-consuming mechanical treatment of the deformity, even in its early stages, may sometimes fail to produce a perfectly normal spine, and therefore the development of a good carriage is in the end the most important part of treatment.

(5) *Ankylosing Spondylitis*.—The characteristic deformity of this condition is a rigid flexion of the whole spine from the upper cervical to the lower lumbar region. It is not a true kyphosis in the sense of a localised increase in dorsal convexity at one part of the spine, but is merely the abolition of the normal concavities in the lumbar and cervical regions (p. 296).

(6) *Paget's Disease*.—An increase in the curvature of the thoracic spine is common in generalised Paget's disease, and accounts partly for the patient's loss in height. In this condition the bones are invaded by fibrous tissue and, though in many cases they appear radiologically denser than normal, they are at the same time brittle and also capable of slow bending under body weight.

(7) *Senile Kyphosis*.—As a normal feature of old age, the thoracic spine develops an exaggerated convexity which explains the loss in height which is so characteristic of old age. The underlying pathology is that of a disuse atrophy of the vertebral bodies. There is no pain which might bring the patient for treatment, and therefore it does not rank as a disease.

(8) *Acute Osteoporosis of the Spine*.—This is very similar to senile osteoporosis in its general appearances but occurring in patients of fifty to sixty years of age. It is almost always encountered in females, and if occurring rather earlier is called 'post-menopausal' osteoporosis of the spine. This

condition seems to be related to the failure of some ovarian secretion, because great relief from symptoms can frequently be obtained by the administration of dienestrol which these patients can usually take over long periods without causing uterine bleeding. There is rarely much re-ossification of the spine, but the symptoms are held in control.

In severe cases a characteristic radiological appearance of 'fish vertebrae' is seen. This is caused by the bone of the vertebral bodies being too soft to resist the internal pressure of the intervertebral discs which thus tend to assume a biconvex, or even spherical, shape (fig. 386).

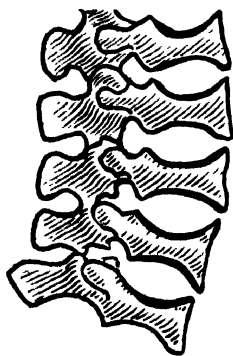


FIG. 386. — 'Fish' vertebrae in severe osteoporosis of the spine.

Strictly speaking, acute osteoporosis of the spine should not be considered here under the heading of kyphosis because it does not usually cause this deformity. It manifests itself by pain and rigidity of the back. It is probably, however, related to senile osteoporosis and cannot be considered in relation to 'general diseases of bone' because it is essentially a local condition affecting the spine alone.

Lordosis is an exaggeration of the dorsal concavities of the spine and is most commonly encountered in the lumbar region. If for any reason, such as Pott's disease, a kyphosis exists in the dorsal region, then a compensating lumbar lordosis will develop to restore the centre of gravity of the body. In patients with gross abdominal obesity the lumbar spine becomes lordotic to

restore equilibrium, and the same happens as a transitory mechanism during the later stages of pregnancy.

Spondylolisthesis.—This is a condition in which a lower lumbar vertebra, usually the fifth, slips forward through the plane of the intervertebral disc below it and so carries with it the whole of the upper portion of the spine. The essential lesion is a separation of the body of the vertebra from the posterior articulation, lamina, and spinous process as a result of a defect in the pedicles which hold these two parts of the vertebræ together (fig. 387). Because the lamina is left behind in its normal position, the forward displacement of the vertebral body does not narrow the spinal canal. The deformity can, however, cause root pressure which may manifest itself as sciatica.

In general, the main symptoms are usually those of long-standing low-back pain. On examination, in a mild case nothing will be found other than some excessive prominence of the first sacral spinous process. In a severe case, where the lumbar vertebra has become dislocated in front of the sacrum, there will be a severe lordosis and a rather characteristic shortening of the trunk, so that the lower ribs seem to rest on the brim of the pelvis.

Though trauma was frequently regarded in the past as a common cause of spondylolisthesis, accurate X-ray studies now show that a congenital defect in the development of the pedicles is much the commonest explanation. X-ray studies often reveal a condition sometimes known as 'prespondylolisthesis', or 'spondylolysis', where slipping of the vertebræ is absent, or minimal, in the presence of defects in the pedicles.

The majority of these cases never require any special treatment other than simple placebos for backache, such as local heat and rest from time to time. A stiff lumbosacral corset will give relief to the majority, but a few will require lumbosacral fusion to abolish pain. As in the management of any case of 'low-back pain', it is unwise to alarm the patient with injudicious reports about the X-ray appearance. Many cases of severe spondylolisthesis go through life with nothing more than an occasional attack of 'lumbago'. Injudicious remarks can convert a mild case into a complete invalid.

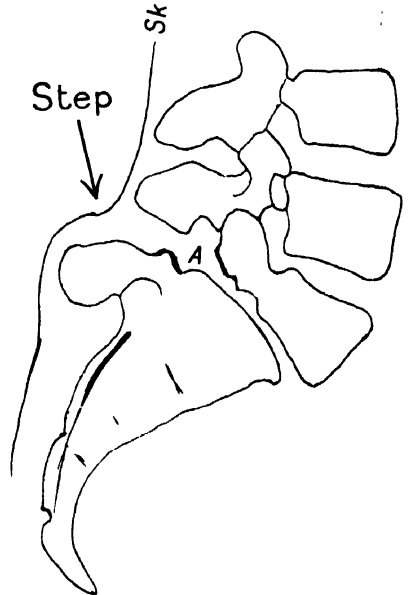


FIG. 387.—Spondylolisthesis at the level of L5 — S1 disc. Note the defect at A and the step over S1 spine.

DEFINITION OF VALGUS AND VARUS

The terms valgus and varus, so frequently used in describing orthopædic deformities, often puzzle the student. Essentially valgus means 'knock-knee' and

varus 'bow leg', and it is difficult to see how these terms could be applied, for instance, to the hip or the foot.

Whatever the level of the deformity, its effect should be considered on the displacement of the end of the extremity towards or away from the midline of the body. Varus deformities are those which displace the hand or foot *towards* the midline; valgus deformities *away* from the midline. In knock-knee (genu valgum) it is not the knee itself which is displaced medially, it is the foot which is displaced laterally.

UPPER EXTREMITY

Cubitus Valgus.—Normally the forearm lies in a slightly abducted position in relation to the axis of the humerus (the 'carrying angle'). This varies from about 10 degrees in men to 15 degrees in women (because of the greater width of the female pelvis in relation to the shoulders). Fractures of the external condyle of the humerus in young children frequently fail to unite, and continued growth of the medial condyle in later life produces the condition of cubitus valgus, where the forearm may be abducted 45 degrees in relation to the axis of the humerus. In this position the ulnar nerve suffers excessive friction at the medial condyle, and an interstitial neuritis develops which produces anæsthesia and an insidious paralysis of the small muscles of the hand supplied by the ulnar nerve. If recognised early, transposition of the ulnar nerve in front of the medial epicondyle will avert the further development of symptoms, but it produces little improvement if serious wasting of the intrinsic muscles has already developed.

Cubitus varus is a less common deformity ('gun-stock' deformity) due to mal-union of supracondylar fractures. It is an ugly deformity but does not interfere with function, and its correction by osteotomy is dictated entirely on cosmetic grounds.

Madelung's deformity (manus valga) is a radial displacement of the carpus with abnormal prominence of the lower end of the ulna. It is a term for a deformity and comprises many ætiological conditions. Anything arresting the growth of radius will produce the deformity. It is usually symptomless and only rarely does the prominent lower end of the ulna need resection.

Congenital absence of the radius occasionally occurs, in which case growth of the ulna pushes the hand to the radial side. The lower articular surface of the ulna is expanded, and articulates with the proximal row of carpal bones. This is a form of Madelung's deformity.

Congenital dislocation of the head of the radius sometimes causes great confusion if the surgeon is unaware of the condition. Dislocation of the head of the radius may be diagnosed radiologically for the first time after an injury, though it has actually been present for many years. Attempts to reduce it will, of course, fail. It can be diagnosed by the fact that the head is not quite normal in shape (not being cup-shaped at its extremity) and that it is longer than it should be for a simple dislocation.

Congenital elevation of the shoulder (*syn.* Sprengel's shoulder) is a condition in which the scapula is smaller than normal and situated at a higher level. The inferior angle is rotated inwards, and abduction is restricted (fig. 388). In bilateral cases the appearance of the patient at first suggests that the neck is abnormally short. In some cases this is true as a result of congenital fusion of cervical vertebræ. The



FIG. 388. — Sprengel's shoulder. (Sir John Fraser.)

homboid muscles are partially fibrous or even cartilaginous or ossified, and the trapezius and serratus muscles are sometimes deficient.

Treatment consists in exercises if such are considered necessary, but surprisingly little disability results. Operations to improve function are unsatisfactory, but the upper and inner portion of the scapula may be excised for cosmetic improvement.

Dupuytren's Contracture.—This is a localised thickening of the palmar fascia which involves the overlying skin of the palm and which shows a strong tendency to contract and eventually draw the affected fingers into rigid flexion. Most commonly it starts near the base of the ring finger and soon draws that finger into the palm of the hand (fig. 389). Later it involves the little finger in the same way. In long-standing cases permanent changes take place in the metacarpophalangeal joints and the proximal interphalangeal joints which render futile any attempts to straighten the fingers.

The pathology of the condition is unknown; the consensus of opinion would seem to favour a chronic inflammatory origin, but in all probability it will prove to be a primary disorder of the 'collagen' substance of fibrous tissue. In the past it was customary to attribute it to repeated trauma in persons using tools, etc., which pressed on this region. On the other hand, it is known to be familial and is frequently seen in persons who have never done a day's manual work in their lives. The fact that it is so often bilateral is against any traumatic origin. It is distinctly more common in men than in women.



FIG. 389.—Dupuytren's contracture. Note puckering of adherent skin in palm.

Treatment.—Early cases can be treated by night splintage and gentle stretchings done by the patient. On the other hand, the most perfect surgical cases are those which are operated on before serious contracture develops, and radical excision of the affected area is followed by quicker rehabilitation in the early cases than in the later cases.

In the past the operation of 'subcutaneous fasciotomy' was often practised; through puncture wounds the tight bands were divided and the finger partially straightened. There is nowadays very little need for this procedure, and radical excision can give very excellent results in all but the severe cases where permanent joint changes have developed. In the latter it may be advisable to amputate.



FIG. 390.—Syndactyly.

Congenital deformities of the fingers include the conditions of syndactyly, macrodactyly, and congenital contracture. *Syndactyly*, or webbed fingers, is a condition in which two or more fingers are joined together (fig. 390). An X-ray should be taken, and if normal bones are present, separation of the fingers will improve the function of the hand (fig. 391).

Syndactyly of the toes needs no treatment, and may even benefit swimmers!

Macroductyly consists in overgrowth, possibly enormous, of a digit. A plastic operation, or amputation, is occasionally necessary.

Congenital contracture of the little finger is a condition not encountered in the other digits. Hyperextension occurs at the metacarpophalangeal joint, and flexion at the proximal interphalangeal joint (fig. 392). The condition is usually bilateral and does not produce symptoms. It is easily differentiated from Dupuytren's contracture as there is no skin involvement and it usually occurs in young people, whereas Dupuytren's contracture is rarely seen under middle-age. Treatment is seldom necessary.



FIG. 391.—Syndactyly. The fingers are divided by means of a modified 'Z' plasty. There is insufficient skin to bridge the gap totally; the defect is therefore covered with a split thickness graft to both fingers. (Simple division with 'Z' plasty will nearly always result in scar contracture across each interphalangeal joint.)

DEFORMITIES OF THE LOWER EXTREMITY

Congenital Dislocation of the Hip.—

Though this is a comparatively rare condition in England it is an extremely important matter to the orthopaedic surgeon because, by early diagnosis and skilful treatment, children may grow into complete normality who otherwise would go through life seriously crippled.

It would seem that, as the result of some gene, the embryo affected in this way develops with the head of the femur at some slight distance from the socket, and though the head of the femur and the acetabulum



FIG. 392.—Congenital contracture of the little finger.

both retain the 'primary modelling' of their parts they do not later receive the 'secondary modelling' which comes from the natural pressure of the head and socket against each other. For this reason, the acetabulum becomes flat and shallow with an undeveloped upper lip which offers no stable platform under which the head of the femur can lodge. The genetic aspect of the problem is evident in the fact that certain races are especially prone to congenital dislocation of the hip, notably the northern Italians, and the fact that it is ten times commoner in female children than male. Certain strains of certain species of dog are prone to congenital dislocation (e.g. the poodle).

The problem of ætiology is complicated by primary elements and secondary features which are difficult to disentangle. Somerville believes that, in addition to the genetic basis, too early extension of the hips is the principal feature precipitating dislocation. Von Rosen believes that an excess of œstrogen in the maternal blood, which is related to the relaxation of the maternal pelvic ligaments at full term, may lead to laxity of the fetal hip structures.

If the condition is undiagnosed and untreated, the child walks with a characteristic limp because the pelvis is merely slung to the upper end of the femur by capsular and ligamentary structures.

Clinical Features.—The clinical features which lead to a diagnosis are very different in the infant, as compared with the established case which has been walking for some years without treatment, and for this reason the two clinical types are dealt with separately.

In Infancy.—The difficulty in diagnosis in infancy is, of course, the fact that bony points are not palpable and the errors of the tape-measure are too great to be of value. The child moves both legs freely, and frequently the first suspicion that all is not well suddenly occurs to the parents when the child is about the end of its second year of life. Up till this time any slight limp on one leg is attributed to difficulty in learning to walk, and if the condition is bilateral the diagnosis may be even more difficult. If the diagnosis can be made during the first year of life, the prospects of a very successful result are appreciably greater than if the child has been taking weight on the dislocated joint for two or three years. The features which should be observed in infancy are therefore:

(1) *Asymmetry of skin creases on the inner aspects of the thighs.* Asymmetry of creases can

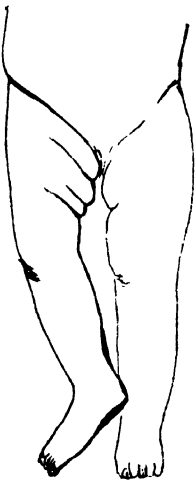


FIG. 393. — Asymmetrical skin creases in C.D.H.



FIG. 394.—Limited abduction of left thigh in C.D.H.
(J. C. R. Hindenach, F.R.C.S., London.)

be present with normal hip joints, but the reverse is uncommon and should arouse suspicions (fig. 393).

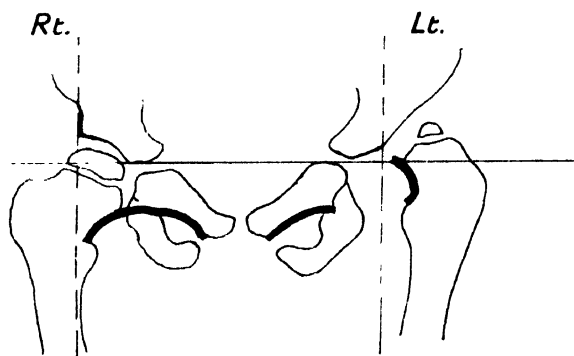
(2) *Telescoping of the Hip.*—Traction may reveal the ability to draw the hip down and then feel it telescope upwards.

(3) *Limitation of abduction compared with the opposite side* (fig. 394).

This is only likely to be noticeable if the head of the femur is grossly dislocated. This may be noticed when applying the infant's 'nappy'.

(4) *Ortolani or von Rosen Sign.*—This is a 'click' or 'snap' elicited by manipulating the hip from the adducted to the abducted position with the thigh flexed. It is the most important test of all, because it can be elicited in the first week of life in infants with congenital subluxation. The preceding signs, (1), (2), and (3), are easily seen in fairly gross cases of dislocation in children over one year of age where some contraction has already developed. It is incumbent on any doctor handling newborn babies to train himself in eliciting the Ortolani sign because treatment can be started immediately, and established congenital dislocation thus eliminated from the adult community.

(5) *X-ray Examination.*—Radiological diagnosis in a newborn infant is not as easy as might be imagined, except in gross cases. This is why the Ortolani test takes precedence over radiography and treatment should be initiated on this test alone in the first week of life. Because the epiphysis of the head of the femur does not appear until one year after birth, it will be realised that mild degrees of subluxation may not be obvious. The most important radiological test at between six and twelve months old is Shenton's line. In the normal hip this is a regular arcade which can be traced without any break in



continuity from the under-surface of the neck of the femur to the under-surface of the pubic ramus (fig. 395). If the cartilaginous head of the femur is slightly subluxated upwards, a 'step' will be visible in this arcade.

FIG. 395.—Congenital dislocation of the hip. Shenton's line (black) broken on left side. Note small ossific nucleus on dislocated side, above the horizontal and lateral to vertical lines, and the shallow acetabulum and its undeveloped roof.

When the capital epiphysis appears, the radiological diagnosis is much easier. In congenital dislocation the epiphysis is

often a little later to appear on the affected side than on the normal side, and even when present it is never quite as large as the normal side. When the epiphysis is present, subluxation can be detected by its position in reference to two lines, one horizontal and one perpendicular, drawn through the acetabulum. The horizontal line is drawn through the centre of the 'triradiate cartilage' of the acetabulum (fig. 395) and the ossific nucleus of the head should lie below this line. The perpendicular is drawn through the outer lip of the acetabulum and the ossific nucleus should lie medial to this. It will be appreciated that this line is sometimes rather difficult to place because a lack of distinctness of the lip of the acetabulum is characteristic of congenital dislocation.

Treatment.—*The diagnosis having been established within the first year of life, treatment consists of nothing more complicated than holding the thighs widely separated with the hips flexed. In this position the head of the*

emur lies opposite the defective acetabulum, and the pull of the adductors in this position will help to compress the head against the acetabulum and stimulate it to model itself into a deep cup to surround the head. Obviously this position must be maintained for at least one year. In the early weeks of infancy a pillow or specially folded diaper can be used to abduct the thighs, but in the later stages some type of abduction splint is needed.

So important is the early detection of subluxation that infants in whom the Ortolani sign is merely suspected should be treated as if they had congenital dislocation, because in this condition there is no penalty for over-treating. Six to twelve weeks on a simple splint at this age is all that is needed for a perfect result. Very simple and convenient rubber-covered splints have been developed for this purpose.

In Later Childhood

(1) *True Shortening*.—This will take place above the great trochanter as revealed by Nélaton's line (p. 218).

(2) *Prominence of the Great Trochanter*.—If the condition is bilateral, this will be associated with widening of the perineum which shows as a space between the inner surfaces of the thighs at the root of the limb.

(3) *Difficulty in palpating the femoral artery* at the level of Poupart's ligament because of the absence of the femoral head which should be behind it, so indicating emptiness of the acetabulum. Direct palpation of the femoral head in its abnormal position, which is the logical counterpart of this procedure, is not always possible.

(4) *Limitation of abduction*, due to shortening of the adductors, but full movement in all other directions.

(5) *Trendelenburg's Sign*.—To elicit this sign the patient stands with her back to the surgeon, and is first told to stand on the 'good' leg and bend up the knee on the 'bad' side. The patient will then be balancing on the good leg, and it will be noticed that the opposite, unsupported, side of the pelvis is lifted above the horizontal. This is the normal action of the pelvis in walking, and it is due to the pull of the gluteus

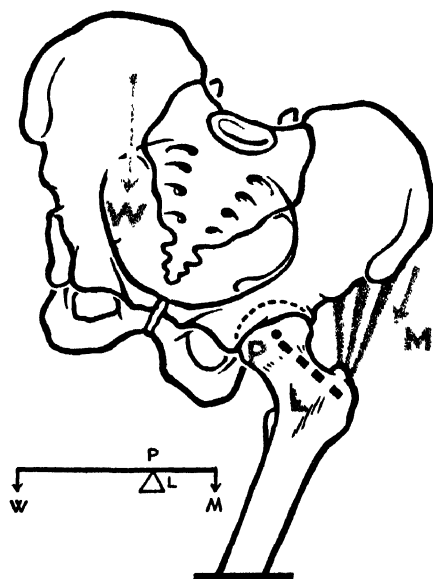


FIG. 396.—Mechanism of Trendelenburg sign. Tension of gluteus medius elevating pelvis.

medius acting between the crest of the ilium and the great trochanter (fig. 396). This is a negative (normal) Trendelenburg sign (fig. 397 (b)).

The patient is now told to "stand on the 'bad' leg and bend up the knee on the 'good' side". If a positive Trendelenburg sign is present, the opposite, unsupported, side of the pelvis will drop below the horizontal (fig. 397 (a)).

This means that the mechanics of the hip are so defective that the gluteus medius cannot elevate the pelvis as it should.

This 'sign' is repeated at every step when the patient is walking and gives rise to the characteristic 'dipping' gait of congenital dislocation. If the condition is bilateral, the gait is well described as 'waddling.'

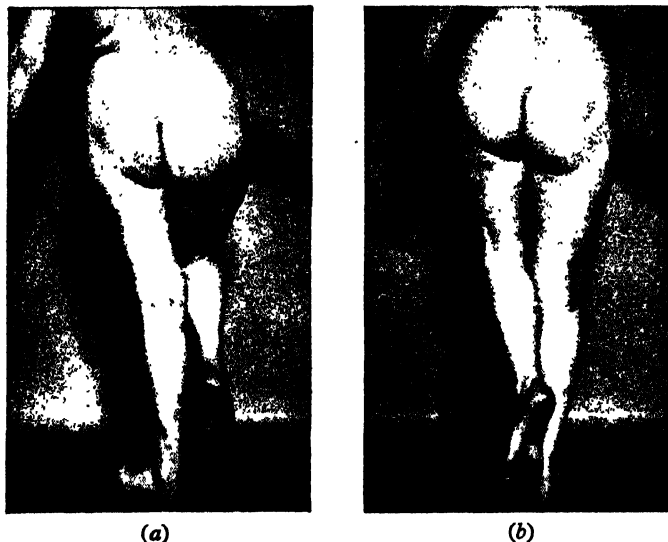


FIG. 397.—(a) Trendelenburg's sign positive — standing on defective hip. (b) Trendelenburg's sign negative — standing on sound hip.

The most marked examples of the Trendelenburg sign are seen in congenital dislocation of the hip, but it is also present, in a less marked degree, in many other disorders of the hip (e.g. un-united fracture of the femoral neck, defective arthroplasty of the hip, osteoarthritis, and paralysis of the gluteus medius).

Radiological diagnosis at this age offers no difficulty, the condition being obvious. In tracing Shenton's line, a word of caution should be sounded; in some severe dislocations a first glance may suggest that Shenton's line is intact because the curve denoted by the under-surface of the *lesser* trochanter may be level with the under-surface of the pubic ramus. It is important to note that the essential line is the under-surface of the *neck* of the femur.

Treatment.—The case first seen at one and a half to two years of age is first *manipulated* under anaesthesia, and if a satisfactory reduction is obtained the hip is held in plaster for about six months. The classical plaster is the 'frog' plaster (fig. 398) but there are grounds for believing that internal rotation of the hips is better than the 'frog' position.

An alternative method favoured by some is the gradual reduction by traction and abduction on a special frame, followed by a plaster or by open operation.

Many very successful results have been obtained by the simplest conservative methods, but there is always a 'hard core' of cases (perhaps 25 per cent.) which relapse and eventually require operative treatment. The radiological

criteria on which a satisfactory conservative reduction is judged tends to change as the experience of surgeons increases. What would have been considered satisfactory even ten years ago would probably not be so regarded today.

Operation is required in cases which re-dislocate after initial treatment; in all cases first seen over about five years of age; and in all cases over eighteen months or two years where the radiological appearances are not as perfect as they might be after adequate conservative treatment.

The operation consists of an open reduction with removal of the structures preventing the head entering the socket (i.e. an in-turned glenoid labrum, an hour-glass contraction of the capsule, or a contracted psoas tendon). If the acetabulum is very underdeveloped, a 'shelf' operation may be advisable, but this operation is practised less often today than formerly. In this procedure the superior lip of the acetabulum is turned down, to deepen the socket, and a bone graft inserted between it and the ilium to prevent it falling back.

A new operation which seems likely to have an important rôle in the future is innominate osteotomy (Salter). The iliac bone is divided in a line passing above the acetabulum between the sciatic notch and the anterior inferior spine. The acetabulum can be rotated downwards and forwards and held in the new position by a bone graft inserted into the osteotomy line.

The rôle of the anteverted state of the femoral neck which is commonly associated with congenital dislocation is becoming recognised as an important feature. A few months after open reduction many surgeons recommend a 'derotation' osteotomy as the final stage in treatment.

Late Cases.—Sometimes cases of congenital dislocation will present themselves at, or after, the age of puberty (fig. 399). At this age any attempt to reduce the hip by operation will produce a stiff and painful joint, and as most of these cases are quite remarkably active and pain-free despite their limp, it is wise to wait until pain from osteoarthritis starts in later life, when arthrodesis may be required.

COXA VARA

The normal angle between the axis of the neck of the femur and the shaft of the femur is about 130 degrees. In coxa vara this angle is diminished, and in some cases may be as little as 90 degrees. The effect of this will be to lower the level of the femoral head below the level of the tip of the great trochanter and so cause true shortening of the limb. The most important causes of coxa vara are :

- (1) *Congenital.* This is a very rare condition, often called 'infantile coxa vara', but it is of interest in that it produces some of the most extreme cases of coxa vara ever encountered, the shaft-neck angle often being less than 90 degrees.
- (2) *Conditions causing softening of the bones*, i.e. rickets, osteomalacia, Paget's disease.
- (3) *Trauma.* Fractures of the neck of the femur which unite with a



FIG. 398.—Application of plaster of Paris for bilateral congenital dislocation of the hips.



FIG. 399.—Bilateral congenital dislocation of hips, typical lordosis and prominent buttocks.

shaft-neck angle of less than normal are examples of 'traumatic coxa vara'. This does not cause any symptoms.

(4) *Pseudocoxalgia* (Perthes' disease). Pseudocoxalgia has been described elsewhere (p. 250), but special mention should be made of the very important condition of slipped epiphysis as a cause of coxa vara.

(5) *Slipped epiphysis*.

Slipped epiphysis (epiphysiolysis) affects children between the ages of ten to fifteen years and consists of a displacement of the epiphysis of the femoral head in relation to the neck of the femur taking place through the plane of the epiphyseal cartilage. The epiphysis slips into the varus position

at the same time as slipping posteriorly (being the same as if one considers the shaft and neck of the femur sliding into external rotation in relation to the head) (fig. 400). After a period of disability and pain, during which the slipping process is taking place, the epiphyseal line ossifies and the head of the femur will unite and the patient will spontaneously recover almost full function, though with the permanent physical signs of limited abduction (i.e. the varus element) and limited, or absent, internal rotation (i.e. the external rotation of the shaft). Later in life symptoms of osteoarthritis will develop.



FIG. 400.—Slipped epiphysis.

Many children suffering from this disease are grossly over-weight and

show pituitary dysfunction of the Fröhlich type, but the remainder appear to be children otherwise normal in all respects.

Two quite distinct clinical types of slipped epiphysis are encountered : (1) acute displacement and (2) chronic displacement.

(1) **Acute Slipped Epiphysis**.—Careful questioning will often reveal that the acute case is really an acute episode in what has been a chronic slip, but the symptoms prior to the acute slip can be so slight as to pass without notice. The acute slip often follows an injury of some magnitude, such as a fall off a bicycle or at football, and thereafter the patient is unable to move the joint and experiences severe pain. These cases on X-ray will show a complete displacement of the epiphysis and the clinical signs will be identical with those of a subcapital fracture in an adult (fixed external rotation, shortening, and limited abduction).

Treatment.—The acute case is treated exactly like a subcapital fracture of the femur in an adult. It should be reduced under anæsthesia without

undue delay (by internal rotation and abduction, i.e. the opposite of the deformity) and a Smith-Petersen nail (or preferably some form of screw) is inserted under X-ray control to maintain the reduced position.

(2) **Chronic Slipped Epiphysis.**—In this condition the child, between ten and fifteen years of age, is noticed to have a limp, but this may arouse no comment until after a week or two it gets worse and the patient then starts to complain of pain.

On examination the condition may at first be difficult to diagnose, because there may be spasm and limitation of movement in all directions, suggesting an inflammatory process (e.g. early tuberculosis). Rest in bed for a day or two will relieve the pain and spasm, after which limited internal rotation, limited abduction, and slight shortening may be demonstrable. X-ray will show some degree of slipping of the femoral epiphysis. If treatment is not instituted, the early slip will continue slowly or it may culminate in a sudden complete slip. It is to be noted that the earliest sign of slipping is best seen in the lateral radiograph *even when the anteroposterior view seems normal*.

Treatment.—Attempts to reduce the chronic slipped epiphysis are not to be encouraged because they will need great violence to move the head and thus may cause damage to the blood supply of the head. If the head has slipped only a slight degree, the position can be accepted and treatment directed to preventing further deformity. This can be by (1) *conservative* methods—i.e. skin strapping traction in abduction for nine to twelve months or till radiological evidence of fusion of the epiphyseal line is appearing; or (2) by *operation*, in which case a nail, bone graft, or threaded wires can be inserted under X-ray control so that the child can be allowed up, non-weight bearing, in a much shorter period.

If gross slipping has occurred by the time the patient is first seen, operative treatment is indicated, and in this case an osteotomy should be performed to correct the external rotation and the varus deformity.

GENU VALGUM

Knock-knee of a mild degree is very frequently encountered in healthy, well-nourished, and well-cared-for children without any recognisable pathology. The common cause of severe knock-knee in the past was simple rickets, but this is now very rare.

The degree of knock-knee can be measured by the intermalleolar separation present when the inner aspects of the knees are just allowed to touch. In children of about four years of age this separation can be as much as 2 to 2½ inches (5 to 6.25 cm.) without there being any evidence of systemic disease to account for it. This type of knock-knee often corrects itself spontaneously, and it is certainly true that in healthy children it never gets worse than when first seen. To the parent one of the main sources of alarm is the fear that if a child has 2½ inches (6.25 cm.) of knock-knee at four years, then by continued growth of the tibia, in the absence of treatment, one might expect twice this amount of intermalleolar separation when the tibia has grown to double its length. In fact, the opposite happens; even in the absence of

treatment the intermalleolar separation may diminish slightly as the child gets older, but even if it does not, the total appearance of knock-knee is concealed by the fact that $2\frac{1}{2}$ inches (6.25 cm.) of intermalleolar separation constitutes a negligible deformity in the adult.

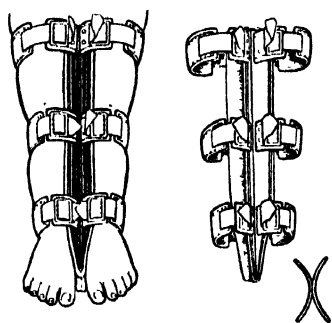


FIG. 401.—Mermaid splint for knock-knee.

In this common type of mild knock-knee the only treatment needed is to elevate the *inner* borders of the shoes by $\frac{1}{4}$ inch (6 mm.) and to prohibit, as far as is reasonably possible, the child from running about barefoot or in uncorrected shoes. This regimen must be adhered to for at least five years. More severe cases will benefit from a 'Mermaid' night splint (fig. 401) designed to exert a continuous moulding force on the knees, and in all cases the mother should be taught to mould the knees in the varus direction night and morning for about five minutes each day.

General bone diseases in childhood are often accompanied by knock-knee, and one of the most severe of these is *renal rickets* (p. 244). Most of the generalised diseases of bone are associated with dwarfism or other evidence of generalised growth disturbance.

Genu varum (*syn.* bow-legs) is less common than knock-knee, and in the past was most commonly due to rickets. Since rickets is now almost extinct in civilised communities, bow-legs are very rarely encountered and never to the gross degree of deformity which was a common occurrence in the past. If the deformity cannot be explained as part of a generalised disturbance of bone growth, it is usually the result of a local error in growth of the upper tibial epiphysis ('tibial varus').

The treatment of mild cases is by daily moulding, night splints, and the wearing of shoes raised $\frac{1}{4}$ inch (6 mm.) on the *outer* borders. Severe degrees of deformity are corrected by osteotomy.

Genu recurvatum is due to abnormal hyperextension of the joint, and severe deformities are often congenital in origin (fig. 402).

Serious genu recurvatum may follow poliomyelitis, where the hamstrings are paralysed and the quadriceps is left with some considerable power. This deformity is sometimes seen in spastic paraplegia, though more commonly in this disease the child walks with the knees partially flexed.



FIG. 402.—Genu recurvatum.

FLAT-FOOT (PES PLANUS)

Though it is a simple matter to diagnose 'flat-foot' on anatomical grounds, it is often difficult to decide whether such a deformity is to be regarded as pathological. The normal Negro foot cannot be accommodated in a European shoe, yet this foot is strong and symptomless. It is exceedingly difficult to decide what is the 'normal' human foot, because symptomless feet can vary in anatomical shape from the flat arch of pes planus to the high arch of pes cavus. Under conditions of active service in first-class

infantry regiments great variations in the shape of the foot were observed in unselected groups of men not complaining of painful feet.

In examining painful feet there are occasions when the surgeon is at a loss to know whether the complaint is really as serious as it seems to be from the patient's story. The patient with an anatomically flat-foot and a slight amount of discomfort may subconsciously, or hypochondriacally, use this feature as a psychological escape from unhappy circumstances. Similarly, patients will often be found who have worn 'arch supports' for many years and who allege that they are quite unable to get about without them, and yet the supports, when examined scientifically from an anatomical and physiological point of view, are utterly valueless.

If one rejects mere anatomical 'flatness' as the least important aspect of *pes planus*, one may ask what physical signs should be sought to decide whether a patient's symptoms are entirely based on organic disorder of the foot? The two most important physical signs are: (1) limited mobility of the tarsal joints and (2) localised tenderness. In general a patient with a completely mobile foot (mobile at subastragaloid, mid-tarsal, and metatarsotarsal joints) is unlikely to have disabling pain, but one with limitation of movement and spasm (i.e. pain elicited in certain specific movements) undoubtedly has a local disorder in the foot. Similarly, consistently localised tenderness in one part of the foot (e.g. region of the 'spring ligament', inferior calcaneonavicular ligament) is strong evidence of a local source of organic pain.

Types of *Pes Planus*.—(1) *Infantile*.—Infants and young children often show extreme degrees of flat-foot, and the term *pes valgus* is often preferable because the flatness in these cases is not so much an absence of the normal 'arches' of the foot (though it can be) but rather an eversion of the whole foot which makes it appear flat. Probably some of these congenital valgus feet should be classified under the heading of *talipes calcaneo-valgus* (p. 344). Some of the older children with this type of valgus foot also show knock-knee.

Treatment consists in instructing the parents to mould the feet into inversion at regular intervals during the day, and to supply night splints to hold the feet in an over-corrected position of inversion. When the child is old enough to walk, the inner borders of the shoes should be raised with a 'crooked and extended heel'. The child should be taught to practice walking on the outer borders of the feet with the 'toes turned in' until this becomes almost a habit.

(2) *Acute Foot-strain*.—This is not primarily a flat-foot, but if neglected may develop into chronic flat-foot. The foot is anatomically normal but is acutely painful, often swollen and tender, and resists passive movements of the tarsal joints by painful spasm. Though common in the past it is not now very frequently seen, as working hours are controlled and employees in their first job are not exposed to duties for which they have had no previous training. *Treatment* is by two or three weeks' partial rest, foot-baths, and graduated return to duty.

Sometimes 'acute flat-foot' is the result of local inflammation, and in this connection the best-known cause is gonococcal fasciitis.

(3) *Chronic Flat-foot*.—This is the end-result of untreated foot strain. It is seen frequently in waitresses and others who spend long hours on their feet carrying weights. In the later stages the feet become painless and the gait shuffling and inelastic. In its final stages this type of foot can be painless though rigid.

The treatment of chronic flat-foot applies mainly during the years when the foot is progressively breaking down. Arch supports moulded to the corrected shape of the foot and exercises to encourage walking on the outer borders of the feet with toes in-turned will prevent further deterioration if the patient co-operates.

Theoretically some of these feet in the stage of painful collapse might be rendered painless and capable of more prolonged stress if all the tarsal joints were fused (so-called triple arthrodesis of the tarsus). In fact the results of this operation, though occasionally done in the past, are not very encouraging, and the patient should be readjusted to some employment not demanding such heavy strains on the feet.

(4) *Traumatic Flat-foot*.—This is the flat-foot resulting from fractures which abolish the longitudinal arches. Fractures of the os calcis produce a very severe grade of flat-foot with a rigid subastragaloid joint. Mal-united Pott's fractures cause the appearance of flat-foot if the foot is everted (fig. 266).

(5) *Spastic Flat-foot* (Peroneal Spasm).—This is not an uncommon cause of some of the most severe degrees of flat-foot, and its early recognition is important if treatment is to be effective.

The condition is often unilateral, which tends to distinguish it from the other more common types of flat-foot. It occurs in children about the age of ten years, and in another group it seems to present itself for the first time in early adult life (twenty to thirty years). The presenting symptom is pain in the foot coming on after standing or at the end of the day.

On examination the foot is grossly flat and strongly everted, and the essential feature in the diagnosis is the inability of the surgeon passively to invert the foot. Passive attempts to invert the foot will cause the tendon of peroneus brevis to stand out like a bow-string above and below the external malleolus.

The essential pathology of this condition is unknown. The consensus of opinion is that the primary site of the disorder lies in the interosseous ligaments between the astragalus and the os calcis and that spasm of the peronei is a protective mechanism which relaxes these ligaments in the everted position of the foot. Certainly in early cases the condition is merely one of spasm, because under anæsthesia (or even local anæsthesia of the external popliteal nerve) the foot can easily be fully inverted. In later neglected stages of the condition, the foot becomes rigidly fixed in eversion and bony changes take place in the shape of the tarsal bones rendering the deformity permanent.

Treatment.—In children manipulation of the feet under anæsthesia and fixation in a walking-plaster for as long as six months is a procedure well worth trial, although some cases relapse.

In the adult, treatment is more difficult because the pathology of the condition is obscure. There are some who believe that the condition is nothing

more than an hysterical phenomenon and that the bony changes which result are due to long-standing spasm. In the adult the only treatment of value is an operative correction of the deformity by 'triple arthrodesis' in which suitable wedges are cut out of the subastragaloid and midtarsal joints to restore the normal shape of the foot. The amount of disability, the patient's occupation, and the possibility of a functional basis must be ascertained before such a radical step is taken, requiring as it does some six months before the patient is fit for work.

PES CAVUS (*syn.* CLAW-FOOT)

Pes cavus is an increased concavity of the arch of the foot, so that the instep is unduly high (fig. 403). It is sometimes associated with nervous diseases, e.g. poliomyelitis and Friedreich's ataxia.

In most cases the condition occurs idiopathically, and a possible explanation is a transient mild poliomyelitis affecting the lumbrical and interosseous muscles of the foot. The clawing of the toes could be explained because it is the intrinsic musculature of the foot which extends the toes (i.e. flexes the metacarpophalangeal joints and extends the interphalangeal joints). Without this mechanism the pull of the long flexors of the toes would cause them to assume the claw position.

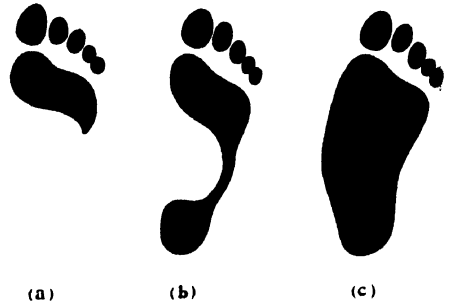


FIG. 403.—(a) Pes cavus. (b) Normal. (c) Pes planus.

Another mechanism in the deformity of pes cavus is shortening of the plantar fascia, which can be felt as a tight band stretched across the arch.

The treatment of pes cavus should be commenced when the condition is first diagnosed in childhood. If treatment is started in adult life, it is likely to be disappointing, therefore at this age before considering the treatment of the anatomical deformity the surgeon must satisfy himself that the patient has sufficient disability to warrant a time-consuming surgical programme. Many cases of pes cavus are able to play games and walk long distances. Such symptoms as are present in some of these cases can often be traced to painful callosities under the metatarsal heads due to the 'high loading' to which the small area of skin is subjected in this shape of foot. Carefully moulded insoles will do much to alleviate this source of discomfort.

In childhood the attempt to prevent the development of pes cavus is well worth major surgical intervention. The mildest cases are treated by subcutaneous fasciotomy of the plantar fascia accompanied by wrenching of the foot to lower the arch. A more radical method of abolishing the 'tie-bar' of the plantar fascia is the Steindler operation in which the plantar fascia and all muscle attachments are erased from the lower surface of the os calcis by open operation. Often the long extensor of the great toe can with advantage be transplanted to the neck of the first metatarsal to elevate this bone and so reduce the arch.

In cases where the toes all show considerable clawing, good results can be obtained by transplanting the tendons of the flexor digitorum longus on to the dorsal surface of the proximal phalanges of the toes so that this will flex the metatarsophalangeal joints.

In very advanced cases in elderly patients the clawed toes may be a source of great discomfort, due to callosities, and it may be impossible to wear ordinary shoes. In these cases with rigidly contracted toes, often associated with rheumatoid arthritis, very great relief can be obtained by amputation of all the toes through the metatarsophalangeal joints. When this is done, an ordinary shoe can be worn if it is fitted with a cork insert to fill the space left in the toe of the shoe.

TALIPES (*syn.* CLUB-FOOT)

The term talipes, or club-foot, is used to cover a group of foot deformities in which the sole of the foot is no longer plantigrade. In the commonest club-foot, which originally gave the condition its name, the foot is so twisted that the sole faces medially and the patient walks on what was originally the dorsal surface of the foot and its outer margin (equino-varus). Pes planus and pes cavus are not classed as club-foot because in these the sole of the foot is plantigrade, though the foot itself may be abnormal in shape.

To describe the deformity of any particular club-foot the term talipes is qualified with four adjectives describing the pure elements of the deformity, i.e. equinus, calcaneus, valgus, and varus. Equinus (i.e. like a horse) means that the patient walks on the tips of the toes because the tendo Achillis is too short. Calcaneus means that he walks on the heel, usually because the calf muscle is paralysed. Valgus indicates that the foot is everted and varus that it is inverted. It will be realised that only rarely are these deformities encountered in their pure form and usually combinations such as talipes equino-varus and talipes calcaneo-valgus are encountered (fig. 404).

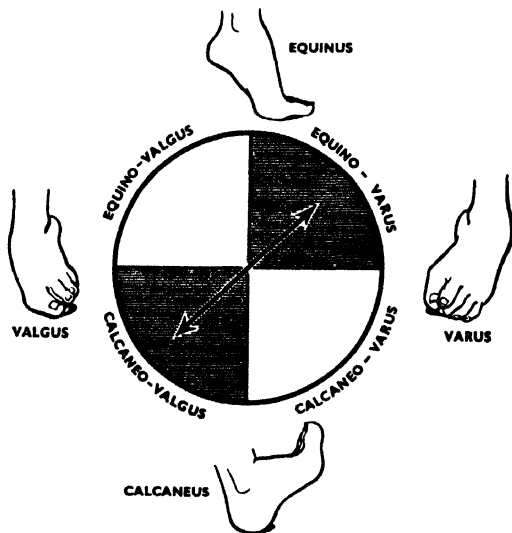


FIG. 404.—Nomenclature of talipes. Shaded quadrants indicate the common varieties. To treat equino-varus over-correct into calcaneo-valgus and vice versa.

Club-foot is either congenital or acquired.

Congenital Club-foot.—Though club-foot in newly born children may be part of a widespread error of development, as shown by association with club-hands, congenital dislocation of the hips, spina bifida, or harelip, these account for only a minority of cases. The common club-foot usually occurs in what is otherwise a completely healthy baby.

The exact ætiology of club-foot is still obscure. The old theory of malposition in utero is no longer tenable because simple mechanical malpositions are easily corrected and, once corrected, the limb will hold its correction and grow normally. In club-foot, however, there is a persistent tendency for the deformity to revert to its original condition, and the surgeon and nursing team are faced with a continuous fight against relapse until the child is three or four years old. It is probable that the fundamental abnormality lies in the muscles of the leg and that certain muscle groups

are less extensible than others. As the tibia grows in length, it is suggested that some muscle groups do not elongate at the normal rate and thus there is a tendency for the foot to be pulled into deformity. This theory would explain the constant tendency to relapse as growth continues.

In the common talipes equino-varus the muscular imbalance would affect the calf muscles, tibialis anterior, and tibialis posterior rather than the peronei. It is certainly a fact that even when excellent results have been achieved, a child who has had a unilateral club-foot never has a calf which is as well-developed as the normal side.

Treatment.—Basically there is nothing more subtle in the treatment of club-foot than is used in making a tree or plant grow in a predetermined direction by holding it in the desired position for long enough. The sole difficulty is in the devising of apparatus to be effective and convenient. The foot is held in slight over-correction, i.e. an attempt is made to convert the foot into the opposite type of deformity—equino-varus into calcaneo-valgus and vice versa (fig. 404). The treatment is as follows (though details vary greatly according to the surgeon):

(1) For mild cases: manual moulding into the direction of over-correction by the parent or visiting nurse. This may be combined with strapping. This is usually sufficient for calcaneo-valgus which, unlike equino-varus, tends to recover spontaneously.

(2) Denis Browne splints strapped on to both feet (connected by a metal bar) so that the feet are held everted for six months (fig. 405).

(3) Plaster-of-Paris applied at regular intervals in an attempt to hold the correction, used for more resistant cases or for neglected cases.

(4) After six months Denis Browne splints are attached to the boots, and later used as a night splint till walking is well established.

During this process of correction, in the case of the common congenital talipes equino-varus (C.T.E.V.), it is customary to correct the varus element first, and later to start correction of the equinus deformity.

In cases of relapsing club-foot or incompletely corrected cases first received two or three years after birth, the 'Kite' method of correction has now received widespread approval. In this a plaster is applied to the limb and, when hard, it is wedged along a line chosen to correct the selected deformity.

In a residue of cases an operation is needed. The operations available are :

- (1) Elongation of the tendo Achillis for the equinus deformity.
- (2) Transposition of tibialis anterior tendon to the outer side of the foot for persistent recurrence of varus.
- (3) Division of all contracted ligamentary structures around the tarsal joints on the inner side of the foot to permit correction of varus deformity.
- (4) Mid-tarsal-subastragaloid fusion with removal of wedges of bone so that the foot will be plantigrade.

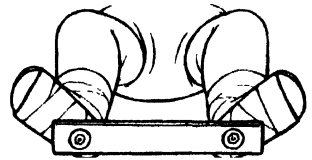
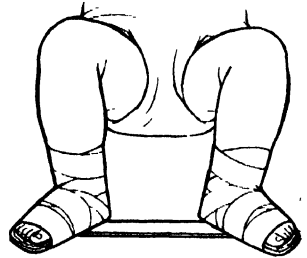


FIG. 405.—Denis Browne splint.

Acquired Talipes.—By far the commonest cause of acquired talipes is poliomyelitis (paralytic talipes). In neglected paralysis of the muscles of the anterior compartment of the leg, the foot will develop a rigid and fixed equino-varus deformity. The patient will have difficulty in wearing normal shoes, and may develop painful callosities on the outer aspect of the foot. On the other hand, many patients have minimal disability but considerable deformity, and judgment is needed in deciding whether they will be improved by operative intervention. If the equinus deformity compensates for loss of length due to interference with growth, conversion to a plantigrade foot may be a poor exchange if the patient then has to wear a surgical boot with an ugly 'raise.'

Other acquired deformities result from a multitude of different causes, such as fixed equinus from decubitus in bed without a cage and foot support, burns of the calf, ischæmic contracture of the calf, spastic paraplegia in children, and spastic hemiplegia in older people.

The treatment of these deformities is by tenotomy of the tendo Achillis or by operations on the tarsus to remove appropriate wedges.

CALCANEAN SPUR

is a result of plantar fasciitis following foot strain, which is perhaps aggravated by some focus of infection. Persistent local tenderness suggests the diagnosis, and an X-ray shows the bony outgrowth projecting forward from the under-surface of the bone. A horseshoe-shaped Sorbo insole should be fitted in order to relieve pressure, but if disabling symptoms persist excision is occasionally performed through a lateral incision.

HALLUX VALGUS

Hallux valgus is encouraged by wearing of boots or shoes with pointed toes. The deformity consists of abduction, i.e. outward displacement of the big toe. It tends to be progressive, because the direction of pull of the extensor longus hallucis tendon further increases the deformity by a 'bowstring' mechanism when outward displacement has occurred (fig. 406). The following conditions are often associated:



FIG. 406.—Hallux valgus.

(1) *Hammer-toe*, owing to the misplaced big toe displacing the adjacent second toe.

(2) *A bunion*, which is an inflamed adventitious bursa, developing over the prominent head of the first metatarsal bone as a result of pressure. Suppuration sometimes follows, and the big toe joint may be secondarily affected.

(3) *Osteoarthritis of the big toe joint*, owing to pressure, malalignment of the bones, etc. Severe pain results, and X-rays frequently

show osteophytic outgrowths.

Treatment.—The conservative treatment of hallux valgus is unsatisfactory. As the patient is almost invariably a female, it is usually too much

to expect that she will accept the scientific advice of wearing shoes with low heels, wide fronts, and straight inner borders. A small rubber pad can be worn in the cleft between the first and second toes, but this also demands the use of a wide-fronted shoe.

In general, a young adult with hallux valgus will eventually require operative treatment no matter what conservative techniques are tried, but it is important not to operate merely for cosmetic purposes if the foot is reasonably comfortable. The most satisfied patients after hallux valgus operations are those in which very marked discomfort and gross deformity were present.

Operation.—Local removal of the bunion together with the underlying osteophytes, without interference with the metatarsophalangeal joint, is not a satisfactory operation because symptoms almost always recur, and a more radical operation is later required. There are perhaps exceptional cases in young people where the operation is performed if the valgus deformity is only slight.

The standard operation for hallux valgus is the making of a pseudarthrosis by excision of the base of the proximal phalanx (Keller). The Keller procedure in younger patients can be augmented by an osteotomy of the base of the first metatarsal to abduct the first metatarsal and close the enlarged gap between it and the second metatarsal (Stamm). The corrected position is maintained by inserting excised bone to hold the osteotomy open on the medial aspect.

After the operation, at least three months will elapse before the foot will tolerate an ordinary shoe, and the patient may not feel the full benefit of the operation for as long as six months.

Hallux rigidus occurs as two distinct varieties.

(1) The adolescent type is due to synovitis of the metatarsophalangeal joint following injury, and is associated with muscular spasm. There are no radiological changes. It is relieved by wearing a metatarsal bar $\frac{3}{4}$ inch (2 cm.) wide and $\frac{1}{2}$ inch (1.25 cm.) thick (fig. 407).

(2) The adult type is nothing more than mon-articular osteoarthritis, sometimes precipitated by injury. The limitation of movement is due to interlocking of osteophytes, and also to flattening of the metatarsal head (fig. 408). Treatment is by operation, the Keller procedure being the one most widely employed. In this operation the painful rigid toe is replaced by a pseudarthrosis at the metatarsophalangeal joint.

Hammer-toe consists in hyperextension of the metatarsophalangeal joint and flexion of the proximal interphalangeal joint (fig. 409). Callosities form over the bony prominences, and in long-standing cases adventitious bursæ



FIG. 407.
—Metatarsal
bar.



FIG. 408.—Hallux
rigidus.



FIG. 409.—Hammer-toe with callosity.

develop. Fascia and ligaments become secondarily contracted.

Hammer-toes sometimes develop from overcrowding, either by small or pointed shoes, or as a result of hallux valgus. Pes cavus, as an associated condition, has already been mentioned.

Treatment consists of correcting any predisposing cause and wearing a corrective splint.

If the deformity is established, operative intervention is required. The 'spike' operation (Higgs) ensures bony ankylosis, and consists of drilling the base of the middle phalanx and impaling it on the shaft of the proximal phalanx after shaping the condyles into a spike.

INFANTILE PARALYSIS (POLIOMYELITIS)

Infantile paralysis has been greatly reduced in incidence and severity in Western countries as a result of widespread immunisation campaigns. Two types of vaccine are currently used: (1) Salk vaccine is an injection of organisms killed by formalin and (2) Sabin vaccine is an oral preparation of virus particles of attenuated virulence.

This condition occurs most commonly in the late summer months, and there is still a great deal which is not understood about the method of spread of epidemics. Anterior poliomyelitis is a notifiable disease, and may be transmitted by nasal secretion or faecal contamination of 'carriers'. It is now certain that the route of ingress is through the alimentary canal. The responsible organism is ultra-microscopic, passes through a porcelain filter, and can be transmitted to apes. The virus causes a meningeal reaction, the cerebrospinal fluid being under pressure and containing an excess of cells and albumin. The anterior horn cells in the cord are attacked and the corresponding muscles are paralysed. There is good evidence that undue muscular exertion during incubation can direct the paralysis to the exhausted muscle groups and also render more profound the resulting paralysis of these groups. Therefore rest should be strictly enforced in the early stage of any febrile condition where the possibility of poliomyelitis is considered.

The disease is divided into three stages:

(1) *Stage of Invasion*.—The onset is usually sudden, and is characterised by a rise of temperature, pain in the head and spine, and usually more or less widespread cutaneous hyperæsthesia. Owing to meningeal irritation stiffness of the neck or back is constant and early—the 'spine sign.' Paralysis is evident after two or three days, and unless an epidemic is rife the true nature of the condition is frequently unsuspected until the paralysis is discovered. Lumbar puncture shows a preliminary rise in polymorphonuclear cells, followed by an increase in lymphocytes. In a severe paralysis the proximal muscles, i.e. shoulder and arm groups, hip and thigh, are usually more completely palsied than the distal groups controlling hand and foot. Spinal and abdominal muscles are frequently affected, the

former leading to scoliosis. Paralysis is very variable in extent and distribution; in severe cases the bulk of the skeletal muscles are initially affected, while in others merely one or two isolated muscle groups suffer.

(2) *Stage of Recovery*.—If a muscle group remains *completely* paralysed for four to six months, then paralysis is permanent, and further efforts to improve muscular tone are a waste of time. If there is a little voluntary movement after the acute phase has subsided, then continuous improvement in strength is likely for two or three years.

It is a matter of consolation that the extent of the paralysis at the onset is not an indication of the final result. Some cases start with almost total paralysis may be left with very localised and tolerable permanent disabilities, but the main recovery is apparent in a few weeks.

(3) *Stationary Stage*.—This is approximately after the lapse of two years, but further slight improvement is possible. Shortening of the limb may result from inequality of growth. Sensitivity to cold may also be troublesome with chilblains and ulceration, requiring sympathectomy (p. 139).

Treatment.—(a) *Stage of Paralysis*.—Symptomatic treatment is all that can be attempted. The affected limbs should be splinted in the positions which place the joints in neutral position and so avoid undue stretching of paralysed muscle groups. Joints should be moved at regular intervals to prevent stiffness. Massage or electricity is harmful, in that the affected muscles require rest. Lumbar puncture is indicated for meningeal irritation, as withdrawal of fluid under pressure gives relief.

(b) *Stage of Recovery*.—During this period three principles of treatment must be observed :

(i) Relaxation of paralysed muscles. If paralysed muscles are allowed to become permanently stretched (by the pull of healthy antagonistic muscles or by the influence of gravity), any subsequent recovery of tone is neutralised by the 'slack' which has been allowed to develop. Therefore splints, surgical boots, and other forms of apparatus must be worn to prevent over-stretching of affected muscles (fig. 410). A plaster-of-Paris shell is required for spinal cases, to be succeeded by a jacket.

(ii) Maintenance of nutrition. Massage, electrical baths, radiant heat are all useful in stimulating the circulation and improving nutrition. Weak muscles must not be over-stimulated, and the limb is maintained in such a position as to relax affected muscles during treatment.

(iii) Exercise. As recovery occurs, active muscular contractions are encouraged, provided that the effort is well within the powers of the weakened muscle. Exercise beyond the point of fatigue may be harmful.

(c) *Stationary Stage*.—After a period of two years the value of the affected muscles can be assessed, although it should be remembered that further slight improvement is still possible. The principle of treatment is now to

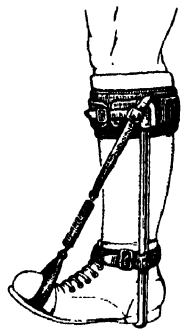


FIG. 410.—Surgical boot—lateral iron to the knee, a valgus strap, and a toe-raising spring to correct talipes equino-valgus.

restore muscular balance, either by means of surgical apparatus or by operative procedures, with the advantage that appliances can then be discarded or simplified. The following procedures may therefore be adopted:

(i) Tendon transplantation, by which means the action of a stronger muscle is transferred to a weaker group, or the direction of muscular pull is altered so as to overcome deformity.

The following principles should be considered in connection with tendon transplantation:

(a) If possible the tendon is selected from muscles with the same innervation as those affected, so that re-education is simplified.

(b) The joint must be fully mobile, i.e. the transplant must not be expected to work against contractures which have not been abolished prior to the transplant.

(c) The path of the transplanted tendon should be as direct as possible.

(d) The limb must be relaxed before the tendon is fixed.

(ii) Tendon fixation (tenodesis) has been used in order to assist in the fixation of a flail joint. Thus in the case of the ankle joint the selected tendon is fixed to the bone on both sides of the joint in order to secure stabilisation. Results are disappointing, as paralysed tendons readily stretch when subjected to strain.

(iii) Tenotomy, which has little scope in the treatment of infantile paralysis, as muscles are already weakened and the contraction of stronger muscles should have been prevented.

(iv) Arthrodesis is a useful procedure, in that it obviates the continued use of surgical apparatus devised to stabilise a flail joint. The operation consists in removal of the articular cartilage and securing bony union between the bones which comprise the joint. Arthrodesis is often combined with tendon transplantation.

In paralysis of the foot it is necessary to stabilise the tarsus so that a firm plantigrade foot is presented to the floor at each step, without any tendency to roll over into inversion as would happen if it strikes the ground while slightly inverted. The Naughton Dunn operation is widely used for this purpose.

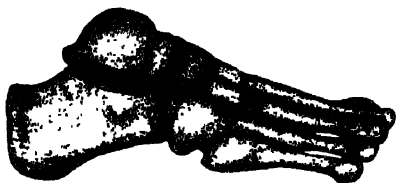


FIG. 411.—The shaded areas indicate the bone to be removed in Naughton Dunn's arthrodesis.

Naughton Dunn's arthrodesis consists in excision of the subastragaloid and mid-tarsal joints. This includes, on the inner side, the scaphoid and adjacent parts of the astragalus and cuneiform bones, and on the outer side adjacent parts of the os calcis and cuboid (fig. 411). The foot is displaced backwards, and becomes stabilised

by ankylosis of the mid-tarsal and subastragaloid joints, but ankle movements are preserved.

(v) Bone lengthening is valuable in cases of shortening. The femur or tibia is divided obliquely and continuous skeletal traction will augment the length of the limb up to 2 inches (5 cm.).

Some surgeons prefer to excise a part of the femur in order to shorten the normal leg. Alternatively, in a growing child staples are driven into the bone to bridge the epiphyseal line and so prevent further growth.

CHAPTER 17

THE HEAD

GEOFFREY KNIGHT

THE SCALP

THE **scalp** consists of four layers—skin, subcutaneous tissue, epicranial aponeurosis or galea, into which is inserted the occipito-frontalis muscle, and the subaponeurotic areolar layer. The scalp is well supplied with blood-vessels, and the walls of the arteries are adherent to the fibrous tissue in the subcutaneous layer. Therefore scalp wounds bleed freely, as the muscular coat of a divided artery cannot retract readily when the vessel is severed.

Wounds of the scalp are treated by adequate shaving of the surrounding scalp. The wound is excised, hæmostats applied to the galea are allowed to fall back over the skin edge. The scalp is closed in two layers, galea to galea and skin to skin, with interrupted black-silk sutures. In the case of tissue loss the gap is closed by making a reversed 'S' incision, by which means most defects in the scalp can be closed (fig. 412). This leaves the pericranium exposed at the ends of the incision. Healing occurs by granulation from the pericranium, which should be protected with tulle gras, and subsequent epithelialisation of the granulated area.

Owing to its rich blood supply wounds of the scalp heal readily. Many cases are on record where the scalp has been avulsed almost completely, but after cleansing and suturing into position, union has occurred in a satisfactory manner.

Hæmatoma of the scalp may be subcutaneous. Subaponeurotic hæmatomas spread extensively beneath the epicranial aponeurosis and are only limited by the attachment of the aponeurosis around the base of the calvarium. Subpericranial hæmatomas are limited by the suture lines which border the underlying bone, to which the pericranium is attached.

Infection of the scalp may be limited in a similar manner. In the subaponeurotic areolar layer infection is circumscribed only by the attachment of the epicranial aponeurosis, and may extend from the superior curved line of the occipital bone behind to the supraorbital ridge in front, and laterally be bounded by the zygomatic arch, temporal ridge, and upper border of the mastoid process. The subaponeurotic areolar tissue is termed the 'dangerous area', since intracranial infection may occur secondarily

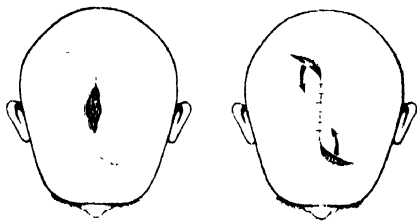


FIG. 412.—Letter S scalp plastic incision.

(p. 375) via the emissary veins. Treatment is by chemotherapy and incisions parallel to the main vessels and nerves.



FIG. 413.—Sebaceous cysts of the scalp.

Sebaceous cysts (*syn. wens*) (p. 100) are often multiple (fig. 413) and may grow as large as hen's eggs. The surrounding scalp becomes bald if pressure interferes with its blood supply. Infection and ulceration are described on p. 101.

Lipomas arise from fatty tissue incorporated with the areolar layer (fig. 55).

Cirsoid aneurysm is rare and difficult to treat. Capillary nævi sometimes occur in the skin, beneath which abnormal arteries communicate directly with distended veins. Most commonly the superficial temporal artery and its branches are affected. The underlying bone becomes thinned by pressure which also causes the hair to fall out. Radiography may show perforations in the skull which indicate that part of the tumour is intracranial. These tumours tend to enlarge slowly with a risk of serious hæmorrhage if ulceration occurs.

Treatment consists in extirpation of the tumour in the early stages (ligation of one or both external carotid arteries is advisable as a preliminary step to local excision). If its size forbids such radical treatment, then the main vessels are ligated, but this is often unsuccessful, as a large tumour on the surface usually possesses intracranial connections which maintain the blood supply.

A **dermoid cyst** occurs most frequently over the external angular process (fig. 583). Although congenital, it may not appear until the child is some years old. It may communicate with an intracranial dermoid by a narrow neck, which passes through the underlying bone. In this case a cough-impulse can sometimes be detected. X-ray shows a bone defect with a sclerotic margin (fig. 414).

Treatment.—Superficial cysts are excised locally. If there is evidence of intracranial extension, the cyst is approached by osteoplastic craniotomy, as the intracranial portion may be larger than the superficial.

Papillomas are common and cause discomfort on combing the hair. Constant irritation of this nature encourages malignant changes.

Fibrosarcoma has already been described (p. 59).

Epithelioma of the scalp is not common and presents no special features.

Melanomas may be unrecognised, owing to the small growth being hidden by hair. If malignant changes supervene, the appearance of secondary deposits should lead to the discovery of the tumour.

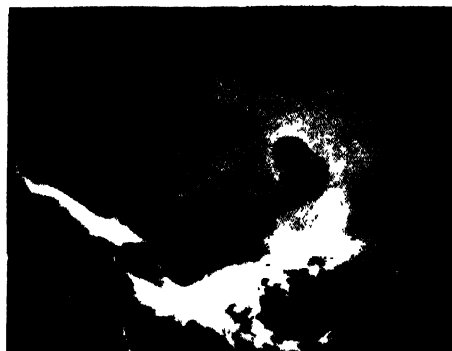


FIG. 414.—Lateral X-ray showing dermoid cyst of the diploë. The inner table has been penetrated by intracranial extension.

THE SKULL

Microcephaly may be associated with agenesis of the brain and imbecility, or result later from premature synostosis in a normal child.

Oxycephaly (*syn.* steeple-head, Gk. *oxus* = sharp) is a condition in which the skull is egg-shaped, following premature obliteration of sutures. Most cases develop increased intracranial pressure.

Treatment.—*Cranioplasty* by the formation of Gigli saw cuts between burr holes and wrapping the bone edge in tantalum foil allows normal skull expansion and cerebral development to proceed.

Meningocele.—*Clinical Features.*—Protrusions of a pouch of dura mater through a congenital defect in the skull are usually situated at the root of the nose or over the occipital bone. Trans-sphenoidal projections protrude through the base of the skull into the naso-pharynx and have been mistaken for nasal polypi: attempted removal has resulted in meningitis. A meningocele is present at birth, and forms a tense rounded swelling which is translucent and sometimes pedunculated, and which yields an impulse when the child cries or coughs. Growth of the skull may occlude the neck of a small sac; in this case a cyst remains which is non-pulsatile and unaffected by coughing.

An *encephalocele* is a similar condition, but some portion of the brain is also extruded (fig. 415). Should this cerebral extrusion contain part of a ventricle, it is known as a *hydroencephalocele*. In these conditions vascular pulsation is present. The child may be still-born, some degree of idiocy or deformity may be associated.

Treatment.—The skin surface should be protected with padding and tulle gras to prevent ulceration and infection. If at the age of a month the child shows normal development, operation is performed under local anæsthetic, the child being tied to a cross and given a feeding bottle during operation. A curved incision is fashioned in one margin of the sac, so that when the wound is sutured the incision will not overlie the bone defect. The neck of the sac is defined, transfixed, and ligated and the sac and excess skin removed. Muscle and fascia are brought together over the bone defect. Many cases are found to have a small encephalocele at the base. This is removed with the sac, without harmful effect, as the tissue is functionless.

Skull Fractures.—See Cranio-cerebral Injuries (p. 354).

INFLAMMATION OF CRANIAL BONE

Osteomyelitis occurs as a result of:

- (i) *Direct infection*, such as a compound fracture.
- (ii) *Local extension* of infection from the frontal sinus or mastoid antrum.

The diploe is sometimes infected as a result of cellulitis of the scalp.



FIG. 415.—An encephalocele. A myelocele is also present.

Leonardo Gigli, 1863–1908, an Obstetrician of Florence, invented his saw for pubiotomy.

(iii) *Blood-borne* infection by circulating organisms is practically unknown, except when rarely it follows bruising of the bone in young children.

Acute osteomyelitis of the skull is a serious condition. Infective thrombosis of emissary veins, extradural abscess, subdural abscess, brain abscess, and meningitis are grave complications (p. 375).

Treatment consists in the administration of antibiotics, which are extremely effective in controlling both acute and chronic osteomyelitis. Hence the wide excisions of both tables of the skull, at one time practised in the acute stage, are seldom required.

In the chronic stage, sequestra are sterilised under antibiotic treatment, and then excised. Bare areas in the overlying scalp are treated by skin grafting.

Tuberculous disease of the skull is uncommon, but occasionally occurs in association with tuberculous lesions elsewhere. As with other bones, the infection commences either in the pericranium or in the medulla, i.e. the diploe. The diseased bone should be removed widely, otherwise abscesses are likely to form and erode the scalp. Antituberculous drugs are given (p. 23).

Syphilitic pericranitis is nowadays a rare affection. Localised swellings occur which are slightly tender and fixed to the bone. Under suitable treatment disappearance is usual, although a small bony swelling occasionally remains.



FIG. 416.—Lateral X-ray showing irregular clear area produced by a secondary deposit from carcinoma of the breast.

NEW-GROWTH OF THE SKULL

Innocent tumours are rare. An *ivory* osteoma occasionally arises in the region of an air sinus (p. 256).

Malignant new-growths resemble those of other bones. *Pericranial sarcoma* forms a tumour, the consistency of which depends upon its vascularity and rate of growth. Thus it may be pulsatile or of an almost bony hardness. *Osteoclastoma* occasionally develops in the diploe.

The commonest malignant tumour is secondary carcinoma, usually derived from primaries in the breast, thyroid, prostate, or suprarenal.

Hypernephromas produce rapidly growing vascular tumours, which pulsate when the outer table is eroded. Secondary deposits usually produce a single clear area on the skull X-ray with irregular margin (fig. 416). Deposits from carcinoma of the breast are often multiple.

CRANIO-CEREBRAL INJURIES

Head injuries are injuries of a composite structure in which, according to the mechanism, either the brain, the skull, or intracerebral vessels may be injured separately or together. Except in exceptional circumstances, some

degree of brain injury, either minor or major, is inflicted at the moment of impact producing a neuronal lesion, the effects of which determine the immediate clinical picture and often the outcome of the case. The phenomena of skull fracture, œdema and intracranial hæmorrhage are superimposed upon this basic picture of neuronal injury. In the mechanism of intracranial injury the direction and plane of the injuring force and its velocity are, in general, as important as the magnitude of the forces involved. The serious complications of intracranial hæmorrhage in particular are produced by relatively minor forces acting in a certain plane.

INJURIES OF THE BRAIN¹

Mechanism and Pathology.—At the moment of impact a diffuse neuronal lesion is inflicted on the brain which is responsible for the immediate clinical picture of brain injury. Secondary changes of œdema or intracranial hæmorrhage take time to develop. The rise in pressure resulting from these causes may lead to a deterioration in the patient's level of consciousness at an interval after injury; the clinical picture in the early stages results from the neuronal lesion alone. All degrees of brain injury resulting in loss of consciousness, concussion, contusion, or laceration of the brain are

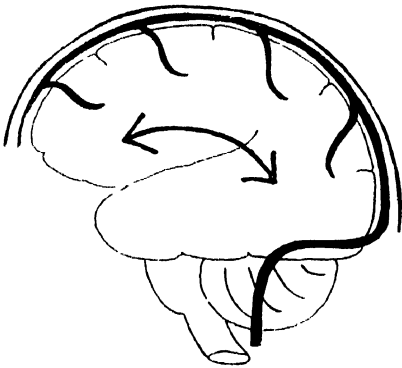


FIG. 417.—Suspension of the brain in the slings formed by the superior cerebral veins.

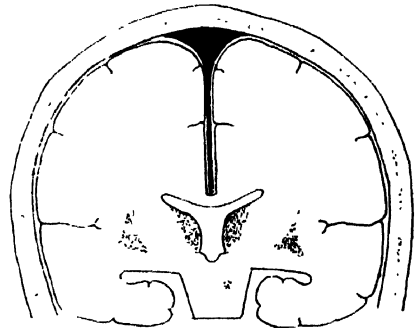


FIG. 418.—The midline septum of the falx.

produced by one mechanism, namely displacement and distortion of the cerebral tissues occurring at the moment of impact.

The brain is not a perfect fit within its membranes; it is capable of a certain degree of antero-posterior riding movement within the spaces of the cerebrospinal fluid in which it is suspended by slings formed by the superior cerebral veins (fig. 417). Lateral displacement of the brain inside the skull is restricted by the midline septum of the falx (fig. 418).

Blows on the front or back of the head therefore lead to the maximum displacement of the brain, with proportionately severe cerebral injuries, the cerebral hemispheres being displaced in relation to the brain stem and hypothalamic region; in this displacement one hemisphere may be distorted

¹ 'No head injury is too slight to neglect, or too severe to be despaired of.'—Hippocrates.

² Hippocrates, by common consent the Father of Medicine, was born in the island of Cos in the Aegean archipelago about 460 B.C. He lived to be 109 years of age in an era when the expectation of life was about 32 years.

in relation to the other, with the resulting stretch and strain on the junctional tissues of the commissures and corpus callosum. Laterally directed blows severe enough to fracture the skull cause less cerebral displacement, and therefore less associated diffuse injury of the brain.

The lines of force set up by cerebral distortion act chiefly upon the region of the third ventricle and hypothalamus and brain stem (figs. 419 and 420).

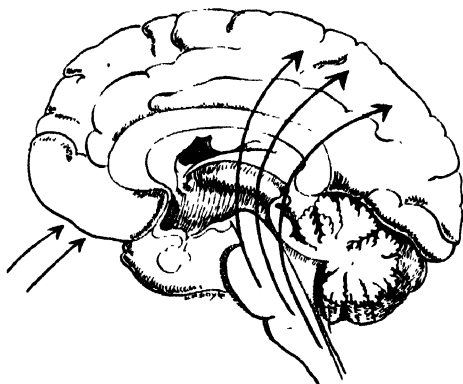


FIG. 419.—Lines of force acting on the hypothalamus and brain stem as the result of posterior displacement of the hemisphere.

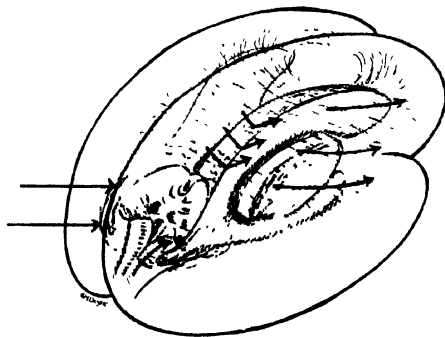


FIG. 420.—Lines of force acting on the corpus callosum and one peduncle as the result of anterior displacement of one hemisphere.

The severity of these forces is demonstrated by the fact that in deaths from boxing injuries, the medulla may be torn across. At first sight it is difficult to understand how stretch exerted upon the brain stem can disturb consciousness, unless it is realised that consciousness is not maintained exclusively by the cerebral cortex, but is also maintained by the basal areas and hypothalamus. After removal of both cerebral hemispheres at a level immediately above the thalamus (Goltz's dog experiment), an animal is deprived of all volition, but it can stand, and will respond to any stimulus by a show of violent and uncontrolled rage, a reaction which is curiously similar to that seen in post-traumatic delirium following brain injury. Since imperfect consciousness is maintained following a lesion at this level in the brain, it follows that for consciousness to be completely lost, a lower level of the brain stem must be affected, and patients are, in fact, rendered completely unconscious by the effect of distortion acting upon the brain stem and affecting ascending stimuli which maintain the central excitatory state of the cortex.

In all cases the instantaneous loss of consciousness which occurs at the moment of injury, whether short or long lasting, is produced by paralysis of conduction in the brain stem following distortion, and this occurs quite independently of any change in intracranial pressure whatsoever.

The clinical terms which are used to describe brain injury, concussion, contusion, and laceration of the brain merely indicate minor and major degrees of this process and do not imply any individual difference in pathology.

In cerebral concussion the stretch is slight. There is a brief temporary

physiological paralysis of function without organic structural damage, which results in a transient loss of consciousness, followed by complete recovery.

In cerebral contusion the stretch is severe. There is some rupture of vessels. Unconsciousness is more prolonged, the initial recovery is imperfect, death may result from the effects of the lesion on the vital areas of the brain stem acting independently of any change in intracranial pressure; indeed, many fatal cases die with an abnormally low intracranial pressure.

In severe cases of cerebral contusion there is an additional sliding movement of the grey matter in relation to the white, which results in damage to nerve cells and axons. Degeneration following rupture of axons accounts for the post-contusional state associated with defective memory and personality change in patients who survive.

Histological Changes.—In fatal cases there may be no visible surface injury, but section reveals rupture and degeneration of axons and pyknosis of nerve cells. Ring hæmorrhages produced by bleeding into the perivascular spaces of Robin-Virchow are produced by rupture of vessels at the points of maximum stress in the region of the commissures, corpus callosum, and deeply in the third ventricle and substantia nigra in the brain stem. Cloudy swelling is seen in the choroid plexuses, and results in reduction of cerebrospinal fluid formation and the low intracranial pressure found in many fatal cases.

In cerebral laceration the internal changes are the same as those seen in contusion, but the brain surface is torn, with effusion of blood into the cerebrospinal fluid owing to laceration against bony ridges and the edges of dural septa during brain displacement. Lacerations are common on the inner aspect of the hemisphere against the falx, on the under aspect of the frontal lobe, and tip of the temporal lobe, the latter producing traumatic anosmia. They are less common at the occipital poles, producing blindness or hemianopia. Frequently surface injuries are situated at a point diametrically opposite the site of impact (*contre-coup*), and are produced by displacement of the brain.

Clinical Features.—*Cerebral concussion* may produce either a momentary loss of consciousness or a short period of unconsciousness and cerebral shock of minutes only, which is then followed by a complete and perfect recovery with no residual signs. No treatment is required other than rest and analgesics for headache, provided that there is no associated evidence of skull fracture (p. 368).

Cerebral contusion is diagnosed when unconsciousness is prolonged, if the patient recovers to a state of imperfect consciousness, such as confusion, irritability, or delirium, or if physical signs of focal damage are revealed on examination. During the initial period of unconsciousness the patient is pale, with shallow respiration, the pulse is increased in rate and feeble. The temperature is subnormal, musculature is relaxed, and the reflexes are diminished. Following this state of cerebral shock, which is also seen for a short while in concussion, a phase of reaction develops, the respirations become deeper, the pulse increases in tension and the face becomes flushed. On recovery of consciousness patients complain of headache or photophobia

and often vomit and exhibit confusion, irritability, or delirium, indicating damage to the cortical or hypothalamic levels of the brain.

In cerebral irritation the body assumes a position of flexion, the patient curls up in bed with knees drawn up and arms flexed and interference of any kind is resented verbally or physically. In serious cases deep coma persists from the moment of injury; dilated and inactive pupils, Cheyne Stokes' respiration and irregular pulse indicate a brain stem lesion which will probably prove to be fatal. In less serious cases physical signs such as anosmia, aphasia, or hemianopia, may be detected on recovery of consciousness but not before. If the brain surface is lacerated the patient may suffer from stiff neck, pain in the legs, and retention of urine due to the irritation of blood in the cerebrospinal fluid. Retrograde amnesia involving loss of memory for events prior to the accident and post traumatic amnesia vary in duration according to the severity of the injury. In cerebral contusion post traumatic amnesia continues for many hours or days after apparent recovery of consciousness has been observed.

Secondary pathological changes leading to a rise in intracranial pressure occur at an interval after injury and include:

(a) Local swelling in the region of surface bruises, retention of fluid in the lateral ventricles, blockage of the basal cisterns and arachnoid granulations by effused blood, with defective absorption of cerebrospinal fluid.

(b) Massive intracranial hæmorrhage, extradural, subdural, or intracerebral.

These additional factors may produce a fatal cerebral compression in a patient whose neuronal injury could otherwise have been survived.

Analysis of the mortality of head injury shows that deaths in the early stages are usually produced by the effect of the neuronal lesion alone representing an immediate mortality which is irreducible, but that in deaths occurring after twenty-four hours there are many cases of unrecognised cerebral compression.

The treatment of closed head injury consists of:

(1) Immediate clinical examination to determine the severity of the neuronal lesion and establish a base line for observation.

(2) Nursing care to counteract the effects of the neuronal lesion by posture, attention to airway, nasal feeding, and catheterisation when required.

(3) Continuous observation to detect the onset of œdema or intracranial hæmorrhage and the necessary measures to deal with these complications (p. 362).

(4) The treatment of skull fracture, and its complications, if present (p. 369).

Clinical Examination.—The head is examined for evidence of surface bruising or fracture. An X-ray of the skull is essential in all cases from the medico-legal aspect, if not from the clinical. The neck is tested for stiffness. Cervical spinal injury may be present in head injury. The central nervous system is examined in so far as the state of consciousness permits. Particular attention is paid to the level of consciousness, the duration of unconsciousness, the size, equality, and reaction of the pupils,

movement or paralysis of either side of the body. *Physical signs are carefully noted, so that the subsequent appearance of new signs can be checked.* *Nursing Care.*—*The patient must be placed in a position where regular observation can be maintained by the nursing staff.* Seclusion in a dark room is only justified in a patient who has recovered consciousness and complains of photophobia (abnormal sensitivity to light).

Posture.—*The unconscious patient* must be nursed on his side, so that secretions may dribble from the mouth. Patients may drown in their own secretion, become cyanosed, and deeply unconscious if allowed to remain on their backs (this may be fatal during transport over long distances). Careful attention is given to the airway to be sure the tongue is not swallowed. If the patient is bubbling, the throat must be cleared by suction. The patient is turned regularly. Physiotherapy and postural drainage in Coleman's position for an hour at a time help to prevent pneumonia supervening (fig. 421). *Tracheostomy* is required to keep the airway clear in cases of brain stem lesion when reflexes are lost and unconsciousness is prolonged.

The delirious patient should not be forcibly restrained; restraint provokes aggression. A cot bed covered with netting beneath which the patient may move at will is the best solution.

The conscious patient should be propped up or lie flat, according to which relieves his high- or low-pressure headache.

Elderly patients with chest injuries or complications must be treated propped up.

Catheterisation is performed if necessary; a full bladder induces restlessness.

Nasal tube feeding is required if unconsciousness is prolonged.

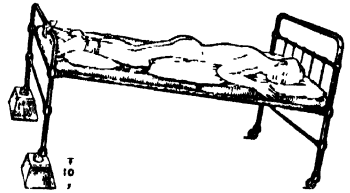


FIG. 421.—Coleman's postural drainage for the unconscious. The position is maintained by pillows, and bandages anchor the ankles to the foot of the bed.

Continuous observation is maintained from the outset, including a half-hourly record of the pulse and regular observation of the state of consciousness, of the size and reaction of the pupils, and the power of movement on the two sides of the body.

Progressive slowing of the pulse and deterioration of consciousness from co-operation to confusion, to stupor, to coma, indicates cerebral compression, probably due to oedema or hæmorrhage.

A deterioration of consciousness, together with the *appearance of new localising signs*—e.g. pupil changes or paralysis occurring at an interval after injury—*indicates focal compression by hæmorrhage* and should be regarded as an absolute indication for immediate burr-hole exploration, at the site indicated.

In doubtful cases where the patient is deteriorating and there are no new signs, lumbar puncture, echo encephalography, and arteriography may be needed to establish the cause.

Lumbar Puncture is helpful as a diagnostic measure to determine the pressure by manometry and to establish whether pressure is abnormally high or abnormally low. Raised pressure with crystal clear fluid is strongly

suggestive of hæmorrhage outside the membranes, for in cerebral contusion the fluid is usually slightly blood stained and the pressure high. In serious brain stem lesions the pressure is often abnormally low. If focal hæmorrhage can be excluded a phase of compression due to secondary oedema is treated by dehydration therapy. Dehydration will remove that part of the syndrome produced by secondary compression but the effect on the level of consciousness will not be to return the patient to normality but to return him to the level permitted by the underlying neuronal lesion, thus a patient originally confused and disorientated who became stuporose and comatose will return to a state of confusion.

Dehydration Therapy.—An increase in the osmotic tension of the blood diminishes cerebrospinal fluid formation at the ventricles, and also increases absorption at the lacunæ, with reduction of intracranial pressure. Six ounces (180 ml.) of 60 per cent. mag. sulph. is given slowly as a warm rectal infusion twice daily, and retained for twenty minutes to extract fluid from the circulation. The fluid intake is restricted to 2 pints (1,000 ml.) in twenty-four hours.

In more serious cases 50 ml. of 50 per cent. sucrose may be given slowly intravenously four times in twenty-four hours; the effect lasts six hours. Neither of these measures should be continued for longer than three days, owing to the risk of altering the sodium-potassium balance by prolonged dehydration.

The diuretic action of urea may be used for dehydration in patients whose renal function is normal. Increased concentration of urea in glomerular filtrate hinders reabsorption of fluid at the renal tubules and therefore diuresis results. A 30 per cent. solution in 10 per cent. invert sugar should be freshly prepared. Stored samples must not be used after ten days storage at 2–5° C. The solution is given slowly at not more than 60 drops per minute, which is sufficient to lower intracranial pressure markedly within twenty minutes. The method is useful in the treatment of head injuries when the patient is stuporose or comatose, when the lumbar pressure is known to be high and large clots have been excluded. It is also of value in reducing intracranial pressure at craniotomy, and relieving comatose patients suffering from intracranial tumour in whom the improvement resulting from lowering pressure may secure co-operation at examination or facilitate transport to a suitable hospital. Despite its advantages the method increases the bleeding experienced at operation from the scalp, the epidural vessels, and cortical vessels, owing to the increase in blood-pressure, and for this reason excessively rapid administration must be avoided.

Therapeutic lumbar puncture on alternate days is employed to remove 4 or 5 ml. of the most heavily bloodstained fluid in cases of cerebral laceration with gross extravasation.

Special measures are required in cases of serious brain stem injury. The combined use of tracheostomy, intravenous aminophylline, and hypothermia, reduce some of the mortality from serious brain stem lesions. *Intravenous aminophylline*, 250 mg. intravenously, by its vaso-dilator action improves the blood supply in cases of brain stem ischæmia and can relieve Cheyne-Stokes respiration. The respiration is also improved by dilatation of the bronchi.

Hypothermia.—The survival of serious brain stem lesions may be prolonged by the induction of hypothermia by the use of ice bags, regular cold sponging, and ice blankets. This serves not only to diminish the rise in temperature or hyperthermia, often seen in brain stem lesions, but also by

minishing the oxygen requirements of damaged tissue, improves the prognosis.

Drugs.—The fewer and the simpler, the better. Paraldehyde is required to control restless cases. Bromide should not be used, particularly in the elderly, as this is liable to provoke delirium.

Conscious patients require little more than aspirin for headache and, occasionally, barbiturates for insomnia.

Convalescence.—Minor contusions recover speedily, but in serious cases prolonged convalescence is needed. Account should be taken of the severity of the injury as measured by the duration of the post-traumatic amnesia; also the mental make-up of the patient and the amount of concentration associated with his occupation, in assessing the duration of convalescence. Usually some symptoms of the post contusional state consisting of headache, giddiness, defective memory, defective concentration, irritability, impaired emotional control, impaired sleep, or susceptibility to alcohol, persist for a period of eighteen months. In severe cases post-traumatic dementia constitutes a permanent handicap sometimes necessitating Institutional care. Schizophrenia is a rare complication of even a minor injury.

Cerebral Compression.—The detection and treatment of lesions producing cerebral compression is the most important factor in reducing the mortality from head injuries. Cerebral compression gives rise to progressive deterioration in level of consciousness, slowing of pulse and slowing of respiration with or without, the appearance of new localizing signs. Acute brain swelling and œdema in areas of infarction around contusions may produce urgent compression in patients who have already sustained a serious cerebral contusion. In such cases compression may occur without new localising signs. Syndromes of traumatic intracranial hæmorrhage may arise in patients who have only sustained an initial minor concussion followed by a period of recovery of consciousness after which the patient becomes drowsy and lapses again into unconsciousness.

An unconscious footballer was carried off the field but later recovered sufficiently to finish the match; headache and drowsiness supervened so that he retired to bed early and was later found dead from middle meningeal hæmorrhage. Similar tragedies have occurred at school from cricket or on the links from golf balls.

The majority of cases of middle meningeal hæmorrhage are due to an injury associated with fracture of the temporal bone, bleeding from this fracture line causes an extracranial extravasation producing boggy swelling deep to the temporal muscle, therefore all cases of even minor concussion must be under observation for 24 hours if there is any bruising in the temporal fossa. Focal compression produced by a clot is usually accompanied by the appearance of new localising signs produced by the local pressure.

When syndromes of hæmatoma formation develop in patients already deeply unconscious as the result of cerebral contusion a lucid period is absent and the collection of the hæmorrhage can only be recognised by determining

the alteration in the level of consciousness and the appearance of the new localising signs.

With patients under observation, a deterioration in the level of consciousness, with the appearance of new localising signs not observed at the original examination, must always be taken as an absolute indication for exploration of the area indicated by the localising signs. The formation of a burr hole opening under local anæsthesia is sometimes the only method of recognising or excluding a dangerous complication. Similar signs are occasionally produced by œdema spreading from adjacent bruises; but this cause should never be assumed until the possibility of hæmorrhage has been disproved. If acute compression should develop and there are no localising signs and if a fracture is present, a burr hole must be made in the region of the fracture in case extradural bleeding has occurred from the bone. Even in the absence of signs or fractures, a progressive deterioration in consciousness, drowsiness, headache, and slowing of the pulse, is always an indication for urgent investigation. If available, an ultrasonogram is valuable, an echo encephalogram taken at the time as a skull X-ray will indicate a shift of the mid-line structures and suggest the presence of a unilateral expanding lesion.



FIG. 422.—Antero-posterior arteriogram showing (1) displacement of cortical vessels away from the inner table of the skull and (2) displacement of the anterior cerebral artery across the middle line produced by a moderate associated subdural hæmatoma on the right (see arrows).

Carotid arteriography may often demonstrate the site of the lesion:

1. By displacement of the cortical vessels away from the inner table of the skull in subdural hæmatoma (fig. 422).

2. By inward displacement of the middle cerebral artery and displacement of the anterior cerebral across the middle line in extradural hæmorrhage.

3. By upward displacement of the middle cerebral artery in œdema or subcortical bleeding in the temporal lobe or a subdural subtemporal hæmatoma.

4. By transverse displacement of the anterior cerebral in acute swelling or subcortical hæmatoma in the frontal lobe. Only very rarely is compression due to an extensive area of indriven bone and in these rare cases evidence of compression is

likely to follow immediately after the injury has occurred.

Traumatic intracranial hæmorrhage is of three types: (1) Subcortical; (2) Extradural; (3) Subdural.

Subcortical hæmorrhage is produced by arterial bleeding either extending from an area of surface laceration or produced by rupture of a central artery. The bleeding may terminate rapidly and fatally by rupturing into the ventricles, causing hyperthermia and death by *intraventricular hæmorrhage*; it may, however, form a clot under the cortex.

Clinical features combine evidence of cerebral compression and epileptic fits. In the frontal lobe—cerebral compression associated with generalised epileptic fits and few signs. In the temporal lobe—cerebral compression, fits, and hemiparesis. Similar syndromes are sometimes produced by swelling of infarcted areas of the lobe following surface contusion without actual clot formation. Symptoms of clot formation are usually observed within the first twenty-four hours after the injury, or the onset may be delayed for a further ten days if the artery ruptures late as a result of softening near a surface bruise (delayed apoplexy of Bollinger). Chronic clots may simulate a cerebral tumour months after the injury.

Treatment.—Arteriography may be required to localise the site of the lesion. Fluid blood may be aspirated through a ventricular cannula. Solid clots may be evacuated or removed by suction after exposure of the cortex through a small osteoplastic flap or a large trephine opening. Areas of acute brain swelling and œdema resulting from infarction are treated by local decompression and removal of softened areas by suction. Pre- and post-operative use of urea greatly diminishes the need for removal of softened tissue.

Extradural Hæmorrhage of Arterial Origin.—Extradural hæmorrhage of arterial origin may arise from branches of the *internal maxillary* or *anterior meningeal* vessels torn by fractures of the anterior fossa. In such cases a deterioration in level of consciousness occurs in association with an antero-basal fracture, but usually there are no accompanying localising signs.

The classical syndrome of extradural hæmorrhage results from injuries of the anterior or posterior branches of the *middle meningeal artery*. In such cases the injuring force, which comes from a lateral direction, is often relatively trivial, such as a blow from a golf or cricket ball which strikes the thin bone of the temporal plate, inflicting a fracture which drives the dura inwards and in the case of anterior branch bleeding, snaps the meningeal artery at a point where it leaves the bony canal in the pterion and crosses to gain attachment to the dura mater. In the elastic skull of children the injury may occur without fracture, but in all adults, comprising 90 per cent. of cases, X-ray reveals a fracture line crossing the middle meningeal groove.

The detection of this fracture is an absolute indication for the admission of the patient for observation.

From the torn vessel blood passes in three directions :

(1) Outwards through the fracture line to form a boggy swelling under the temporal muscle, the finding of which is an additional indication for the admission for observation of a conscious patient.

(2) Downwards into the middle fossa.

(3) Upwards over the motor cortex.

Clinical Features.—Usually a laterally directed blow of small magnitude causes a short initial period of concussion, followed by a characteristic 'lucid interval',¹ during which the hæmorrhage is collecting intracranially and also forming a superficial swelling under the temporal muscle. The

¹ When the complication develops in a patient who never recovers consciousness, it would be detected only by clinical signs.

importance of this sign cannot be overestimated, and one may frequently forecast the development of the complication on the basis of this sign alone. The next change is in the level of consciousness. The patient becomes confused and irritable, and at this stage is in danger of being arrested if found wandering and smelling of alcohol ; therefore, beware of the patient brought to Casualty in a confused state with a bruise in the temporal region. Persons alleged to be found drunk have been locked in cells only to be found dead in the morning, the unconsciousness and stertorous breathing of compression being mistaken for a drunken stupor. Confusion changes to drowsiness, and at this stage pressure of the clot on the motor cortex produces cerebral anoxia, causing twitching, followed by paralysis of the face, then the arm, then the leg, on the opposite side of the body as the clot spreads upwards over the motor cortex. At the same time inward displacement of the temporal lobe causes the inner portion of the lobe to press against the third nerve above the edge of the tentorium (fig. 423(B)), causing restriction, rapidly followed by dilatation of the pupil on the side of the hæmorrhage. If the

pressure is not relieved, displacement of the brain stem at the tentorial opening forces the opposite crus against the rim of the tentorium, producing further hemiplegia, which this time occurs on the side of the hæmorrhage (fig. 423 (A)).

Finally, impaction of a mid-brain cone produces decerebrate rigidity and fixed dilatation of *both* pupils, at which stage the case is probably too late to remedy by operation.

In the presence of these confusing signs, the side of operation is determined by :

- (a) The site of bruising in the temporal muscle.
- (b) The side of skull fracture.
- (c) The side of the initial dilatation of the pupil.

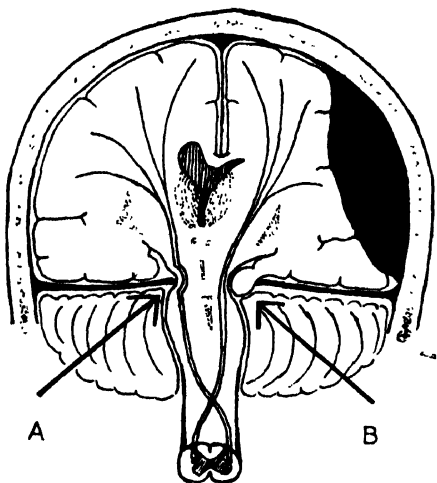


FIG. 423.—(A) Impaction of the opposite crus against the opposite rim of the tentorial opening. (B) Displacement of the inner edge of the temporal lobe (uncus) descending into the tentorial opening (mid-brain cone). (G. B. Northcroft, F.R.C.S., London.)

Posterior branch bleeding is less than one fifth as common. Here the clot is situated at a distance from the motor cortex, the surface bruising is situated farther back, or there may be a steady arterial hæmorrhage from the ear when the fracture line involves the middle ear. The level of consciousness deteriorates, and the patient passes into a stage of cerebral irritation or stupor with a dilated pupil from pressure inwards on the third nerve, but there may be no motor paralysis for several days, or the paralysis appears on the same side of the body as the hæmorrhage as the result of brain shift and impaction of the contralateral crus. The site of operation is determined by the site of the fracture, surface bruising, and dilatation of the pupil.

Treatment is an urgent matter. Patients may stop breathing within half to one hour of the onset of the first sign ; therefore the dangers of trans-

port must be assessed carefully. If possible, operation should be performed in the hospital where the complication is recognised, especially if the case is deteriorating rapidly. If the site of the hæmorrhage is definitely established, half the head may be shaved. If there is any doubt or the possibility that an alternative exploration for a subdural hæmatoma may be required, the whole head should be shaved, as subdural hæmatomas are often bilateral.

A low temporal osteoplastic flap gives an excellent exposure of all branches and is far the best method of approach. Alternatively a craniectomy is formed through a curved hook incision (fig. 424 (A)) centred on a point 2 inches (5 cm.) behind the eye and 2 inches above the zygoma which corresponds primarily to the anterior



FIG. 424.—(A) Curved hook incision used to expose artery. (B) Position of bone opening. (C) Exposure of artery which is underrun by a dural stitch.

branch of the artery. On division of the temporal muscle blood clots should be found between the muscle and the fracture line. If there is no fracture one should suspect an error in diagnosis and the compression will probably be found to be due to subdural hæmatoma. Normally, a burr hole is made beside the fracture and the skull is opened, the bone opening being enlarged with nibbling forceps as required. The bleeding point may be seen lying on the dura (fig. 424 (B)) and may be secured by underrunning with a fine curved needle or coagulated with diathermy. If the torn vessel is bleeding from the bony canal, the bone is plugged with Horsley's bone wax which stops the hæmorrhage. Alternatively, blood may continue to trickle in from the back of the opening, indicating that the posterior branch has been involved, and the bone excision must be extended in this direction until the bleeding-point is found. Occasionally an artery is ruptured near the foramen spinosum, where it must be arrested with diathermy. When the artery has been secured, bleeding continues from veins in the margins of the compression area if the dura has been seriously displaced. Venous bleeding is controlled by placing muscle, fibrin foam, or oxycel over the bleeding-points and stitching the dura back firmly against the skull. Troublesome capillary oozing may continue from the dura, and it is often wise to drain for the first twenty-four hours.

Post-operative Treatment.—The patient should be kept lying flat in order to encourage the brain to re-expand. If the level of consciousness does not improve rapidly and the patient shows signs of persistent coning with dilated pupil, a diagnostic lumbar puncture should be performed. If the pressure is abnormally low, sterile saline should be injected intrathecally in order to raise the pressure to the normal level of 120 mm. of cerebrospinal fluid or slightly above, and so produce a free rise and

fall on jugular compression. This may necessitate an injection of 90 or 100 ml. of saline; when successful it will occasionally relieve cone formation and result in rapid restoration of consciousness. It probably does so by floating the brain stem up into its normal position, thus straightening out the vessels supplying the stem.

Extradural hæmorrhage of venous origin is produced by fractures which injure the major sinuses. Injury of the superior longitudinal sinus produces a massive subgaleal hæmatoma, together with evidence of clot compression, causing deterioration of consciousness with generalised epilepsy if the clot lies over the frontal areas, or unilateral or bilateral leg weakness with extensor plantar responses if the clot is over the upper end of one or both motor cortices.

Extradural hæmorrhage of the posterior fossa is produced by injury to the transverse sinus. Surface bruising appears behind the ear. There is a deterioration of consciousness occurring at an interval of days after the injury. Signs of cerebellar

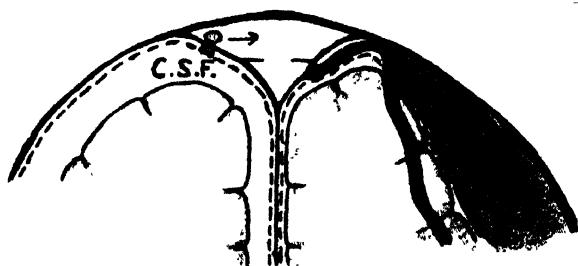


FIG. 425.—Position of a subdural hæmatoma produced by tearing of the superior cerebral veins at the level of the arachnoid. The diagram also shows the partial or occasionally complete septum which divides the superior sinus. Cerebrospinal fluid drains into the upper compartment through the arachnoid granulations. The superior cerebral veins drain into the lower. (G. B. Northcroft, F.R.C.S., London.)

compression are few; often there is merely a reduction of tone and reduction of reflexes on the side of compression.

Treatment.—Clots of this type are evacuated after elevation of the causative fracture, hæmorrhage from the sinus is arrested by holding a muscle graft firmly against the aperture whilst it is secured by an overlying letter-‘N’ stitch.

Subdural hæmorrhage is produced by rupture of the veins passing from the cerebral hemispheres to the venous sinuses as the result of displacement of the brain inside the skull. Usually the superior cerebral veins are ruptured, producing hæmorrhage over the convexity of the hemispheres; very rarely veins passing from the temporal lobe to the sphenoid or petrosal sinuses may produce clots which collect on the under-aspect of the brain. This complication, which is potentially fatal, is at least six times as common as middle meningeal hæmorrhage and is produced particularly by blows of small magnitude applied to the front or back of the head which may be insufficient to produce even transient concussion, but which are sufficient to move the brain suddenly. Cerebral atrophy renders this displacement easier, and hence the condition becomes commoner with advancing age. The superior cerebral veins pass from the convexity of the hemispheres and pierce the arachnoid membrane before crossing the potential subdural space to join the inner aspect of the dura 1 inch (2.5 cm.) or more from the middle line; they then run inwards to drain into the lower compartment of the superior longitudinal sinus.

One end of the vein is fixed to the dura, the other to the mobile hemisphere. Sudden displacement may snap the vein at the level of the arachnoid, allowing blood to pass downwards into the potential subdural space between the arachnoid and dura. Frequently, corresponding veins on both sides are

affected and the condition is bilateral in 50 per cent. of cases. The hæmatomas are often large, bilateral collections up to 60 ml. a side or unilateral collections of 120 ml. being quite usual.

In the acute form the clots produce symptoms of cerebral compression within hours or days of the injury; in the chronic form not until months after the injury. The normal pressure in the superior cerebral veins is extremely low; hence venous bleeding by itself could never compress the brain and overcome arterial pressure without one of the following additional factors. Vomiting after injury may force blood out at a high pressure and account for the acute onset of pressure occurring in a period of hours. In the subacute or chronic cases, the pressure increases as a result of the osmotic traction of the blood in the subdural space drawing fluid from the cerebro-spinal fluid system through the semipermeable arachnoid, so that the cyst increases steadily in size and produces compression symptoms in days or months after the injury.

Clinical Features.—The symptoms may follow a preceding concussion, but owing to the slight nature of the force required to produce displacement, this complication may occur without preceding loss of consciousness and without the head being even struck. It can follow a sudden jolt, as when a driver is thrown against the steering-wheel of a car, or be produced by knocking the head against the lintel of a door, or landing heavily on the feet when jumping from a height; it has followed dental extraction and electro-convulsive therapy.

In the acute form developing within hours the onset of coma, hemiplegia, and dilated pupil readily attracts attention and simulates an extradural hæmorrhage without appropriate evidence of skull fracture. In subacute or chronic syndromes however, the symptoms are undramatic and consist of (1) Headache which is unduly severe or unduly prolonged, following a blow on the front or back of the head. (2) Cerebral anoxia, causing mental apathy, slowing of cerebration, slowness of response to questions merging into stupor. (3) When the stupor develops, it comes and goes as the brain volume varies, the patient being inaccessible at times and then rousing sufficiently to answer questions accurately, but very slowly and after a considerable pause. When this phase passes on to coma as the result of the formation of a mid-brain pressure cone, the operative mortality rises from nil to 30 per cent.; hence the significance of these early symptoms.

Physical signs vary. Acute compression may cause hemiplegia in young subjects. In older patients, where more room is available and if the cyst collects slowly, there may be no signs, or at most a unilateral or bilateral extensor plantar response from pressure on the motor cortex or brain-stem displacement. Pupillary changes occur last when the brain stem is affected and pressure-cone formation is imminent. Lumbar puncture shows a fluid at low pressure with protein increased to 120 mg., often stained yellow from the transudation of pigment, but no cells. Papilloedema is exceptional. Success in treatment comes from acting on suspicion and employing arteriography, or the formation of exploratory burr holes in any suspicious case in

which excessive headache or stupor follows upon an appropriate injury without waiting for physical signs which may never develop or may only develop later when the patient has a potentially fatal pressure cone.

Treatment.—Bi-parietal burr holes are made under local anæsthesia to expose the dura, which is often stained blue-green from the blood pigment. On incising the dura, black tarry blood is removed by suction through a soft rubber catheter (fig. 426), which should be passed downwards and



FIG. 426.—(A) Site of clot. (B) Lateral diagram showing extent of clot and site of incision. (C) Burr-hole exploration. Emptying of cyst with catheter.

also backwards in the subdural space. If the blood is thick, bi-frontal burr holes are fashioned and the subdural space is washed through with saline. Solid clots must be removed by craniotomy. The results are excellent if the operation is performed before a mid-brain cone develops.

Post-operatively, the patient is nursed with the head low in order to encourage expansion of the brain. Repeat X-rays show absorption of air from the subdural space. Rarely, repeat of the puncture is required if the brain is not expanding or if symptoms persist, indicating that the clot has only been partially evacuated.

FRACTURES OF THE SKULL

Mechanism.—Fractures of the vault and base of the skull are produced : (1) by compression of the sphere ; (2) by local indentation ; (3) by tangential injury. These mechanisms vary, not only in their local effect upon the skull, but also in the severity of the cerebral injury that accompanies the fracture. The mechanism can be visualised when interpreting the skull X-rays.

Fractures of the vault produced by compression of the sphere are the commonest skull fractures, and result from distortion of the skull when it comes into contact with a hard flat surface. Usually closed linear fissures are produced which, although not important in themselves, are often associated with severe diffuse cerebral injury resulting from brain displacement occurring at the moment of impact. Contact with a flat surface renders the spherical skull more ovoid. The fracture line starts at the points of maximum convexity thus produced and spreads through thin areas of bone, being deflected at an obtuse angle from the bony buttresses of the temporal and

ipital crests towards the base of the skull ; hence many fractures of the base are produced by the extension of fissures starting in the vault.

Fractures of the vault produced by local indentation give rise to closed or compound depressed fractures according to the surface area of the injuring agent.

Blows from large rounded objects produce *closed pond depressed fractures* ; the scalp bruised but remains intact. The skull fractures at the points of maximum convexity ; is dented but not indriven, the underlying dura remaining intact. The brain surface bruised under the intact dura, but not penetrated. This bruising may cause local signs, such as paralysis or aphasia. Healing occurs by absorption of the tips of damaged convolutions forming a shallow erosion on the brain surface, surrounded by a zone of reactive gliosis. The risk of epilepsy is about 4 per cent. (fig. 427).

Blows from small round objects produce *compound depressed fractures*. The scalp is irregularly torn by the indentation. The fractured bone is depressed, lacerating both the dura and subjacent brain. Fragments of bone may separate from the inner table and lodge in the brain. There is immediate risk of infection, and a later risk of epilepsy resulting from the contracting fibrous scar through the open dura, which increases the incidence of epilepsy to 25 per cent. in untreated cases (fig. 428).

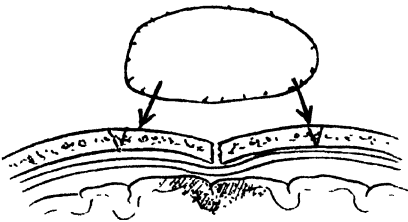


FIG. 427.—Pond fracture produced by large round object.

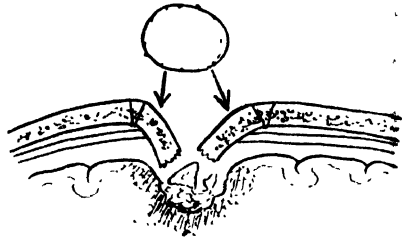


FIG. 428.—Compound fracture produced by small round object.

Fractures of the Vault by Tangential Injury.—Tangentially directed violence may secure a grip on the skull and prise up large segments of bone, which are elevated leaving the dura intact.

When a window cleaner, falling to earth, catches his brow on a window ledge, the vault of the skull can lift like a lid of a coffee pot, a horse-shoe fracture surrounding the calvarium. Such injuries are compound and the skull X-ray is alarming, but there may be little or no associated cerebral injury, as the injuring force was not directed on to the brain. In one case the swinging door of a railway wagon, striking above the right ear, prised up both frontal plates and the frontal sinuses which were hinged laterally on the left temporal muscle, but the patient was unconscious for only half an hour.

Treatment of fissured fractures of the vault is governed by the degree of associated injury to the brain. A careful watch is maintained for evidence of cerebral compression, indicative of bleeding from the fracture line.

Closed depressed fractures of the vault are rare. The scalp is relatively non-elastic and an injury sufficient to fracture and depress the bone is almost certain to lacerate the scalp. Hæmatomas in the scalp sometimes simulate depressed fractures. Clotted blood in the margin of a hæmatoma may, on palpation, resemble the edge of a fractured zone (fig. 429) but the edge of the clot can sometimes be indented, often X-ray is required to distinguish the two conditions.

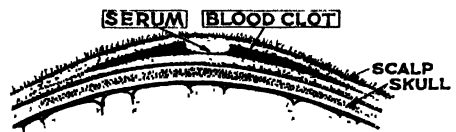


FIG. 429.—Hæmatoma simulating a depressed fracture.

The **treatment** of a closed depressed fracture depends on the extent and site of the depression. Operation is not indicated if the fracture is small provided that

there are no sharp spicules of bone which have penetrated the dura (fig. 430) and the fracture does not overlie an important cerebral area. Surgical intervention is required if the fracture lies over the speech or motor areas, or if the X-ray suggests that spicules may have penetrated the dura mater, when scarring may cause adhesions between the brain and membranes, with risk of subsequent epilepsy (p. 375).

Operation.—Merely to trephine and elevate the depressed bone is quite inadequate, as a proper examination of the brain and membranes is not possible. An osteoplastic flap, which includes the fracture, is raised (fig. 430), and the depressed bone is either moulded into position or removed with suitable forceps. The dura is examined, penetrating spicules are removed, and the membrane is then opened. Pulsed brain is carefully removed by gentle suction at the site of penetration. Adhesions are separated. Hæmostasis is secured and the dura is carefully sutured, if necessary a fascial graft being inserted to prevent the ingrowth of fibrous tissue. The flap is then replaced and the wound closed.



FIG. 430.—Sharp bone spicules penetrating the dura.

Pond-shaped depressions occur in infants, as a result of prolonged pressure of the head against the promontory during birth, or of direct injury during the first few months of life. Spontaneous elevation usually occurs within a few weeks. Early elevation is required if an extensive depression overlies the speech or motor areas or if there are signs of compression. Operation is also indicated if a depression anterior to the hair line constitutes a cosmetic deformity which does not undergo a spon-

taneous elevation within six weeks. As the formation of an osteoplastic flap would lead to extensive damage to the extremely thin dura of an infant, a simple elevation is performed by means of a guarded gimlet, i.e. silk is wound around the gimlet to within $\frac{1}{4}$ inch (6 mm.) of its point. A small incision is made over the centre of the depression, and the skull is perforated by the gimlet. An aneurysm needle is introduced through the aperture, and by this means traction is applied to the depressed bone. The elastic skull of the infant readily yields and springs back to its normal position.

Compound fractures of the vault vary in severity, from severe crushes, with the escape of cerebral substance, to relatively insignificant compound fissured fractures. High-speed motor-cycle accidents produce the worst examples of severe frontal crushes, many are rapidly fatal. Severe cases which survive exhibit serious disability, such as hemiplegia or mental deterioration from loss of cerebral substance. The incidence of these injuries has been reduced by the general acceptance of crash helmets.

When the velocity of impact is low, large compound depressions can occur with less diffuse cerebral injury, particularly when produced by blows from the side—in which the midline septum of the falx will limit the associated brain displacement.

Rarely, if the skull is fixed at the moment of impact, as when the head is trapped against the edge of a kerb, the skull may be crushed, with injury to cranial nerves, without any loss of consciousness at all, because the brain is not suddenly displaced.

Clinical Features.—An irregular lacerated wound of the scalp is associated with a boggy subgaleal swelling produced by blood escaping from the fracture line, which is partly exposed in the depth of the wound. A normal suture line should never be mistaken for a fissured fracture for a

fracture line is irregular and free from oozing blood. All scalp wounds should be explored adequately in order to exclude or discover a fracture. In compound depressed fractures the fracture lines extend far laterally beyond the limits of the surface wound (fig. 431), and this must be allowed for in planning the requisite exploration.

Treatment.—Operation should be undertaken as soon as possible to avoid infection unless the patient's condition is so precarious that delay is advisable, in which case antibiotics should be given in full doses.

Pre-operative X-ray is essential to determine the extent of the fracture.

The head is shaved well beyond the margins of the fracture.

Unconscious patients may be operated upon under local anaesthesia.

In frontal fractures heavy bleeding from the nose must be cleared by suction.

The field of operation is infiltrated with 1:100,000 adrenalin in saline to diminish scalp bleeding. Small compound fissure fractures may be exposed by extending the primary scalp wound after excision of its edges. Extensive depressions are exposed by forming a horseshoe scalp flap around the margins of the fracture site after previous excision and suture of the scalp wound (fig. 432). A burr hole is formed beside the fracture to permit unlocking of the fractured particles (fig. 433). Bone which has been contaminated by dirt is discarded. Clean bone is preserved. The dura is inspected.

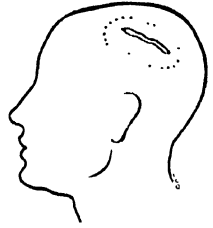


FIG. 431. — Relative extent of the fractured area (shown by dotted line) in comparison with the surface wound.

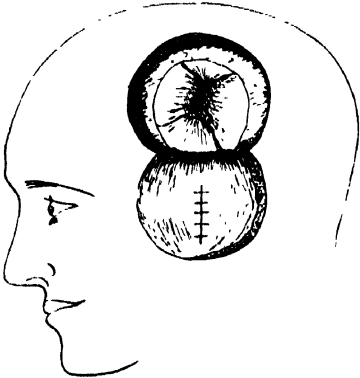


FIG. 432. — Exposure of depressed compound fracture. The original scalp wound has been sutured and a horseshoe scalp flap reflected to expose the full extent of the fracture. (G. B. Northcroft, F.R.C.S., London.)



FIG. 433. — Formation of burr hole beside a fractured area to allow of unlocking the bone fragment.

If intact, it is not opened. If penetrated, indriven material and damaged brain tissue are removed by suction and the dura is then sutured. Clean portions of bone may now be moulded into place, the wound being then sutured and antibiotic treatment commenced. Careful judgement is required concerning the discarding of bone. If a tangential injury has lifted up large areas of the vault, the frontal plates, or roof of the orbit, far beyond any *exposed wound*, it is only necessary to remove contaminated areas in the wound. Removal of the entire detached area of bone would produce enormous unsightly deformities which could not be adequately restored.

Compound fractures of the frontal sinus become infected from the nose; hence the sinus must be obliterated. After excision of depressed bone and removal of the mucous lining a pressure pad is applied over the scalp to cause the scalp to

sink into the depression resulting from excision of the sinus and to close the cavity by adherence to the dura. Three months later the sinus area is exposed by turning a scalp flap forward, the scalp is freed from the dura, and the deformity is restored by skull reconstruction.

Skull Reconstruction.—Areas of skull defect are restored at intervals of three to six months after injury, by the insertion of moulded tantalum plates or acrylic inlays. Slow setting acrylic powder and monomer when mixed together form a plastic dough which gradually hardens to form a sheet having the consistency of rubber. Within thirty minutes this may be cut to shape and inserted to fill a bone defect over an intact dura. Adaptation to the local curvature of the dura produces a perfect reconstruction of the skull outline when the acrylic hardens *in situ* during the following twenty-four hours. Concealed horseshoe or transverse incisions within the hair line are used for these purposes.

Head Wounds due to Missiles.—The outcome depends on the explosive impact of the missile in the cranium, which is the commonest cause of fatality, and the relation of the wound track to the great vessels and the ventricles. Through-and-through tracks, from side to side or front to back, may be survived. Survivable injuries can be transported to a suitable hospital. Projection of the swollen brain through the dura at the site of entry or exit forms a hernia cerebri, which seals off the subarachnoid space during transport (fig. 434). A pressure dressing is applied.

Operative treatment consists of the excision of the surface wound and suction, cleansing, and removal of foreign material from the track. The dura is closed by suture or grafting at entry and exit points. Intrathecal penicillin treatment will then prevent serious infection unless an infected track crosses the ventricles or median fissures (see also p. 378).

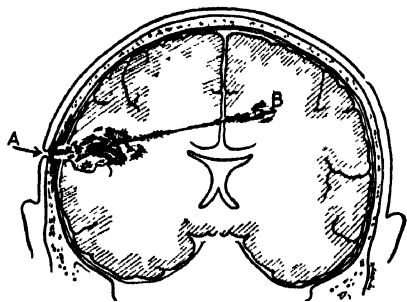


FIG. 434.—Transverse penetrating wound, entry at (A) showing: (1) Protrusion of brain through the dura at entry site forming hernia cerebri. (2) Pulped brain and bone close to entry. (3) A metal fragment at (B) has crossed the open superior fissure.

Penetrating wounds produced by sticks are always to be regarded very seriously. Although scissors or pokers may enter the roof of the orbit when a child falls and thereafter be successfully withdrawn, a stick which goes in through the orbital roof, or backwards behind the orbit into the temporal lobe, cannot be withdrawn intact; portions which are left behind may, from previous contact with the ground, be infected with gas gangrene or tetanus, with probable fatal infection.

Fractures of the base of the skull are usually produced by compression of the sphere and extension of fissures radiating from the vault. The complex fracture lines thus produced are accounted for by deflection of the fissure from the buttresses of the petrous bones, the basi-sphenoid, and the sphenoid wings, which can only be crossed respectively at the weak points formed by the cavities of the middle ear, the sphenoidal air sinus, and the sphenoidal fissure. Elsewhere the fracture lines tend to wander into the foramina for the exit of cranial nerves. The fractures become compound at the middle ear, accessory air sinuses, and the cribriform plates, but the evil reputation of fractures of the base is derived from the severe brain injury accompanying the fracture rather than from the risk of sepsis, which has been greatly reduced by modern methods of treatment.

Fractures of the Base by Local Indentation.—A ring fracture sometimes forms around the foramen magnum from the upward impact of the spinal column in fatal head-on collisions. Violence transmitted from the maxilla to the internal

angular process may rotate the cribriform plate upwards on to its side, with laceration of the anterior fossa dura, a common cause of cerebrospinal rhinorrhœa in association with injuries of the middle third of the face. Rarely, the roof of the orbit may be penetrated by direct violence from sticks, umbrella ferrules, scissors, or similar objects.

Fractures of the Base produced by Tangential Injury.—The mastoid process is sometimes knocked off by an oblique blow, e.g. from the edge of a kerb; this is a very painful but relatively harmless lesion.

Clinical Features.—The essential features are :

(1) The escape of cranial contents, i.e. blood, or cerebrospinal fluid, or rarely, brain.

(2) Injury to cranial nerves. All cranial nerves may be involved but the twelfth is rarely affected. As the condyloid foramen is protected by a stout ridge of bone, the fracture line is usually diverted into the foramen magnum. Laceration of nerves in fracture lines produces an immediate paralysis which will not recover. Compression of nerves by blood clot produces paralysis which, after a period of a few days, usually recovers. Paralysis occurring after a period of weeks is produced by contraction of scar tissue or callus and will not recover.

ANTERIOR FOSSA

(1) **Escape of Cranial Contents.**—Epistaxis occurs if the cribriform plate is involved. Should the fracture extend into the orbit, an effusion of blood follows. This is distinguished from a 'black eye' by the following features :

- (a) The skin round the orbit is not damaged.
- (b) The orbital effusion occurs some hours after the injury (fig. 435).
- (c) The eye is sometimes pushed forwards, as the extravasation occurs into the tissues at the back of the orbit.
- (d) This extravasation impedes the action of ocular muscles, and movements of the eyeball are limited.
- (e) Should subconjunctival hæmorrhage follow, the patch is wedge-shaped with the apex in front, and the posterior limit cannot be seen.

Cerebrospinal fluid trickles through the cribriform plate, often in surprising quantities, a condition referred to as 'traumatic rhinorrhœa'.



FIG. 435.—Fracture of the anterior fossa. Effusion of blood appeared twelve hours after injury. No bruising of skin.



FIG. 436.—Intracranial ærocœle collecting in the frontal lobe from a fracture involving the posterior wall of the frontal sinus.

Traumatic rhinorrhœa may persist for several weeks. Air may enter the cranial cavity producing an intracranial ærocœle (fig. 436). In view of the risk of meningitis, operative closure is indicated, the torn dura being sealed with a patch of fascia lata.

Injury to Nerves.—The olfactory nerve is frequently torn, but unless its fellow is also damaged, the partial anosmia is likely to pass unrecognised. The optic nerve usually escapes, but if it is contused, any loss of vision will be permanent. The third, fourth, first division of the fifth, and sixth nerves may be involved at the sphenoidal fissure. Third nerve palsy produces a dilated pupil in a conscious patient.

MIDDLE FOSSA

Escape of Cranial Contents. Epistaxis occurs if the fracture involves the nasal sinuses.

Escape of cerebrospinal fluid and blood from the ear is common. Blood from torn tympanic membrane will clot, but when blood continues to drip for days, it is because it will not clot, owing to mixture with cerebrospinal fluid.

Serious arterial hæmorrhage from the ear may follow injury to the posterior branch of the middle meningeal artery (p. 363).

Injury to Nerves.—The facial nerve is commonly injured at the time of the accident. Usually the nerve is torn and paralysis is permanent. Occasionally paralysis occurs at an interval of a few days after injury from compression by blood clot. Recovery is to be anticipated if the facial muscles are supported and treated by electrical therapy. If paralysis develops some weeks after the injury, it is produced by pressure from fibrous tissue or callus, and the paralysis will be permanent. The eighth nerve is sometimes injured, with resulting deafness. The sixth nerve is occasionally implicated, with the resulting internal strabismus.

POSTERIOR FOSSA

Escape of Cranial Contents.—Extravasation of blood in the suboccipital region produces a boggy swelling at the nape of the neck, or discoloration posterior to the mastoid process.

Injuries to Nerves.—The ninth, tenth, and eleventh nerves are occasionally damaged at the jugular foramen, but the hypoglossal nerve usually escapes, as it is protected by a bony buttress.

Treatment is that of the associated brain injury. The patient is propped up to lower pressure and diminish the escape of cerebrospinal fluid. Antibiotics are administered. If the discharge persists undiminished for ten days, fascial repair of the dural gap is required. The skull is X-rayed to see if an intracranial ærocoele has developed in association with cerebrospinal rhinorrhœa. Air collections may be observed either in the substance of the frontal lobe or in the subarachnoid space, occasionally filling the ventricle. Sepsis does not occur at first because the upper respiratory air cells are sterile, but later, septic granulation tissue forms outside the dura, and the moment the discharge of cerebrospinal fluid stops, meningitis arises from this source; hence an early repair is necessary.

LATE EFFECTS OF HEAD INJURY

Post traumatic headache is not always cerebral in origin. Referred pain of spinal origin resulting from associated strains in upper cervical joints may be referred through the great occipital and posterior auricular nerves to the vertex, forehead, or temple. This pain is increased by neck flexion, and since the neck is flexed during reading or writing, the pain may appear to be a post traumatic headache which is made worse by mental concentration. Examination, however, reveals tenderness over the upper interspinous ligaments and great occipital nerves. Many cases respond well to local treatment by injection of local anæsthesia or occipital neurectomy.

Post-traumatic epilepsy is of two types.

True post-traumatic epilepsy is produced by the formation of contracting fibrous scar tissue in the brain, or between the brain and the membranes (p. 369). Scar formation takes time to develop and the fits occur within six months to twenty-one years from the date of an injury which has been severe enough to lead to penetration of the dura mater or laceration of blood-vessels on the brain surface, thus allowing

fibrous tissue to mingle with the normal gliosis of brain healing and so produce a contracting scar. When fits occur within days or weeks of a minor injury, they are not produced by scar formation, but by activating a latent idiopathic epilepsy which becomes evident for the first time following injury. This may be distinguished by finding gross bilateral abnormalities on electro-encephalography. In true post-traumatic epilepsy, the electroencephalogram is either normal or shows a focal lesion.



FIG. 437.—(A) Normal air encephalogram. (B) Traction on and expansion of the lateral ventricles following injury. This lesion is too diffuse for excision.

Clinically, post-traumatic epilepsy may be Jacksonian in type. Attacks of uncontrollable twitching occur commonly in the hand or thumb, spreading progressively to other muscle groups and the arm and shoulder and face before consciousness is lost and the convulsion becomes generalised. Alternatively, focal lesions in the frontal lobes cause generalised convulsions exactly like idiopathic epilepsy. In other areas a typical aura precedes a generalised fit (p. 384).

Treatment investigation by air encephalography shows whether the scar is focal or too diffuse to be excised (fig. 437). If a focal scar is present, the ventricle is drawn out to a fine point or spike at the site of contraction. Surgical treatment consists of the excision of the scar in the membranes of the brain down to the wall of the ventricle, and the repair of the dura by fascial grafting or insertion of nylon membrane to prevent more fibrous tissue growing in from the surface (fig. 438).

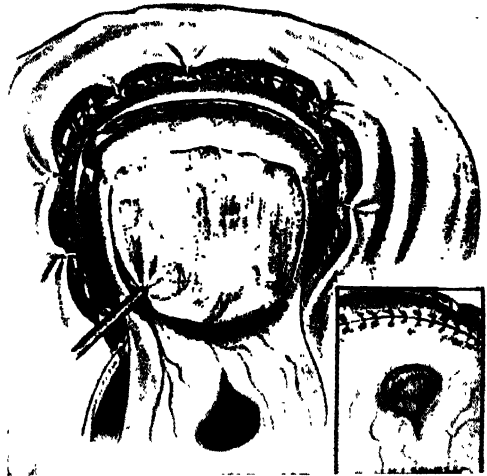


FIG. 438.—Operation sketch showing area of scar excised and insertion of nylon membrane.

INFLAMMATORY CONDITIONS

Acute infective meningitis may result from implantation of infection in infected wounds and compound fractures, from local extension from adjacent disease of the ear, or sinuses, from the various types of intracranial abscess, or be blood borne from distant foci of infection, such as staphylococcal infection in acute osteomyelitis or pneumococcal infection following pneumonia. The condition is ushered in by headache and vomiting. Severe constitutional symptoms follow, and if meninges over the convexity are affected, convulsions, delirium, and photophobia are in evidence. Should the base of the brain be chiefly involved, head retraction, papilloedema, and implication of cranial nerves are the main features.

Lumbar puncture is performed in order to clinch the diagnosis and to identify the causative organism.

Treatment consists in dealing with any causative lesion, e.g. an infected middle ear. Lumbar puncture and intrathecal penicillin administered daily (10,000 units) are valuable. Larger doses or more frequent injections are apt to cause complications, such as fits or retention of urine. Chemotherapy and antibiotics are administered according to the causative organism. The cerebrospinal fluid is examined regularly for cells and organisms. Intrathecal and systemic treatment is continued for at least ten days after the infection has subsided in order to sterilise outlying loculi and prevent relapses.

INTRACRANIAL ABSCESS

Intracranial abscess is of three types. (1) Extradural. (2) Subdural. (3) Intracerebral.

The first two varieties require immediate drainage to prevent infection of the subarachnoid space. Intracerebral abscess however must be allowed to localise. Hasty intervention in the acute stage will spread infection towards the ventricles.

Extradural abscess is produced by osteomyelitis of the skull (p. 353 for causes), and is usually secondary to spread from infection from the middle ear or frontal sinus. In the case of the middle ear, infection most commonly reaches the extradural space by extending through the tegmen tympani. Following frontal sinusitis, a large collection may form behind the frontal bone, infection having passed through the posterior wall of the sinus. Spread in the diploic veins may carry the infection to bone areas an inch or more above the ear or frontal sinus and cause local sequestration.



FIG. 439.—Pott's puffy tumour. (The late Professor Lambert Rogers, F.R.C.S., Cardiff)

Clinical features are those of osteomyelitis. Acute localised pain, acute localised tenderness, and swelling occur as in the long bones, but in the skull these are represented by acute localised headache, tenderness on local percussion of the skull, and localised pitting œdema of the scalp over the affected area, as described by Percival Pott and known as Pott's puffy tumour (fig. 439). There is a variable constitutional disturbance. These symptoms and signs alone demand urgent exploration. Only rarely, if the abscess is large, will there be evidence of pressure or focal neurological signs.

Treatment consists of drainage, the approach depending on the cause of the abscess. Many cases are dealt with by removal of the posterior wall of the frontal sinus or tegmen tympani during the course of an operation for frontal sinusitis or mastoiditis. Alternatively, a burr-hole opening is made at the site of the œdema. The dura is pressed slightly inwards to allow pus to escape. Twenty or 30 ml. may be removed by suction. Penicillin powder is insufflated and the wound drained for twenty-four hours. Antibiotic treatment serves to sterilise the bone infection.

Subdural Abscess.—This condition, at one time invariably fatal, can now be treated with a 30 per cent. mortality. It is produced by septic thrombophlebitis of the superior longitudinal sinus, spreading usually from

infections of the frontal sinus or accessory air cells. Infection extends from the superior sinus to the superior cerebral veins, and thus infects the subdural space. The abscess extends in this space over the cerebral hemispheres, often bilaterally, and must be treated before it spreads to the inner or under aspects of the hemispheres. Successful treatment depends on early recognition and intervention.

Clinical features are distinguished by marked toxæmia or dehydration and the development of intracranial pressure, epilepsy, and paralysis within a matter of days. Following a heavy 'cold' or 'influenza', the patient runs a high pyæmic temperature, becoming sallow and dehydrated with wrinkled skin. Blockage of the superior compartment of the superior sinus (see fig. 425) into which cerebrospinal fluid is absorbed from the lateral lacunæ produces raised intracranial pressure with headache and later papilloedema. Blockage of the lower compartment receiving the superior cerebral veins may cause epilepsy and paralysis of sudden onset as additional features.

Treatment.—Bilateral frontal burr holes are made just within the hair line above the temporal crest. On opening the dura, thin pus is found in the subdural space and is allowed to escape. A fine catheter is now introduced to instil antibiotics into the space. Systemic treatment is given in full doses.

Intracerebral Abscess.—Success in the treatment of cerebral abscess is determined far more by the pathological type of the abscess than by the method of treatment adopted. Some abscesses rapidly spread to infect the ventricle; others localise readily, becoming walled off, and pass into the favourable subacute or chronic stages.

Intracerebral abscesses are produced by (1) implantation of infection; (2) by blood metastasis; and (3) by local extension of adjacent infection.

Implantation abscesses result from penetrating wounds and have, as a rule, a low mortality, except in the case of wounds from sticks (p. 372). The infecting organisms are normally derived from the skin surface and are of low virulence. The entry track is closed at the dural level by protrusion of a hernia cerebri, which seals the subarachnoid space and provides a pathway for the extension of the infection to the surface away from the ventricles (fig. 440).

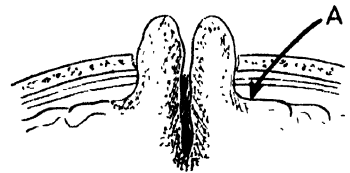


FIG. 440. — Protrusion of a hernia cerebri through a penetration of the dura, closing the subarachnoid space at A.

Clinical features may consist of pyrexia and the progressive extrusion of a hernia cerebri during the acute stage following a penetrated wound, but many abscesses pass insidiously into a chronic stage and merely produce a discharging sinus, or evidence of an intracranial space-occupying lesion at the site of a wound years after injury. Peter de Marchettis, a monk in Padua, treated such cases successfully by the insertion of lampwick drains in 1665.

Treatment.—In the acute stage the track is explored from the surface, bone and foreign material are removed, and pus is evacuated by repeated aspiration or tube drainage. Antibiotic treatment is given freely. Chronic abscesses may be excised intact.

Metastatic abscess is a complication of lung abscess or bronchiectasis or pyæmic states and was at one time uniformly fatal. Infected clot from the lung passes to the left heart, thence via the carotid, usually into the middle cerebral circulation, which forms a direct continuation of the carotid syphon; hence the infection is

implanted deep in the white matter of the parietal or temporal lobe close to the ventricle. The blood supply of the brain is poor in the white matter, but rich in the cortex; tissue reaction is therefore feeble, and the septic encephalitis spreads rapidly and produces fatal septic ventriculitis within a matter of days.

Clinical Features.—The patient with a causative disease is seized with sudden headache, collapse, and pyrexia, and rapidly develops well-marked localising signs. Hemiplegia, hemianæsthesia, or hemianopia indicate widespread destruction of the white matter.

Treatment.—Under modern conditions antibiotic treatment may cause the infection to localise. After a few days an exploratory burr hole and cerebral puncture is made to see if suppuration has occurred. If so, pus is aspirated and antibiotics are instilled. Further treatment is continued, as in the case of local extension abscesses. The average mortality is now approximately 30 per cent.

Local extension abscesses are produced by septic thrombosis spreading from foci of infection in the ear or frontal sinuses. Usually three out of five abscesses arising from the ear are situated in the corresponding temporal lobe; the remainder are in the cerebellum. Frontal lobe abscesses are usually on the same side as the infected frontal sinus.

Very rarely, aberrant abscesses may occur, for example, contralateral frontal abscesses produced by infection crossing the superior sinus, or frontal or parietal abscesses from ear disease. The site of implantation may be (a) subpial, or (b) subcortical.

Subpial Abscess.—The infection is conveyed as far as the cortex only. A minute abscess forms, which is covered on its surface only by the pia mater. This invariably ruptures into the subarachnoid space, producing meningitis without focal signs. It is probable that most cases of meningitis complicating ear or sinus disease are produced in this way; hence the need for prolonged antibiotic and intrathecal therapy to sterilise the subpial abscess and prevent relapse.

Subcortical Abscess.—The infection is conveyed across the unobliterated subarachnoid space to lodge in the white matter beneath the cortex. Three stages may be recognised—acute, subacute, and chronic.

In the acute stage there is septic encephalitis without pus formation. This may extend to produce ventriculitis or localise to form an abscess.

The subacute stage commences at three weeks by the formation of a glial wall, the thickness of which is determined by the local blood supply and is therefore thickest towards the cortex and thinnest towards the ventricle. A unilocular or multilocular cavity is produced, containing active organisms. The wall becomes thick within six weeks and infecting organisms die off, staphylococci living longest. The pus gradually becomes sterile and extremely thick, at which stage the abscess is inert.

Clinical Features.—*In the diagnosis of intracerebral abscess, general features are far more important than focal signs, which are few in number and of late onset.*

In the acute stage, persistent pyrexia and headache are the most important features and should lead to the suspicion of an abscess when seen in association with ear or sinus disease. The temperature and pulse-rate are raised at the commencement, but as the abscess enlarges, so intracranial pressure rises also, with slowing of the pulse. Leucocytosis is present, but its significance must be discounted if some other active condition is also present. Headache, irritability, drowsiness, and vomiting are commonly in evidence. These symptoms demand investigation by lumbar puncture, which will show an increase in cells and a protein figure raised from the normal 40 mg. to approximately 80 mg. per cent.

Acute fulminating syndromes are rarely seen under antibiotic treatment. Many cases are undoubtedly sterilised and the abscess aborted at the acute stage of septic encephalitis without pus formation, as the result of the administration of antibiotics in the treatment of the primary focus of infection from

which an abscess has extended. Focal signs are often absent in the acute stage except where there is extensive cortical thrombosis.

In the subacute stage the temperature and pulse often fall to a subnormal level and physical signs appear—these are few in number.

In frontal lobe abscess—a contralateral facial weakness only.

In temporal lobe abscess—a contralateral hemiparesis with absent abdominal reflexes and an extensor plantar response.

In cerebellar abscess—nystagmus, hypotonus, and inco-ordination on the side of the lesion.

Lumbar puncture shows a reduction in number of cells but increase in the protein figure to 120 mg. per cent.

In the chronic stage intermittent headaches and ill health persist with pallor and cachexia. There may be no physical signs. The cerebrospinal fluid may return to normal, but the abscess continues to enlarge and gradually uses up available adaptation within the skull and then, when no more space is available, suddenly produces urgent pressure symptoms. The patient may then develop severe headaches and exhibit evidence of a threatened mid-brain cone with a dilated pupil, within a period of days, perhaps several years after the initial infection.

Often the symptoms and physical signs of a chronic abscess develop immediately or within a day after operation has been performed on the ear or frontal sinus for the relief of a chronic headache—in these cases operation reveals an abscess with thick walls. Clearly a subacute or chronic abscess was present at the time that the operation was performed, and was itself the cause of the head pain for which the operation had been conducted. The local disturbance of the operation causes a reaction in the abscess cavity, and stimulates the early post-operative onset of physical signs. It is important to realise that the presence of well-marked localising signs developing within a period of days usually indicates re-activation of a chronic abscess requiring immediate treatment.

In every case of suspected cerebral abscess it is important to examine all possible sources of infection. In many cases the probable cause is evident, but discharge from an ear may cease when intracranial complications develop, for hindrance to discharge by inspissated pus predisposes to the extension of infection.

Treatment.—Drainage, formerly the recognised method of treatment of a cerebral abscess, has, in the majority of cases, been replaced by repeated aspiration of the abscess cavity and occasional excision. For example, an abscess which complicates otitis media is no longer treated by opening the middle ear and draining the cavity by that route. Neurosurgeons now regard the primary focus as of secondary importance, and means are taken to encourage the infection to localise. Localisation and subsequent enucleation diminish the risks of cerebral oedema and meningitis (Northfield). Drainage is only indicated if the abscess is superficial, as in the case of one which follows a penetrating wound.

In the early stages of cerebral inflammation antibiotics and chemotherapy are employed, and the infection may thus be overcome. Ventriculography may be required to determine whether an abscess is present or not after a course of antibiotics has been given (fig. 441). When an abscess has localised, or if signs are urgent, a diagnostic burr hole is made and the abscess is treated by aspiration and instillation of antibiotics.

Burr-hole openings are formed : (1) Immediately within the hair line, tapping forwards and inwards for a *frontal abscess*. (2) Immediately above the down-turned ear, tapping straight inwards for a *temporosphenoidal abscess*. (3) Over the occipital



FIG. 441.—Ventriculography. Showing crescentic deformity of the frontal horn produced by a 30-ml. abscess in a child whose only symptoms were headache, cachexia, and wasting following a compound frontal fracture. The cerebrospinal fluid was normal.

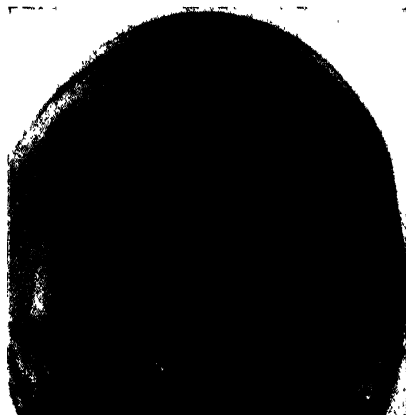


FIG. 442.—Skull X-ray in Towne's position showing thorotrast in a collapsed cerebellar abscess which is at the apex of the petrous bone.

plate, tapping forwards and inwards for a *cerebellar abscess* (fig. 442). Metastatic abscess may have to be located by two exploratory burr holes (fig. 443). The dura is opened and a blunt ventricular needle is inserted and the resistance of an abscess will

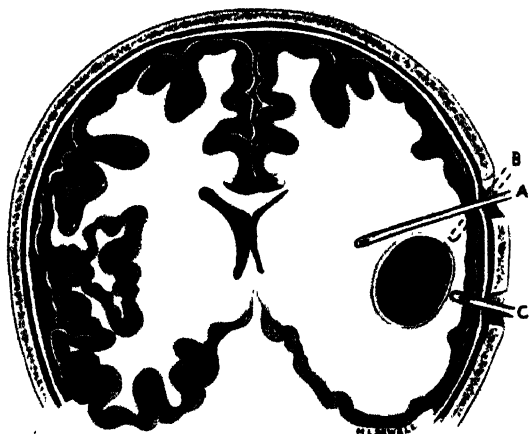


FIG. 443.—Method of localisation of metastatic abscess by burr-hole exploration. Needle at A encounters resistance at B. Puncture is performed by a second burr hole over the site of the abscess C.

cavity begins to diminish steadily in size and finally shrinks to the size of a pea (fig. 444).

Excision of the abscess is needed only for rigid abscesses which will not collapse, or for cerebellar abscesses which respond less satisfactorily to tapping and in which urgent symptoms may develop. If a cerebellar abscess does not respond to two aspirations, excision is indicated at an early stage.

Other abscesses should be enucleated within the third month, as the wall is by then sufficiently tough to separate from surrounding brain tissue. Too long a delay

be felt. Then the cannula is pushed through the wall into the cavity and pus will either well up or have to be aspirated slowly. Thirty ml. is a common volume to obtain. Two hundred thousand units of penicillin and 1 ml. of thorotrast are then injected into the cavity. Pus is examined, and if necessary a further puncture is performed the next day if other antibiotic treatment is required. A single injection of streptomycin may completely sterilise the at one time fatal *Esch. coli* abscesses. The thorotrast is taken up into the wall of the abscess and demarcates its outline. Repeated X-rays are taken at intervals. If evidence of a multilocular cavity is seen on X-ray, or if the cavity is extending in size or signs are increasing, further aspirations are performed. After several taps, the

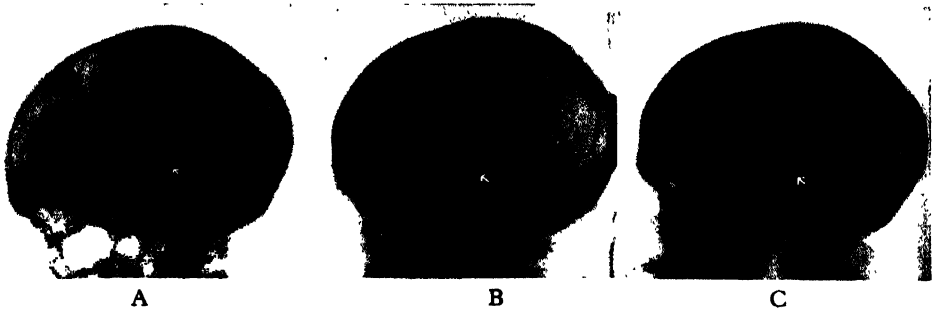


FIG. 444.—Progressive reduction in size of an abscess by repeated tapping.

results in excessive scar around the cavity, also formation of loculi is encouraged. Enucleation of a cerebellar abscess demands special care, as cranial nerves may be adherent to its wall.

INTRACRANIAL TUMOURS

Tumours arise in connection with the meninges, nerve sheath, or cerebral substance (gliomas). Tumours of the pituitary gland, vascular malformations, gummas, tuberculomas, blood-clots, and chronic abscess contribute to the total. Secondary carcinoma is far more common than primary intracranial tumour. Secondary deposits are most common from the lung, but may originate from any organ in the body and from the naso-pharynx. When these have been excluded, an average surgical series will be as follows :

- Meningioma, 18 per cent.
- Neurinoma, 8 per cent.
- Glioma, 43 per cent.
- Pituitary adenoma, 12 per cent.
- Craniopharyngioma, 5 per cent.
- Metastatic carcinoma, 6 per cent.
(not previously excluded on clinical grounds).
- Blood-vessel tumour, 2 per cent.



FIG. 445.—Characteristic globular meningioma.



FIG. 446.—Converging vascular markings overlying a meningioma.

Plus granulomas and unclassified and rare tumours.

Meningiomas, 18 per cent., vary in structure and vascularity and include psammomas (calcified meningioma, usually spinal), fibroblastomas, endotheliomas, and angioblastic meningioma. They are usually globular (fig. 445), but occasionally form a flat carpet work, meningioma *en plaque*, which spreads widely in the meninges. Arising from the arachnoid, the

tumour gains secondary attachment to the dura, the arteries and veins of which enlarge to provide a tumour circulation (fig. 446). Dilated emissaries between the bone and dura carry the venous return to the veins of the diploe and scalp, and along these veins tumour cells invade the bone, causing bone destruction and reactive hyperostosis (fig. 447).

Meningioma occurs in the following situations :

(1) **Parasagittal**—arising from the lateral lacunæ and pressing down on to the upper aspect of the frontal parietal and occipital lobes.

(2) **Fronto-basal**—occurring on the cribriform plate, outer, middle, and inner third of the sphenoid wing, and tuberculum sellæ, pressing on the olfactory, oculomotor and optic nerves as well as the brain.

(3) **Posterior fossa**—in the region of the cerebello-pontine angle and jugular foramen.

They may rarely arise from the falx or from the choroid plexus within the ventricle, or may develop from the depth of a sulcus expanding subcortically as intracerebral 'hæmangioblastoma'.

Neurinoma, 8 per cent., is usually found



FIG. 447.—A large meningioma hyperostosis extending from the right to left parietal bones. Successfully removed together with underlying tumour. This necessitated a nine-hour operation.

on the sheath of the auditory nerve (eighth nerve tumour), and may be multiple in association with von Recklinghausen's disease and sometimes occurs in association with multiple cerebral or spinal meningiomas.

Gliomas, 43 per cent.—The malignancy of the tumour varies in proportion to the degree of reversion to the primitive type of cell.

Astrocytoma in its most adult form is composed of star-shaped cells which resemble adult neuroglial tissue. It occurs in three forms: (1) The diffuse or infiltrating, which cannot be totally removed as its margins are unrecognizable and it often affects the brain stem. (2) The solid, which can be completely excised from suitable areas, if necessary by lobectomy. (3) The cystic, in which the nodule of tumour secretes fluid from its surface, forming a cystic cleft between itself and compressed normal brain. The tumour projects into the cyst cavity, and removal of this mural nodule produces a complete cure. Astrocytoma occurs in the frontal lobes of adults and in the cerebral hemispheres of young subjects. However, most astrocytomas contain a variety of cells, the proportion of which varies in different parts of the same tumour. Biopsy from a single favourable point may thus give a totally erroneous impression of a tumour's malignancy. In the modern classification of Kernohan, astrocytic gliomas are classified Grades 1, 2, 3, 4, according to the proportion of adult and primitive cells which they contain. Grade 1 corresponds to the pure astrocytoma. Grade 4 to glioblastoma multiforme (*Bailey and Cushing's* Classification). Malignant gliomas affect particularly the middle central territory in male subjects and arise as a result of malignant degeneration in an astrocytoma nodule.

Oligodendro-glioma.—An adult cell tumour consisting of cells with short stunted processes affects the deep portions of the hemispheres in adults. It is prone to undergo central cystic degeneration, and forms a ragged cyst the walls of which consist of tumour tissue. Treatment is usually palliative by evacuation of the cyst.

Friedrich Daniel von Recklinghausen, 1833–1910. Professor of Pathology, Strasbourg.

James Watson Kernohan, Contemporary. Emeritus Consultant in Pathology, Mayo Clinic, Rochester, Minnesota, U.S.A.

Percival Bailey, Contemporary. Distinguished Professor of Neurology, Chicago Medical School, Chicago, Ill., U.S.A.

Harvey Cushing, 1869–1939. Professor of Surgery at Johns Hopkins and Harvard University.

Spongioblastoma polare arises from primitive uni- or bi-polar spongioblasts and affects inaccessible regions, such as the optic chiasma, third ventricle, and hypothalamus in young subjects. It is irremovable, somewhat radio-sensitive, and rarely produces seedling metastases in the cerebrospinal fluid.

Medulloblastoma occurs usually in young children, affecting the vermis of the cerebellum. It grows rapidly and produces seedling metastases throughout the meninges. Microscopically, it resembles a small round-celled sarcoma, and it is highly radio-sensitive.

Clinical Features.—All tumours have an initial silent period which varies in length according to position and rate of growth. If the tumour is not near any area which will produce symptoms or signs, it will take up space provided in the subarachnoid cisterns; it will flatten and displace the

CLINICAL COURSE OF CEREBRAL TUMOUR

Stage 1	Stage 2	Stage 3	Stage 4
Initial period of silent growth.	Focal syndromes. Epilepsy.	Raised intra-cranial pressure.	Brain displacement. False localising signs. Cone formation.

ventricle and brain until it can gain no more room, and it will then produce symptoms of raised intracranial pressure, such as morning headache, effortless vomiting, and papilloedema. It may be several years or more before this occurs in the case of a meningioma. If the tumour is situated in an important area, it may, by its local effect, produce symptoms of epilepsy or progressive neurological syndromes before any evidence of intracranial pressure is produced. Hence the absence of headache, vomiting, and papilloedema does not exclude a tumour.

Epilepsy arising for the first time in adult life should always be suspected as being due to a tumour until this possibility has been disproved (fig. 448).

Idiopathic epilepsy does not occur before the age of six. Ninety per cent. of cases of idiopathic epilepsy have their first seizure before the age of thirty. After the age of thirty, epilepsy is usually symptomatic, and in patients between the ages of thirty and fifty tumour is a common cause.

Progressive focal syndromes should likewise be regarded as indicating a tumour until this possibility is

disproved. Degenerative conditions such as disseminated sclerosis are characterised by periods of remission and exacerbation. Vascular lesions

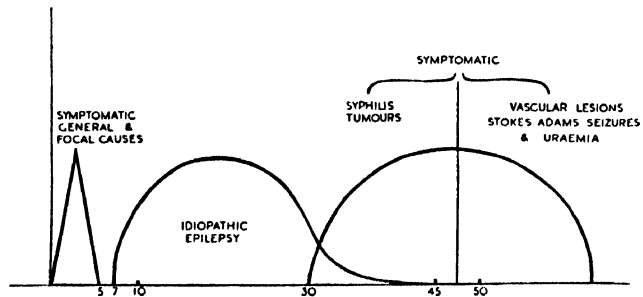


FIG. 448.—The principal causes of epilepsy at different ages.

occur instantaneously and are followed by some degree of improvement. If repeated on several occasions, as in multiple emboli, an appearance of steady progression is produced, but there is only one condition that will produce a steadily progressive syndrome, and that is a tumour.

The stage of raised intracranial pressure can develop in association with focal symptoms or be the sole evidence of tumour formation. Pressure symptoms occur late in the case of tumours in the frontal lobes which push the ventricles back. They occur earlier in tumours in the temporal and

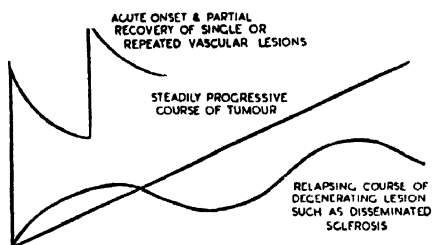


FIG. 449.—Clinical courses of vascular, neoplastic and degenerative lesions.

parietal lobes which partially obstruct the outflow of fluid from the adjacent ventricle. They occur earliest in midline and posterior fossa tumours which obstruct the flow of fluid from both ventricles, producing an internal hydrocephalus. Tumours of these types add to their own bulk the effect of the retained ventricular fluid. The effects of pressure include headache,

vomiting and papilloedema, bradycardia, and retarded-cerebration. The headache often wakes the patient in the early morning, and is aggravated by coughing and straining, which promote cerebral congestion. Vomiting occurs without warning, and is not preceded by nausea or related to food. Papilloedema may cause blindness.

The Stage of Cone Formation.—Certain patients do not present until cone formation is imminent. When intracranial pressure becomes high, the inner border of one hemisphere may be forced under the falx plugging the superior longitudinal fissure and blocking the pathway for the cerebrospinal fluid absorption. The temporal lobe may be forced down from above into the tentorial opening. The cerebellar vermis may be pushed up into this opening or the cerebellar tonsils may be forced down into the foramen magnum. Ominous signs of a threatened cone include violent paroxysmal headache, drowsiness, slow cerebration, slow pulse, and neck stiffness. Unilateral pupillary dilatation is an urgent sign. *Lumbar puncture must be rigidly avoided at this stage.*

Frontal lobe tumours if deeply situated produce a progressive change in personality, with lack of insight, neglect of normal pastimes, occupations, and duties, and an alteration in emotional reaction particularly noticeable to relatives, consisting usually of a simplicity and unwarrantable cheerfulness (euphoria) or irritability. Epilepsy is generalised in type, and localising signs are limited to contralateral facial weakness.

Parietal lobe tumours produce Jacksonian epilepsy and progressive hemiparesis. Examination reveals loss of touch analysis resulting in astereognosis. Deeply situated left-sided tumours may exhibit defects of special relationship and loss of power of calculation (acalculia).

Occipital lobe tumours.—Epilepsy is generalised in type, preceded by an aura of flashing lights in the contralateral visual field. Signs, other than homonymous hemianopia, are few.

Temporal lobe tumours on the left side may produce progressive aphasia or

sual or auditory hallucination sometimes with generalised convulsions. Lesions of the uncinate region produce uncinate hallucination of smell and taste and dreamy states of unreality. Right-sided lesions may cause similar uncinate attacks of generalised epilepsy without aura. Localising signs include hemiparesis and superior quadrantic hemianopia.

Parasagittal meningioma overlying the various lobes produces similar symptoms but with characteristic skull X-ray changes (see fig. 446, 447).

Basal meningioma produces involvement of the olfactory and optic, oculomotor, and trigeminal nerves, in addition to pressure on the brain. Bone changes in the region of the orbit may cause proptosis or fullness in the temporal fossa.

Midline tumours always produce bilateral internal hydrocephalus with pressure symptoms.

Tumours of the third ventricle.—Colloid cysts at first cause a ball-valve obstruction producing intermittent blockage of the foramen of Monro. Severe unilateral headache is produced by lying on one side of the head and relieved by turning to the other side. Later, persistent bilateral hydrocephalus is accompanied by severe pressure symptoms or progressive cerebral atrophy and dementia. Glioma of the floor of the ventricle causes hydrocephalus associated with endocrine disturbances, disturbances of sleep rhythm, and sexual precocity.

Pineal tumours by pressure on the quadrigeminal plate produce hydrocephalus with paralysis of the oculomotor nuclei with loss of upward, lateral, and downward movement of the eye.

Subtentorial Tumours.—Tumours of the posterior fossa produce hydrocephalus and cause raised intracranial pressure. The effect of the hydrocephalus varies with the age of the patient.

Cerebellar vermis tumours are usually medulloblastomas and occur in young children before the sutures have united. The hydrocephalus causes progressive enlargement of the head. At a later age, when the sutures have united, there are serious pressure symptoms and stiffness of the neck from herniation of both cerebellar tonsils into the foramen magnum. Since the vermis controls the co-ordination of trunk and legs, localising signs are only seen if the child is taken out of bed; it will then be seen to walk with the feet wide apart and have a tendency to pitch over forwards when standing with the feet together.

Cerebellar hemisphere tumours are often astrocytomas. They produce hydrocephalus and pressure symptoms in older subjects. Since the hemisphere controls the co-ordination of the corresponding side of the body, there is deviation to the affected side on walking and unilateral inco-ordination of the arm. Nystagmus may or may not be present.

Acoustic neuroma grows from the auditory nerve at the internal auditory meatus and produce enlargement of this structure, visible on X-ray. Arising from the eighth nerve the first symptom is unilateral deafness, which is often first detected by the patient on the telephone. The tumour projects into the cerebello-pontine angle and presses upon the adjacent seventh, sixth, and fifth nerves, causing a syndrome of unilateral deafness, facial weakness, and sometimes squint. The corneal reflex is reduced from pressure on the fifth nerve. There may be trigeminal neuralgia from this cause or trigeminal anaesthesia. Later the tumour presses upon the cerebellum and brain stem, producing cerebellar signs and raised pressure. Finally it grows up into the tentorial opening from below, and here it not only blocks the up-flow of cerebrospinal fluid but assumes a position anterior to the brain stem coming into close relationship with the basilar artery and twisting the pons. Cerebrospinal fluid protein is always high, often over 200 mg. per cent.

The investigation of cerebral tumour must define the site and nature of the tumour.

History taking.—The history alone will sometimes indicate the site of the tumour and also give a hint as to its pathological type. If symptoms have been long-standing, they suggest a slowly growing and favourable tumour.

A short history may, however, be due to the final breakdown of adaptation in a slowly growing tumour or indicate a rapid malignant growth.

The history may suggest a primary disease to which the cerebral condition is secondary. Since metastatic carcinoma, especially bronchial, is far more common than primary cerebral tumour, particular attention must be paid to history of loss of weight, recent cough, or hæmoptysis. Secondary brain abscess is suggested by a history of lung abscess or bronchiectasis; chronic otitic abscess by symptoms of cachexia and a discharging ear. Weight loss is always suspicious, as there is no wasting with primary cerebral tumour.

Clinical examination must include the general examination in search of primary disease.

Neurological examination of cranial nerves and nerve tracts may localise where a tumour is, but never indicates what it is.

Accessory investigations are essential. X-ray of the skull, X-ray of the chest, and the erythrocyte sedimentation rate must be taken in every case. A high E.S.R. is strongly suggestive of secondary tumour.

An X-ray of the chest may reveal an unsuspected bronchial carcinoma. Thirty per cent. of bronchial carcinomas present with cerebral symptoms before any chest symptoms have occurred.

An X-ray of the skull may show that a tumour is present:

(1) By pressure changes including: (a) A beaten silver appearance of the vault from the pressure of tight convolutions (sometimes normal in thin skulls). (b) Separation of sutures in young subjects. (c) *Most important*—erosion of the posterior clinoid processes, a very valuable sign which indicates long-standing pressure and therefore a possibly removable tumour (fig. 450).



FIG. 450.—Separation of sutures, beaten silver appearance, and erosion of posterior clinoids.

(2) By lateral displacement of a calcified pineal shadow indicating the side of the tumour.

(3) By characteristic intracranial calcification produced by astrocytomas, angiomas, 50 per cent. of craniopharyngiomas, and some meningiomas and tuberculomas (fig. 451).

(4) By alteration in skull vascular markings in meningioma, including converging diploic channels, and an increase in the size of the meningeal groove.

(5) By changes in the skull bones, including meningioma hyperostosis, local expansion at the site of a cyst, and evidence of bone destruction in secondary tumours.

Electroencephalogram.—Characteristic wave-forms indicate the site or presence of focal or deep-seated tumours, and distinguish between epileptic seizures produced by focal lesions and idiopathic epilepsy.

Lumbar puncture is employed in early cases where it is necessary to exclude non-tumorous conditions. The pressure and content of the cerebrospinal fluid are

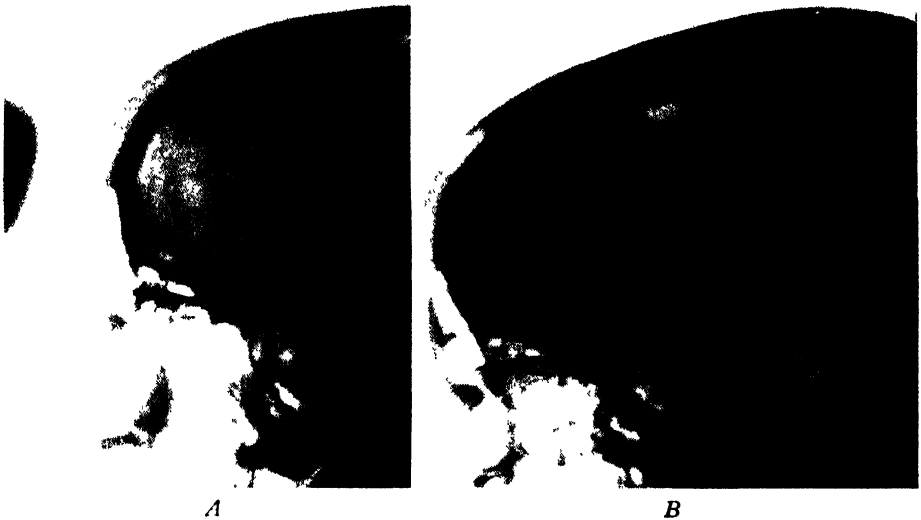


FIG. 451.

Calcified astrocytoma in frontal lobe.

After removal by lobectomy.

recorded. An increase in pressure and protein suggests tumour. The W.R. is examined. Lumbar puncture is avoided in the presence of raised pressure and is absolutely contraindicated by threatened cone formation.

Surgical investigation by arteriography or air study is employed to localise a silent tumour producing pressure but no physical signs, or indicate the extent of a known tumour, and to provide evidence of its type.

Arteriography was originated by Egas Moniz. Twelve ml. of 35 per cent. pyelosit is injected into the common carotid artery either by percutaneous puncture or by open operation. Three lateral films are taken at intervals of one second to show the arteries and then the veins, and this is followed by a further injection to obtain antero-posterior films. This investigation is safe even in high-pressure cases, as it does not disturb the intracranial pressures. The site of the tumour is shown by the displacement of cerebral vessels, e.g. upward or downward deviation of the middle cerebral or displacement of the anterior and middle cerebrals in the antero-posterior view. Pathological tumour circulation is seen in meningioma and glioblastoma multiforme. Meningiomas produce a diffuse blush; glioblastomas produce a group of very primitive and imperfect vessels (fig. 452).

Investigation by Air Study.—Ventriculography was originated by Walter Dandy. Bilateral parietal burr holes are formed under local anaesthesia. A hollow ventricular canula is inserted to a depth of up to 5 cm. to puncture the vestibule of the ventricle (fig. 453). Available cerebrospinal fluid is removed and replaced with 10 per cent. less volume of air. Since air rises and fluid falls the patient is positioned in such a way that the portion of the ventricular system which it is wished to fill with air is brought to the highest level during arteriography, films being taken in various planes to show displacement and distortion of the ventricle. Investigation is dangerous as tapping of a displaced and expanded ventricle on the side opposite to the tumour may allow the brain to be pushed over causing cone formation as the temporal lobe sinks into the tentorial opening. Arteriography is therefore preferable.

Egas Moniz, 1874–1955. Professor of Neurosurgery, Lisbon. Introduced cerebral arteriography and leucotomy.
Walter E. Dandy, 1886–1946. Professor of Neurosurgery, Johns Hopkins University, U.S.A. Introduced ventriculography in 1918.

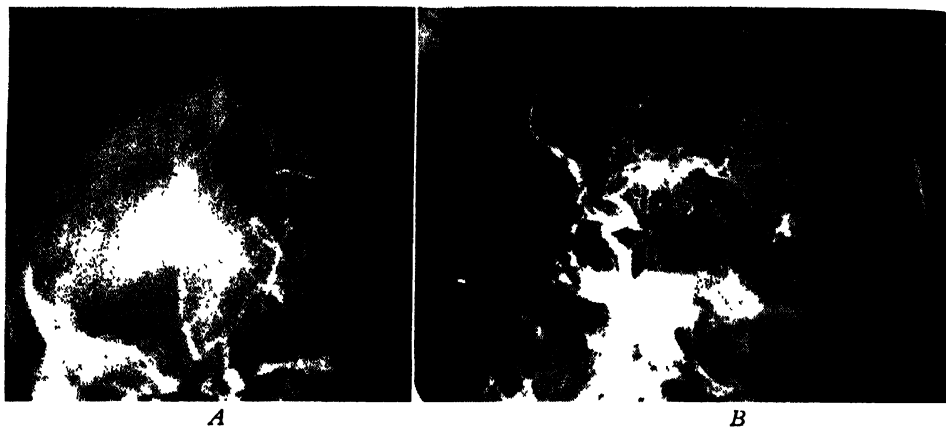


FIG. 452.

(A) A.P. arteriogram showing inward displacement of the anterior and middle cerebral arteries and tumour circulation causing a blush in a meningioma at the outer third of the sphenoid ridge. (B) Lateral arteriogram showing primitive glioblastoma vessels in posterior temporal region.

Myodil ventriculogram in hydrocephalus will demonstrate the third and fourth ventricles and aqueduct after the injection of 3 ml. of Myodil.

Air Encephalography.—Thirty ml. of cerebrospinal fluid is aspirated slowly and replaced by air at cisternal or lumbar puncture. This method is highly dangerous in cerebral tumours and should be reserved for investigation of low-

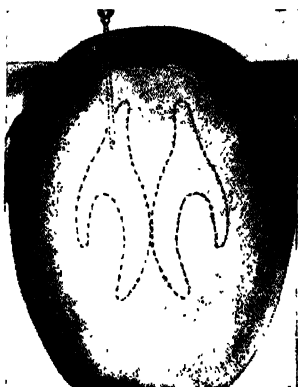


FIG. 453.—Diagram of ventriculography.



FIG. 454. — X-ray indicating dangers of ventriculography. Both ventricles are already on one side of the head. This is not unusual.

pressure cases with symptoms of epilepsy.

Investigation by Cerebral Puncture.—If investigation suggests the presence of an unfavourable lesion in which craniotomy is contraindicated, such as secondary carcinoma or malignant glioma, or if a cyst is suspected, a burr hole is fashioned over the tumour site and the tumour is aspirated by ventricular cannula. If a cyst is defined, air or thorotrast is injected into the cavity in order to produce a picture of the cyst (astrocytic growth).

A favourable cyst produced by astrocytoma or hæmangioblastoma contains golden-yellow clotting fluid.

A malignant cyst contains muddy non-clotting fluid like stale beer.

Astrocytic glioma Grade 3 and 4 renders the brain soft, necrotic, and highly vascular, whereas meningiomas are resistant.

If stained smears show the presence of astrocytic glioma Grade 3, lobectomy can provide a useful decompression prior to X-ray therapy in favourable situations. Open decompression of the dura however is contraindicated as the tumour contains primitive vessels and will enlarge owing to acute edema if the pressure of the dura is relaxed. This 'acute brain swelling' will cause additional symptoms of aphasia and hemiplegia. It may be possible to secure internal decompression by sucking out the necrotic centre of a tumour through a small dural opening prior to deep X-rays. Astrocytic glioma Grade 3 can then be usefully palliated by internal decompression or lobectomy followed by radiotherapy, but any operation on astrocytic glioma Grade 4 is definitely contraindicated.

Treatment.—Temporary dehydration by urea or intravenous 50 per cent. sucrose (see p. 360) may be employed as a pre-operative measure if intracranial pressure is acute.

Operation must not be delayed if pressure is high as sudden and unexpected collapse may occur. Improvement in neurosurgical technique permits a radical excision in many cases.

Craniotomy.—An osteoplastic flap is fashioned over the site of the tumour and must be sufficiently large to expose either the lobe to be excised or an adequate margin of normal brain around the tumour which is to be enucleated. In the case of meningiomas a length of the superior longitudinal sinus greater than the known length of the tumour must be displayed. Various scalp incisions are employed, transverse or horseshoe, all of which are hidden as far as possible within the hair line.

The field of operation is infiltrated with 1 : 100,000 of adrenalin in saline. The skin incision is outlined, the area of operation is sealed off by lint trephine cloth, which is gummed to the scalp with gum mastiche and cut through in the line of the proposed incision. The scalp is now incised. The assistant and surgeon exert digital pressure with the fingers on either side of the cut. Before pressure is released, artery forceps are applied to the epicranial aponeurosis on either side; these are folded back so that the vessels in the scalp are bent on themselves, and each bunch of four or five forceps is secured with a rubber ring. By degrees the incision is enlarged and an extensive exposure can be obtained, almost bloodlessly, using a sufficient number of forceps. The scalp flap is now reflected and wrapped in a warm swab. Skin towels are applied to the wound edge. Burr-hole openings are then fashioned in a suitable pattern, being placed at the summit of each convexity in order to reduce pressure on the dura when the saw guides are passed from hole to hole. Gigli guides and saws are now passed between the burr holes, and the intervening areas of bone are sawn through, the cut being fashioned obliquely, so as to form a mortise edge in the bone in order to prevent the bone flap sinking in when it is replaced. The base of the flap is designed to hinge on the temporal muscle. The bone flap is now elevated and the base fractures, the osteoplastic flap then being turned down on the hinge of temporal muscle and wrapped in a warm cloth. If a meningioma is being dealt with, the bone flap is extremely vascular, and on lifting the flap, hæmorrhage occurs from the torn emissary veins crossing between the bone and dura. The exposed dura mater is now inspected for an excess of vessels and torn emissary veins, indicating an underlying meningioma, or for surface evidence of underlying glioma, such as discoloration of the brain or widening of convolutions visible through the dura.

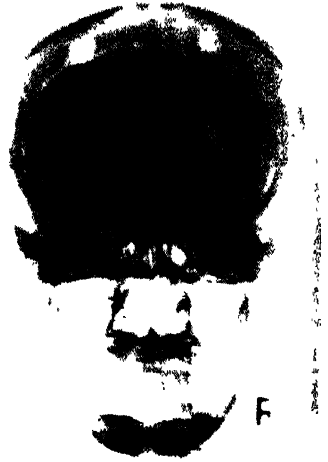


FIG. 455. — Antero-posterior ventriculogram showing flattening and downward displacement of left lateral ventricle by large parasagittal meningioma. The middle meningeal groove is enlarged on the left side.

To delineate the extent of a subcortical tumour, ventricular needles are inserted at three or four points in order to detect the resistance of a tumour margin. When the position and nature of the growth have been decided, the dura is opened by a horseshoe flap, either with guarded scissors or a knife cutting on to a dural guide. Meningeal vessels of large size are secured with Cushing's silver clips.

Excision of a Meningioma.—Parasagittal.—The dural incision is completed all round the base of the tumour unless this invades the superior longitudinal sinus. The margin between the brain and tumour is defined, and vessels crossing this are secured with diathermy or silver clips; the tumour is then rotated upwards from its bed and dissected from the sinus.



A



B

FIG. 456.

Cyst in occipital lobe defined by thorotrast.
Tumour produces defect at A.

Operative sketch of removal
of tumour.

Basal Meningioma.—Occasionally it is necessary to amputate the frontal lobe of the brain to expose tumours on the middle or inner third of the sphenoid ridge,

otherwise the lobe of the brain is retracted and the bulk of the tumour is reduced by cutting out its centre with punch forceps and diathermy, following which the capsule is gently drawn away from important branches of the circle of Willis which surround it. Finally, the collapsed capsule is excised intact, together with the adjacent dura (fig. 457).

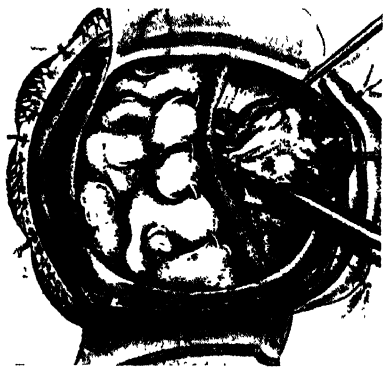


FIG. 457.—Excision of collapsed capsule of a basal meningioma exposed by frontal lobectomy.

Excision of a Glioma.—(a) *By Enucleation.*—The tumour nodule is exposed either by opening a cyst or by 'uncapping' the area of overlying cortex. A medallion of cortex of adequate area is defined. Vessels entering this zone are secured with diathermy or silver clips, the defined area being then removed down to the level of the tumour margin. Vessels crossing from brain to tumour are gradually secured and the nodule is rotated from its bed (fig. 458).

(b) *By Lobectomy.*—Veins passing from the frontal or occipital lobe to the superior sinus are coagulated on a blunt hook, or secured with silver clips, and divided. Veins passing from the temporal lobe to the sphenoidal or petrosal sinuses are likewise secured. The plane of section is now defined, and vessels crossing this are secured with silver clips or diathermy. The incision is now deepened across the white matter to the opposite aspects of the lobe, where vessels are similarly dealt with and the lobe is excised.

If a tumour is irremovable, central necrotic areas are removed by suction in order to reduce pressure. In all cases hæmostasis must be complete before the

ound is closed. The dura is sutured completely, and if necessary with the introduction of a graft of nylon membrane. If bone has been invaded by meningioma hyperostosis, it is excised. Finally, the scalp is sutured in two layers. Blood transfusion is required during operation in vascular cases.

POSTERIOR FOSSA CRANIECTOMY

Intratracheal anaesthesia is essential. The head is fully flexed and supported with the patient in the sitting-up position, which reduces venous congestion. A fine catheter is inserted into the lateral ventricle through a posterior parietal burr hole to provide ventricular drainage and reduce intracranial pressure. A midline incision is formed from above the external occipital protuberances to the level of the third cervical spinous process. The lateral skin flaps are raised from the occipital muscles, retractors are inserted and the ligamentum nuchæ is divided with diathermy to expose the arch of the first cervical vertebra. The occipital muscles are cut through $\frac{1}{4}$ inch (6 mm.) below their attachment and dissected with diathermy from both occipital plates. The arch of the atlas is removed. Both occipital plates are drilled through and then removed with biting forceps including the rim of the foramen magnum. This exposes the posterior fossa dura as high as the transverse sinus, the foramen magnum, and upper cervical dura. The dura is palpated, and if the pressure is high, it is reduced by releasing fluid from the ventricles or tapping the cisterna magna. The dura is incised transversely, the occipital sinus being secured with Cushing's silver clips, and a vertical incision is now made downwards in the midline to the upper cervical region to release the pressure cone at the foramen magnum. If



FIG. 459.—Removal of astrocytoma nodule of cerebellum.

removed. The cisterna magna is tapped to reduce pressure and the dura is opened.

Eighth nerve tumours are exposed by this route. The lower third of the cerebellar hemisphere is cut away and the tumour exposed anterior to the cerebellum. The capsule is incised, its contents are evacuated, and the capsule is gently drawn away from the side of the brain stem and downwards through the tentorial opening before



FIG. 458.—Excision of astrocytoma from cyst in parietal lobe showing (a) the dark mural nodule projecting into (b), the white-walled cyst whose walls consist of compressed cerebral tissue.

both tonsils are forced down, the tumour will be found in the midline. If one tonsil is down, the tumour is in the corresponding hemisphere. Astrocytoma nodules are excised, but medulloblastomas are best left intact with an open decompression for deep X-ray treatment.

Unilateral exposure may be performed through a hook incision extending up from the third cervical spine to the external protuberance and then laterally and finally downwards to the mastoid tip. The ligamentum nuchæ is opened to expose the arch of the first cervical vertebra, and the occipital muscle is reflected downwards from the occipital plate, which is then

being excised. A small portion is left over the facial nerve in order to prevent facial paralysis.

Large tumours are dangerous as they extend into the tentorial opening anterior to the brain stem, distorting and compressing the basilar branches. A fatal brain stem thrombosis may occur as a post operative complication.

Radiotherapy should be used only for tumours showing mitotic activity: medulloblastoma, astrocytic glioma Grade 3, and meningiomas exhibiting mitotic activity which may be liable to recur locally. It should not be employed in insensitive tumours as it produces endarteritis in the cerebral vessels, sometimes with serious symptoms.

TUMOURS OF THE PITUITARY BODY

comprise three varieties of adenoma; chromophobe, acidophil, and basophil, and congenital tumours derived from Rathke's pouch (craniopharyngiomas).

Chromophobe adenomas occur most commonly in female patients between the ages of twenty and fifty. The tumour is solid and slow-growing and expands steadily to form a mass the size of a walnut in a period of several years. Spontaneous involution may occur with cessation of growth and cystic change (cf. adenoma of thyroid). Occasionally highly cellular rapidly growing tumours assume the characteristics of local malignancy invading laterally into the cavernous sinus or extending into the lobes of the brain. Three stages of development may be recognised:

- (a) The stage of intrasellar development.
- (b) The stage of extrasellar extension.
- (c) The stage of massive intracranial extension.

(a) *Intrasellar Development*.—In the sella turcica, the expanding chromophobe cells, which possess no active secretion of their own, compress the acidophil and basophil cells of the pars anterior and inhibit their secretions

which are concerned with growth and sex functions. Since the tumour arises at an age when growth is complete, the effect is felt by the thyrotropic and gonadotropic hormones. The patient becomes fat and sluggish with a lowered metabolic rate, as in myxœdema, and amenorrhœa is invariable. Pressure on the diaphragma sellæ causes severe headache. X-ray of the skull reveals pressure changes of



FIG. 460.—Typical X-ray appearances of enlarged sella in chromophobe adenoma.

the sella (fig. 460). The floor is depressed towards the sphenoidal air sinus, lateral pressure on the petroclinoid ligaments leads to separation of the anterior clinoid processes, and the posterior clinoids become thin.

(b) *The Stage of Extrasellar Extension*.—The tumour breaks through the diaphragm at a weak point between the anterior clinoid processes and extends up in front of the pituitary stalk to press on the under aspect of the optic chiasma. Headache becomes less severe from relief of intrasellar pressure,

amenorrhœa persists, and gradually bitemporal hemianopia is produced by stretching of the decussating fibres in the optic chiasma over the posterior border of the tumour. Involvement of these fibres which come from the inner half of each retina, leads to loss of vision towards each temporal side. Stretching of the optic nerves anterior to the chiasma produces primary optic atrophy with pale white optic discs. The diagnosis is usually made at this stage.

Acute infarction may rarely produce a sudden swelling of the tumour with rapidly increasing visual loss. The risk of infarction occurring after deep X-ray therapy or the implantation of radioactive Yttrium contraindicates the use of either method when the chiasm is compressed. Infarction may cause complete blindness and then be followed by atrophy and disappearance of the tumour.

(c) *The stage of massive intracranial extension is rare.* If the optic chiasma is in an abnormal position, either pre- or post-fixed, the tumour may slip by it without causing visual disturbance and enter the frontal lobe, or highly cellular rapidly growing tumours may spread laterally into the temporal lobe or upwards and backwards behind the chiasma into the third ventricle. These extensions will act as a space-occupying tumour and produce symptoms of raised pressure or epilepsy. Extension into the temporal lobe produces an additional homonymous hemianopia from involvement of the optic radiation.

Differential Diagnosis.—The syndrome may be simulated by a meningioma of the dorsum sellæ or an aneurysm of the anterior communicating artery. Arteriography is required in doubtful cases.

Treatment.—Few chromophobe tumours are radio-sensitive. Many alleged successes attributed to deep X-ray have been due to spontaneous cystic involution or an abnormality of the chiasma which allows the tumour to go on growing without serious visual loss developing. Years later the growth is found to have formed a massive intracranial extension.

Operation performed before the stage of massive extension has a mortality of 2 to 4 per cent.; after extension 30 per cent., hence early operation is advisable. Pre- and post-operative treatment by steroids is advisable in the presence of hypopituitarism in order to prevent post-operative adrenal deficiency.

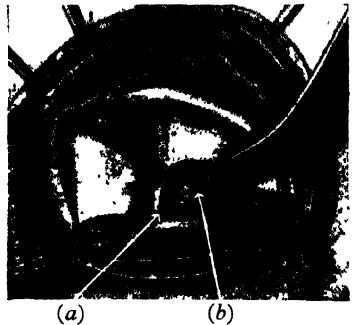


FIG. 461.—Intradural exposure of pituitary tumour, showing (a) optic nerve and (b) pituitary tumour.

In the stage of intrasellar development, if there is no evidence of involvement of the chiasma, radioactive seeds of Yttrium ($Y. 90$) may be inserted into the tumour by stereotactic approach from above, or implanted across the ethmoids through a para-nasal incision under X-ray control. In the stage of extrasellar extension, the chiasma must be decompressed. The size of the tumour is first assessed by arteriography and air encephalography which will show the presence of any upward extension either by the stretching of the carotid arteries or indentation of the front of the third ventricle.

The tumour is exposed by frontal osteoplastic craniotomy, after opening the dura and elevating the frontal lobe. The extradural approach dividing the dura at the sphenoid wing gives less adequate exposure. The tumour looks like a dark plum

stretching the optic nerve like a white ribbon around its margin. Cysts are opened or solid tumour is removed by punch forceps. The capsule of the tumour is then drawn down from behind the chiasma and excised. This step is essential to prevent post-operative hæmorrhage into a nodule lying beneath the third ventricle which stimulates the temperature centres and causes death from hyperthermia. Relief of pressure on the basophil cells restores sex function, and pregnancy may take place after operation. Post-operative deep X-ray is required to prevent recurrence in highly cellular growths.

Acidophil adenomas are small in size and rarely cause pressure on the optic chiasma except in cases of fugitive acromegaly, in which the tumour reverts almost to a chromophobe type. Normally the symptoms are due to excessive production of growth hormone by the acidophil cells and inhibition of the basophil sex secretion.

In children the tumour causes gigantism, while patients whose epiphyses have united develop acromegaly (a term which implies enlargement of the extremities).

Acromegaly is characterised by thickening of the subcutaneous tissues of the scalp, lips, and tongue, the face, hands and feet, and overgrowth of the frontal sinuses, jaw, and distal phalanges (p. 253). There is also overgrowth of hair and sebaceous glands. Asthenia causes slackening of ligaments with kyphosis, so that the enlarged hands hang below the knees.



FIG. 462.—Acromegaly.

This, combined with the atavistic appearance produced by the beetling brow, prognathous jaw, and overgrowth of hair on the chest, produces the 'ape man' of the circus. Fatty degeneration of the heart and herniæ are associated conditions.

Treatment is by deep X-ray, to which the tumour is sensitive, or intrasellar implantation of radioactive Yttrium. Operation

is only rarely required, which is fortunate, since the patients are poor operative risks on account of the heart and chest condition, and approach is difficult, owing to enlargement of the frontal sinuses, which may necessitate a lateral or trans-sphenoidal operation.

BASOPHIL ADENOMAS AND CUSHING'S SYNDROME

Basophil adenomas are small, usually only a few millimetres across; their effects are produced by secretion of adreno-corticotrophic hormones leading to excessive production of hydrocortisone.

Cushing described a syndrome associated with basophil adenomas, although a similar condition may be due to adrenal dysfunction (adreno-genital syndrome, p. 580). Females are most commonly affected. Fat accumulates on the trunk, neck, and face which becomes red and moonshaped but the limbs remain normal. Purple striae appear in considerable numbers on the skin, which is thin and atrophic. Hirsutism, arterial hypertension and glycosuria develop early. There is moderate polycythæmia and atrophic changes affect the bones of the os calcis and pubis. A psychotic state of schizophrenia sometimes develops.

Treatment is directed to reduction of the secretion of hydrocortizone either by adrenalectomy or by operation on the pituitary. Russell Fraser has shown that these tumours may be effectively treated by the intrasellar implantation of radioactive Yttrium 90.

Craniopharyngioma (*syn.* Rathke's Pouch Tumour).—Remnants of Rathke's pouch may persist at four sites :

(1) In the sphenoid bone ; (2) in the sella turcica ; (3) in the pars nervosa ; (4) in the floor of the third ventricle, from all of which tumours may develop. Although congenital in origin, a proportion of cases do not produce symptoms until late in adult life.

The term craniopharyngioma describes these tumours best. In structure, the growths form large masses in which cystic cavities lined with ciliated epithelium and containing cholesterol crystals are separated by areas of connective tissue. More than 50 per cent. of these growths are calcified and may form coral-like masses filling the interpeduncular space and extending backwards to the pons. Since they are adherent to the basal arteries and adjacent nerves, they are irremovable. Occasionally single cysts are found in or above the sella, but the term suprasellar cyst describes only this one variety and is deceptively encouraging, as it suggests an invariably removable lesion. In fact, these tumours are by no means always suprasellar, nor are they necessarily cystic.

Clinical Features.—The symptoms depend upon the site of the tumour and the age of the patient.

(1) Intrasellar craniopharyngioma cysts produce symptoms like the inert chromophobe tumour, but in young subjects they inhibit growth by compression of the acidophil cells as well as inhibiting sexual maturation. The result is a fat, impotent dwarf, who may subsequently develop bitemporal hemianopia from upward pressure on the chiasma (Fröhlich's syndrome). Intrasellar calcification is often visible in X-ray.

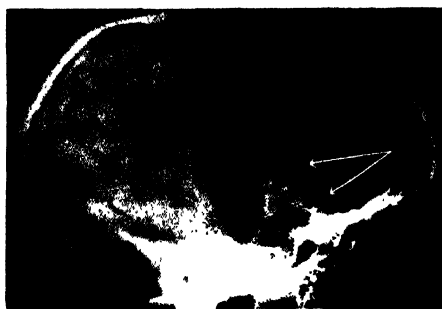


FIG. 464.—Arrows indicate suprasellar calcification in craniopharyngioma.



FIG. 463.—A large craniopharyngioma.

(2) Suprasellar craniopharyngiomata in young subjects, by downward pressure on the sella, also produce a Fröhlich syndrome, but in this case involvement of the optic tract behind the chiasma causes homonymous hemianopia (fig. 464).

Pressure on adjacent centres of the hypothalamus, which control sleep and water metabolism, produce pathological somnolence and diabetes insipidus, so that the subject is liable to fall asleep during the day, and to drink and pass abnormal quantities of fluid. Similar symptoms may occur at a later age in adults.

Intraventricular tumours may produce blockage of the third ventricle with symptoms of acute intracranial pressure, headache, vomiting, and papilloedema, without localising signs ; or an organic confusional state and dementia from cerebral atrophy following long-standing pressure.

Thomas Russell Cumming Fraser, *Contemporary*. Professor of Clinical Endocrinology, Royal Postgraduate Medical School of London.
 Alfred Fröhlich, 1871–1953, formerly Professor of Pharmacology, University of Vienna, described his syndrome in 1901.

Treatment.—Cystic tumours in and above the sella may be evacuated by frontal craniotomy. When large masses block the third ventricle, the cerebrospinal fluid may be short-circuited to the cisterna magna by leading a catheter from the ventricle through a burr opening in the skull, thence under the skin and occipital muscles to drain into the cisterna magna, which has been exposed above the foramen magnum (Torkildsen's operation)—ventriculo-cisternostomy.

Simmonds' disease, 'pituitary cachexia', results from depression of pituitary secretions affecting the gonads, adrenals, and thyroid gland following destruction of the anterior lobe of the pituitary by local thrombosis, hæmorrhage, cyst distension, post-partum necrosis, head injury, abscess, or tumour. The condition has also been caused by swabbing out the pituitary fossa with sclerosing fluid following local operation. Amenorrhœa, impotence, or persistent infantilism occur in a patient whose skin is typically yellow, smooth, dry, and atrophic with loss of hair. Unlike Fröhlich's syndrome (p. 395), the patient is thin and cachectic from anorexia, suffering from weakness, lack of energy, and apathy, with low blood-pressure causing syncopal attacks and hypoglycæmia which occasionally gives rise to coma.

HYDROCEPHALUS

may be congenital or acquired.

Congenital hydrocephalus may be associated with spina bifida and meningocoele. Failure of development of the arachnoid villi through which absorption of the cerebrospinal fluid takes place is the commonest cause.

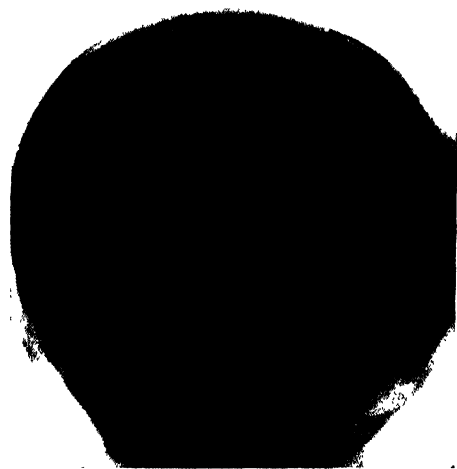


FIG. 465.—Ventriculogram showing enormous size of ventricles in congenital hydrocephalus.

This produces *communicating hydrocephalus* as the distended ventricles communicate freely with the sub-arachnoid space. *Obstruction of the cerebrospinal pathways* is produced by blockage of the outflow from the fourth ventricle associated with an Arnold-Chiari malformation of the hind brain in which the roof of the fourth ventricle lies below the level of the foramen magnum. Congenital stenosis of the aqueduct of Sylvius produces a similar effect.

Clinical Features.—The enlargement of the head may be pre-natal and obstruct labour. After birth, rapid enlargement usually occurs. The brow overhangs the roof of the orbits. The fontanelle is wide and

the scalp veins are distended (fig. 466). Although the ventricles become enormous and the cortex is reduced to a mere shell, there are no motor symptoms. Certain cases stabilise spontaneously, and intelligence is then preserved despite the enormous size of the head, but secondary endocrine effects result from pressure of the distended third ventricle on the pituitary fossa. Congenital stenosis of the aqueduct may not produce serious symptoms until the age of six or later, and then causes evidence of raised intracranial pressure associated with secondary endocrine disturbances.

Arne Torkildsen, Contemporary. Neurosurgeon, Rikshospital, Oslo, Norway.

Morris Simmonds, 1855–1925. Practised medicine in Hamburg.

Georges Froin, 1874–1932. French Neurologist.

Julius Arnold, 1835–1915. Professor of Pathological Anatomy, Heidelberg University.

Hans Chiari, 1851–1916. Professor of Pathological Anatomy, Strasbourg University.

François de le Boe (or Franciscus Sylvius, according to the Latinised form of his name), 1614–1672. Professor of Medicine, Leyden.

Treatment.—Attempts to overcome defective absorption include ventricular jugular anastomosis in which a non-return valve (Spitz Holter) is inserted between the distended ventricle and the lower portion of a divided jugular vein, or ventriculo-peritoneal anastomosis in which a long acrylic tube is laid subcutaneously from the ventricle and placed inside the lumen of a second long tube which is draining into the peritoneum. The overlap provided by a length of the small tube inside the large tube allows for the growth in length of the body. Obstructive hydrocephalus is treated by opening the distended third ventricle from the front—a third ventriculostomy.

Third ventriculostomy is the best operation for congenital stenosis of the aqueduct.

Acquired hydrocephalus of obstructive type is produced by pathological obstruction to the outflow from the ventricles. The outflow from one ventricle may be blocked by the obstruction of the foramen of Monro by adhesions following ventriculitis or by tumour.

Bilateral internal hydrocephalus accompanies midline tumours, e.g. colloid cysts of the third ventricle, craniopharyngiomas, pineal and cerebellar tumours, and arachnoid cysts over the roof of the fourth ventricle.

Treatment of Obstructive Hydrocephalus.—The site of obstruction is localised by air or myodil ventriculography. Adhesions at the foramen of Monro or colloid cysts of the third ventricle are removed by an approach across the anterior horn of the ventricle. Obstruction of the aqueduct is treated by third ventriculostomy. Blockage of the roof of the fourth ventricle is dealt with by suboccipital craniectomy and removal of cysts and adhesions lying in the cisterna magna.



FIG. 466.—A marked degree of hydrocephalus showing the overhanging orbital margin and distended veins.

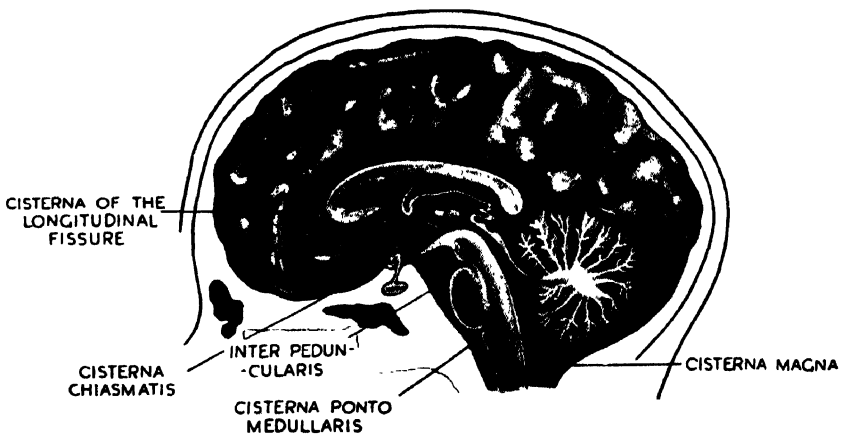


FIG. 467.—The principal arachnoid cisterns.

Communicating hydrocephalus complicates acute or chronic meningitis and sarcoidosis and cisterna chiasmatis, by inflammatory exudate or arachnoiditis throws a dam across the main channel for the ascent of cerebrospinal fluid to the absorption areas, and hence leads to stasis in, and distension of, the lateral ventricles (fig. 467).

An attempt must be made to clear the basal cisterns by promoting absorption of exudate. In tuberculous meningitis—by energetic treatment with PPD and streptomycin, in sarcoidosis—by ACTH. Surgical treatment is limited to the formation of bifrontal burr holes, through which the ventricle can be tapped to relieve acute pressure symptoms or to introduce antibiotics.

INTRACRANIAL ANEURYSM

Intracranial aneurysms may be considered in two main groups (Jefferson):

- (1) *Subclinoid*.—Acquired aneurysms on the carotid syphon of the internal carotid artery within the cavernous sinus.
- (2) *Supraclinoid*.—Congenital aneurysms of the circle of Willis.

Subclinoid aneurysms are produced by weakening of the muscle coat of the internal carotid by uneven distension of its walls at the anterior and posterior curves of the carotid syphon. The condition is most common in women, owing to the finer structure of their arteries. (Jefferson remarked that this is the only known pathological evidence of the existence of a weaker sex.)

Fusiform aneurysms are produced on the anterior and posterior bends, and hence anterior and posterior syndromes may be recognised which vary only in regard to the physical signs produced by pressure of the aneurysm sac on adjacent structures.

Symptomatology.—A female patient is suddenly seized with severe unilateral headache, resulting from stretching of nerve fibres in the wall of the distending carotid artery. This ceases only when the wall of the artery comes against the wall of the cavernous sinus and receives support from this structure. The headache ceases, but soon additional symptoms appear from pressure on the nerves in the lateral wall of the cavernous sinus. These produce a combination of squint and trigeminal pain.

Anterior Aneurysms.—The commonest type press on the third and fourth nerves and first division of the fifth, causing supraorbital pain associated with ptosis, a dilated pupil, and divergent squint, owing to paralysis of the third nerve and unopposed action of the sixth.

Posterior aneurysms press on the sixth nerve and the whole of the trigeminal, causing pain in the entire face with a convergent squint from sixth-nerve paralysis and unopposed action of the third, the eye remaining open.

Treatment is highly successful except in long-standing cases where the sac has filled with hard organised clot and cannot collapse. Ligation of the internal carotid might cause hemiplegia if the circle of Willis were sclerotic or if the anterior communicating artery were absent. Ligation of the common carotid, however, is employed with safety and with good effect. This reduces pressure in the sac and relieves the nerve symptoms without risk of hemiplegia, since there is a second-cross circulation above the level of the ligature. Blood passes across through the branches of the external carotid to reach the site of the ligature, then downwards to the carotid bifurcation above the ligature and upwards with the internal carotid with reduced force but in sufficient volume to prevent cerebral anæmia.

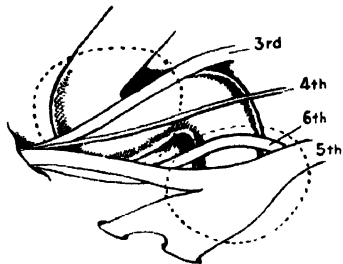


FIG. 468.—The relationship of aneurysmal sacs to structures in wall of the cavernous sinus.

Arterio-venous fistula in the cavernous sinus may occur *spontaneously* from rupture of a subclinoid aneurysm or from the effects of *trauma* on the arterial walls in association with fracture of the middle fossa. Acute back pressure in the veins draining into the cavernous sinus produces pulsating exophthalmos, distension of the orbital and supraorbital veins, and bone erosion and enlargement of the orbit. A few cases heal spontaneously by aseptic thrombosis in the cavernous sinus occurring in the early stages. Usually, however, the condition will progress if untreated, and produce a terrible deformity, with extrusion of the eyeball.

Treatment.—Ligation of the internal carotid in the neck may be sufficient, but if the condition progresses as the result of cross circulation through the circle of Willis, the internal carotid must be occluded intracranially by the application of a silver clip inserted across the vessel above the level of the cavernous sinus.

Supraclinoid Aneurysms.—Congenital aneurysms of the circle of Willis may produce pressure on adjacent nerves. Rupture produces symptoms of subarachnoid or intracranial hæmorrhage with or without associated pressure symptoms. They may rarely produce multiple emboli causing a progressive syndrome of paralysis.

Pressure Symptoms.—Aneurysms on the posterior branches of the circle, posterior cerebral and posterior communicating, produce symptoms of third nerve palsy. Posterior cerebral aneurysms also cause hemianopia or pressure on the crus. Aneurysms of the anterior branches, such as the internal carotid bifurcation, anterior cerebral, and anterior communicating produce pressure on the optic tracts, optic nerves, and chiasma respectively, producing hemianopia, monocular visual loss, or bi-temporal hemianopia and optic atrophy.

Middle cerebral aneurysms produce symptoms of hemiparesis from involvement of the striate branches, often by emboli. These symptoms may occur alone or in association with subarachnoid or intracerebral hæmorrhage.

Subarachnoid hæmorrhage produces sudden headache with severe pain in the neck and between the shoulders, followed by loss of consciousness of brief duration and later by acute pain from root irritation and marked neck stiffness.

Intracerebral hæmorrhage is to be suspected when sudden loss of consciousness persists for more than twenty-four hours and is associated with marked neck stiffness.

Treatment.—The objective of surgical treatment is to prevent repeated hæmorrhage. A patient dangerously ill from subarachnoid hæmorrhage will not be saved by early surgery, wherever the aneurysm is; even angiography may do harm, and therefore it should be delayed until the patient (or his hemisphere) has become fit for intervention. Following the first subarachnoid hæmorrhage, the patient is rested, if possible for ten days in order to allow the aneurysm to clot. If, however, repeated hæmorrhage occurs in the early stages, arteriography must be performed early and the common carotid on the affected side must be ligated in an effort to stop recurrent bleeding. If prolonged unconsciousness indicates intracerebral bleeding the clot is evacuated by a puncture through a burr hole or by craniotomy, in which case the aneurysm may be clipped after the clot has been removed. If there is no immediate urgency, after a ten days' rest, the aneurysm is localised by bilateral arteriography during which the presence or absence of cross circulation in the anterior communicating artery is determined by compression tests. If no aneurysm is seen on carotid angiography, a vertebral angiogram is performed. The best results are found in those cases in which no aneurysm is demonstrated; here a small aneurysm has probably undergone a spontaneous cure by clotting. Arteriography is performed at the tenth day—as statistics show that repeated hæmorrhage becomes more frequent after that interval.

If an aneurysm is demonstrated by routine arteriography at the tenth day, direct operation on the aneurysm is only justified if it is in a position where operation will not cause hemiplegia or mental symptoms.

Aneurysms of the anterior group are accessible to direct surgical approach. Anterior cerebral and anterior communicating aneurysms are approached like pituitary tumours. Middle cerebral and posterior communicating aneurysms are reached across the Sylvian fissure. The neck of a globular sac may be closed by applying a silver clip.

Recently, J. R. Gibbs has opened large sacs and reduced the lumen with a purse-string suture prior to occlusion of the neck. Fusiform aneurysms on the anterior cerebral and anterior communicating artery are treated by clipping the nutrient anterior cerebral artery proximal to the aneurysm.

A direct attack on an anterior communicating aneurysm is extremely hazardous, as this may cause spasm and thrombosis in the antero-median group of perforating vessels which supply the hypothalamus, with resulting infarction of that area and the production of a state of akinetic mutism in which the patient remains alive but deprived of speech or volition.

Direct attack on fusiform middle cerebral aneurysms carries a risk of hemiplegia, and these aneurysms are best treated by wrapping with muscle or acrylic plastic in order to strengthen the wall and diminish the risk of recurrent bleeding.

Intracranial angiomas are congenital malformations and not true tumours. Although an excess of blood passes through the large arterio-



FIG. 469.—Large aneurysm of anterior cerebral artery, producing monocular blindness.

venous malformations, absence of a true capillary system causes defective nutrition of the affected area, with death of tissue, gliosis, and calcification. This may produce epilepsy and paralysis, often in adult life. Alternatively, the angioma may rupture spontaneously or following a minor trauma, producing apoplexy in young subjects. Common carotid ligation always reduces the rate of blood flow through the tumour bed, which can be completely filled by cross circulation. Small surface angiomas may be successfully excised or caused to collapse by occlusion of the nutrient vessels.

SURGICAL TREATMENT OF MENTAL DISORDERS

In recent years the surgical treatment of mental illness has undergone refinement. The early crude beginning followed the observation by Egas Moniz in 1936, that the injection of alcohol into both frontal lobes would produce a diminution in the intensity of emotional distraction in severely disturbed psychotic patients.

The operation of pre-frontal leucotomy sought to secure a similar effect by sub-cortical division of the entire central cone of white matter in both frontal lobes by a vertical incision in front of the ventricles. Although this operation relieved symptoms of agitation and tension it caused extensive brain damage with serious blunting of intelligence, flattening of emotion, and a high risk of epilepsy and could therefore only be considered in seriously disturbed schizophrenic patients who seldom made a satisfactory response—fewer than one fifth being discharged from hospital, the remainder staying in mental hospitals to form a permanent source of discouragement to psychiatrists. However, it could be seen that if a similar reduction in the intensity of emotional reaction could be secured without damaging effect, surgical treatment might be extremely valuable in cases of incurable psychoneurosis such as suicidal depression, melancholia, anxiety states and obsessional syndromes.

Fulton's ablation experiments in monkeys showed that lesions in the region of the primitive agranular cortex on the mesial and orbital aspects of the lobe produced an alteration in emotional tone without intellectual defect, whereas lesions on the lateral aspect of the hemisphere, the parts affected by leucotomy, had a reverse and unfavourable effect, it therefore appeared that the future of surgical treatment would be found to lie in operations that interrupted the connections of the agranular cortex in the limbic system.

Le Gros Clark's observations in 1949 on the distribution of the thalamo-frontal and fronto-hypothalamic pathways concerned in emotional feeling and reaction, provided a target for selective operations on limited cortical areas of the cingulate, convexity and orbital cortex which were excised by lobectomy or isolated by undercutting whilst leaving the majority of the frontal lobe undisturbed in attempts to produce a reduction in emotional tension in psychoneurotic subjects. Certain of these operations, notably those that affect regions of primitive agranular cortex such as cinglectomy and restricted orbital undercutting, were highly successful in the treatment of obsessional illness and depression.

In 450 cases of Restricted Orbital Undercutting (Knight) the best results were among the depressions, over 60 per cent. of whom could be freed from medical care when psychiatric treatment had failed. Certain cases of chronic alcoholism and drug addiction were favourably influenced but the scar in the brain still produced a 10 per cent. risk of epilepsy and 3 per cent. risk of undesirable personality change. Observations indicated that the benefit was derived from the posterior 2 cm. of undercutting incision which enters the substantia innominata beneath the head of the caudate nucleus and here divides fibres which are converging from the primitive cortical areas of the cingulate posterior orbital and amygdaloid regions to pass beneath the head of the caudate nucleus to enter the hypothalamus. At this site these pathways which are concerned in emotional reaction can be selectively divided by small lesions which leave the rest of the frontal lobe and the thalamo-frontal pathways of emotional feeling intact at a higher level.

The operation of stereotactic tractotomy recently devised (Knight) destroys this limited zone by the insertion of radioactive seeds or electro-coagulation under X-ray control and eliminates the harmful effects of cutting in the brain. Satisfactory results are observed in depression, obsessional illness and anxiety states without epilepsy, mortality, or personality change. The accuracy of operation has produced cures in many cases of previous failed leucotomy.

SURGICAL TREATMENT OF PARKINSONISM

Parkinson's disease (*syn. paralysis agitans*) is a condition characterised by tremor, rigidity, and akinesia. Unilateral or bilateral rhythmic tremor, producing the typical pill-rolling movement of the fingers and thumbs, may also affect other sites, e.g. the face, jaw, palate, tongue, and lower limbs. Excessive muscular tonus causes a rigid, expressionless, mask-like facies, an unblinking stare, and limb rigidity. The shuffling Parkinsonian gait with short steps results from rigidity and akinesia. A variable degree of dementia is also present in some cases. It is necessary to estimate the part played by each symptom in causing disability.

The syndrome is produced by degenerative changes in the extrapyramidal pathways—particularly at the substantia nigra in the brain stem. Degeneration of the large pallidal cells of the corpus striatum may be responsible for excessive rigidity.

Surgical Treatment:

Small discrete lesions in the globus pallidus or ventro-lateral nucleus of the thalamus are found by stereotactic technique. The target area is determined by air and myodil ventriculography which shows the position of the anterior and posterior commissures to which the globus pallidus and ventro-lateral nucleus bear definite measurable relationships. A burr hole opening is made in the skull in an appropriate situation. Special aiming apparatus is then fixed to the skull carrying a device adjustable in the sagittal and in the coronal plane, by which a needle or electrode is introduced through the burr hole and advanced towards the target for an appropriate distance, as measured from the X-rays. The position is checked by electrical stimulations, and when the correct site has been reached, small areas can be destroyed by electro-coagulation, by the introduction of radioactive substances, or by injection of alcohol.

Results suggest that tremor responds best to lesions in the thalamic nucleus, and rigidity to lesions in the globus pallidus and adjacent internal capsule. Combined lesions at both sites give the best results. Patients with unilateral syndromes sufficiently severe to interfere with their occupation respond best. Bilateral syndromes are treated in two stages, at an interval of months.

If mental impairment is prominent before operation, added mental inertia resulting from the operation may reduce the patient to inactivity, although symptoms are relieved.

James Parkinson, 1755-1824. English general practitioner who practised in Shoreditch, London.

CHAPTER 18

THE SPINE

GEOFFREY KNIGHT

Sprains of the spinal column are of common occurrence. Spinal ligaments are injured as a result of a sudden jolt, as in car or railway accidents. Fibres of spinal muscles are torn by excessive muscular contraction as in lifting heavy weights. Severe localised pain follows, accentuated by any movement which stretches the damaged structure. Palpation reveals a tender spot, and if muscular fibres are torn some boggiess, due to extravasation of blood, is often detected. Treatment consists of rest and cold applications followed a few days later by massage and graduated movements. If the site of pain can be localised with accuracy, an injection of 5 ml. of procaine (2 per cent.) may cause immediate improvement. This is particularly true in relation to the interspinous ligaments which are liable to be overstretched in association with compression fractures of the vertebræ, or may produce long-lasting syndromes of chronic pain as a result of local injury to the ligament itself.

DISLOCATION

True dislocations can occur only in the cervical region, the oblique and vertical directions of the articular processes do not permit of dislocation without fracture in the dorsal and lumbar regions.

Dislocation following hanging occurs between the atlas and the axis.

Forward displacement of the atlas follows rupture of the transverse ligament, or less commonly fracture of the odontoid process. Death occurs immediately from injury to the brain stem and paralysis of the muscles of respiration. Dislocation at this level has also occurred from lifting a child by means of the hands encircling the neck from behind.

The base of the odontoid process may be cracked following head injuries and delayed displacement may occur after an interval of days. X-rays of the odontoid process, taken through the mouth, are essential where persistent occipital pain complicates head injuries.

Dislocations usually occur at the sites of maximum mobility, between the fourth and fifth, and fifth and sixth cervical vertebra and result from falls on the head when riding, or diving into shallow water, which causes excessive flexion of the upper part of the spine. The inferior articular process of the the fifth vertebra is forced over the front margin of the superior articular process of the sixth. When bilateral there are usually some associated features.

When the dislocation is unilateral the head is turned towards the opposite side and all movements are restricted. Severe head pain occurs if a nerve is nipped in the intervertebral foramen. Unilateral dislocations are readily reduced under general anæsthesia, by further flexion of the head, followed by lateral flexion towards the opposite side. If reduction fails, the joint should be exposed, and a minimum of bone removed in order to allow

locking. If the dislocation is unreduced, pain and deformity will persist. Involvement of the spinal cord is uncommon in one-sided dislocations.

In *bilateral* cases associated damage to the cord often occurs, usually of a complete nature. In more fortunate cases, owing to the large size of the vertebral canal, the cord escapes serious injury. The head is displaced forwards, and obvious deformity is present. Pain is referred to the neck, or arms, along the compressed nerve roots.

Emergency reduction should be attempted under anæsthesia. If reduction is accomplished, traction is maintained and a plaster cast is then applied. Should manipulation fail, continuous skeletal traction is applied by means of a skull calliper. Only as a last resort is open operation performed. A minimal amount of bone is removed from the articular processes until unlocking is possible. To prevent recurrence the spinous processes should be wired together. The head and neck are immobilised in plaster for six weeks.

Pathological dislocation of the atlanto-axial joint occasionally complicates a retropharyngeal abscess, or even tonsillitis. Flexion of the head and torticollis are obvious signs. The condition is confirmed by X-rays. Traction is required for a few weeks, followed by plaster fixation, and the pharyngeal abscess receives appropriate treatment (p. 621).

FRACTURES

Incomplete fractures do not interfere with the continuity of the spinal column. They include fractures of the spinous and transverse processes, laminae, and fissured or compression fractures of a vertebral body. Fractures of the processes or laminae are usually due to direct violence. Fractures of the spinous processes are common in the dorsal region for the processes are long and exposed in injury in falls on the back. Shovellers' fracture is a 'stress' fracture of spinous processes which occurs in men who use a shovel excessively, especially if they are undernourished. Localised pain and perhaps crepitus suggest the nature of the injury which is seldom shewn by radiography. Fracture of the transverse processes occurs most usually in the lumbar region, where these processes are long and comparatively unprotected; a corresponding kidney may be injured. Fracture of a lamina may be associated with depression of bone and injury to the underlying cord.

Compression fracture of a vertebral body may cause immediate hæmatomyelia and cord damage. Usually, however, only severe local pain is experienced necessitating X-ray examination, which may only reveal compression in the lateral view. It is probable that most cases of Kümmell's disease are due to compression of a vertebral body at the time of injury rather than the development of osteitis, as was formerly believed. Pain and deformity lead to an X-ray examination which reveals rarefaction and collapse of the body of a vertebra, usually in the lumbar region (fig. 470). Support to the spine is necessary for one year although Albec's bone-graft operation finds favour with some surgeons.

Traumatic intraspinal hæmorrhage may occur in association with fractured laminae or pedicles, or compression fractures.

Hæmorrhage occurs either in the cord itself (hæmatomyelitis) or as an extramedullary condition (hæmatorrhachis); in the latter case the blood escapes either into the cerebrospinal fluid or the extradural space.

Intradural hæmorrhage produces symptoms of root irritation and is detected by finding old blood and an excess of lymphocytes in the cerebrospinal fluid at lumbar puncture.

Extradural hæmorrhage may fill the spinal canal gradually from below upwards causing progressive compression of the cord, followed by paraplegia (Thorburn's gravitation paraplegia).

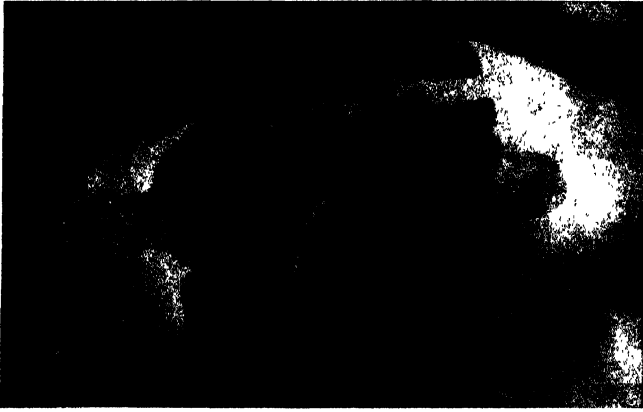


FIG. 470.—Kümmell's disease affecting the first lumbar vertebra.

Intramedullary Hæmorrhage.—Hæmatomyelia causes symptoms which follow the injury immediately. Destruction of the anterior horn cells causes a flaccid paralysis of the muscles concerned, while injury of the pyramidal tracts results in some spasticity of the legs. Pain and irritation are much less than in cases of extra-medullary hæmorrhage. Bruising of the cord against the bony bars produced by cervical spondylosis sometimes results from minor falls or whiplash injuries. In the early stages symptoms and signs are often vague. Subsequently, obvious neurological signs, e.g. muscular wasting, present themselves, and are usually permanent.

Complete fractures interrupt the continuity of the spinal column and consist of fracture-dislocations of the spine. These are usually produced by excessive flexion and occur at the sites of maximum mobility, C.5 to C.7 and L.3 to L.5, or at the junction of fixed and mobile portions of the spine. The prognosis is governed by the presence or absence of injury to the spinal cord and nerve roots.

SPINAL CORD INJURY

is produced by three factors:

- (1) By long axis stretch occurring at the moment of acute flexion, causing concussion or rupture of nerve fibres and vessels within the cord.
- (2) By nipping of the cord between the lamina of the vertebra above and the edge of the fractured body below, producing a local crush of the cord.
- (3) By the protrusion of a disc at the moment of flexion leading to compression of the anterior aspect of the cord.

In the adult the spinal cord ends at the level of the lower border of the body of the first lumbar vertebra; hence fracture-dislocations below this level can only be associated with injury to the cauda equina. Anatomical factors govern the extent of the cord and nerve lesion at various levels.

In the cervical spine the horizontal line of the articular processes and the large intervertebral discs produce a high degree of mobility with little strength, hence dislocation or fracture-dislocation occurs readily as a result of relatively minor forces sustained in injuries at sport, such as falls on the head in riding, diving, cycling, or football. Since there is relatively little 'follow-through' produced by small forces and since the size of the

neural canal in the cervical region is large in relation to the cord, cord injury may be partial or absent. When greater forces are in action, as when the neck is struck by a falling bough, considerable displacement occurs. Cord injury is then complete and often fatal.

In the dorsal spine the vertical articular processes and low mobility, combined with great strength, account for the fact that the spine can only be broken by major forces, such as the collapse of a roof in a mine-working; displacement is therefore considerable, little space is available around the cord, and cord injury is invariably present and usually severe.

In the lumbar spine vertical articular processes and great strength are combined with mobility. Fracture-dislocations are produced by major forces, often combined with some degree of rotation which causes interlocking of the displaced articular facets. But since the roots of the cauda equina enjoy ample space in the lumbar theca, nerve injury may be absent or limited to a few roots of the cauda. The treatment of fracture-dislocations (pp. 407 and 408) is of secondary importance to the treatment of the associated cord injury.

Injuries of the spinal cord occurring in association with fracture-dislocation and dislocation consist of (1) spinal concussion or (2) partial or complete spinal contusion.

Spinal concussion (spinal shock) is produced by long axis stretch on the spinal cord accompanying flexion. The grey matter of the cord consists of numerous nerve cells and synapses supported by a delicate tissue framework. A long axis stretch causes displacement of these structures with resulting disturbance of synaptic conduction. There is therefore an immediate loss of all functions which depend upon synaptic activity below the level of the lesion. Each anterior horn cell receives some hundreds of synapses, the stimuli passing through which maintain the central excitatory state in the nerve cell, by which the cell is maintained in a condition of readiness to respond and act as the final common path for voluntary movement, muscle tone, and reflex activity.

Cessation of the central excitatory state following synaptic disturbance therefore results in complete loss of voluntary movement, abolition of tone and reflex activity below the level of the lesion, producing a flaccid paralysis and retention of urine.

Pain and temperature sensation which cross a synapse in the inferior sensory decussation are also lost, but joint position sense which ascends the posterior columns is preserved. If the injury is merely spinal concussion, voluntary power and sensation will return within twenty-four to forty-eight hours. If concussion is superimposed upon an underlying partial or complete cord injury, the stage of spinal shock is prolonged. As time passes without improvement, recovery becomes increasingly improbable, and the appearance of a mass reflex at three to six weeks renders the outlook gloomy.

Partial or complete contusion of the cord is produced by nipping of the cord between the lamina of the displaced vertebra above and the edge of

the fracture vertebra below ; this causes a transverse zone of contusion and bruising. Above and below this level minute petechial hæmorrhages may be seen, resulting from the rupture of cord vessels by the long-axis stretching force. Œdema developing around these hæmorrhages may lead to a rise in the level of paralysis and sensory loss in the early days after the injury. Massive hæmorrhage 'hæmatomyelia' is occasionally seen in the cord substance. Any damage inflicted on the cord is permanent and irreparable.

Clinical Features.—The immediate effect of injury produces an initial picture of spinal shock resulting from the long axis stretch which masks the effects of the underlying organic rupture. There is, therefore, complete flaccid paralysis below the level of the lesion with retention of urine. The diagnosis of cord contusion can only be made with certainty in the early stage :

(1) If there is complete loss of all forms of sensation below the level of the lesion. This indicates interruption of the posterior columns (which escape the synaptic disturbance of concussion) by bruising on the back of the cord.

(2) If the level of sensory loss and paralysis rises after injury, indicating ascending œdema.

(3) If the stage of spinal shock persists for more than forty-eight hours. In general terms, the duration of the stage of flaccid paralysis is proportional to the severity of the contusion. This stage of spinal shock may last for weeks in severe cases.

The clinical course of cord contusion falls into three stages :

- (1) The stage of spinal shock.
- (2) The return of reflex activity.
- (3) The stage of septic complications.

In the stage of spinal shock, suitable measures are taken to protect the cord from further damage, and await the return of reflex activity in order to judge from the character of the reflex changes observed the nature and extent of the cord lesion. During this period precautions are taken to prevent or combat the onset of septic complications, such as urinary infection, bedsores, and pneumonia, in order to bring the patient to the final phase of active rehabilitation.

The Management of Cord Injuries.—In the stage of spinal shock every care must be taken to avoid unnecessary movement or examination which might increase the cord damage.

First Aid Treatment.—Cervical injuries may be transported on the back with the head supported between sandbags without flexion. Dorsal and lumbar fractures should be transported with the patient face downward with the pelvis and shoulders raised on pillows to hyperextend the spine. The patient must be lifted and turned on to a blanket, placed on a stretcher, by three people (or more, if the patient is heavy), controlling the head, trunk, and pelvis, without bending or torsion of the unstable spine. Morphia or

other available sedation should be given to relieve pain and anxiety.

Clinical Examination.—On arrival at hospital the patient should be left lying on his stretcher until examination is completed and immobilisation is carried out. He should not be moved either for X-ray or clinical examination. Partial cord lesions may be rendered complete by excessive movement during clinical examination or overzealous attempts to obtain a 'good' X-ray picture.

Evidence or history of an accident is usually obtainable. Neurogenic shock frequently accompanies the injury, but the patient may be able to state that he has severe pain in the back, girdle pains encircling the body, or that he 'feels dead' below a certain level.

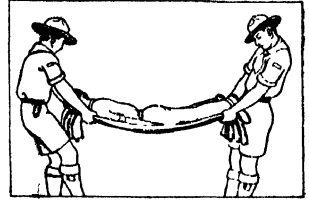


FIG. 471.—Transporting a patient with a fractured spine.

The utmost gentleness must be exercised in order to prevent further injury to a cord which is only partially damaged or to avoid injury to an undamaged cord. Deformity of the vertebral column may be noticed. Paraplegia and anæsthesia extending from the level of the lesion are usually present, and are due either to spinal concussion, or crushing of, the cord. The only examination required is to test the sensory level to pin-prick on the trunk, which will indicate the segmental level of the cord lesion, and to test whether the patient can recognise movement of the foot or knee. If he can appreciate these movements, the case may be one of concussion. If there is complete sensory loss the outlook is grave. Having determined the segmental level of the lesion, as good an X-ray as possible should be taken by the lateral view at the corresponding vertebral level without disturbing the patient. Fracture of the manubrium sterni may be associated with fractures in the dorsal region. If a fracture-dislocation is present the fracture is immobilised as soon as the patient's general condition permits, but it may be necessary before this to apply restorative measures to counteract shock.

Immobilization.—*Cervical fractures* are treated by head traction with skull callipers, the patient lying on an inclined plane. After reduction is complete a plaster collar is applied from the crown of the head to the chest wall encasing brow, head, neck, and upper thorax but leaving the face exposed, producing an appearance reminiscent of an ancient casque and shoulder armour, from which the term Minerva¹ collar derives.

Dorsal and lumbar fractures are treated by hyperextension. The fracture usually follows excessive flexion of the spine, and hyperextension corrects this deformity. In most cases morphia and scopolamine are adequate. Hyperextension is effected by laying the patient prone between two tables, the one which supports the head and arms being 12 to 18 inches (30 to 45 cm.) higher than the table on which the legs and pelvis rest; a sling, if available, gives comfortable support (fig. 472).

In special clinics an Abbott's frame and a canvas sling are used; straps are applied to the patient's ankles, which can be raised to the desired height. The trunk is covered with a stockinette vest, and the plaster is applied from pelvis below to the axillæ above. A window cut to compensate for abdominal distension. Acute dilatation of the stomach is a rare complication of spinal hyperextension as the third part of the duodenum may be thrust forward by the lumbar convexity.

Contraindications to Hyperextension.—In the following conditions the spine is immobilised in a normal posture:

¹ Minerva—Goddess of Wisdom and Arts.

1. Comminuted fractures which involve the neural canal. The spine is immobilised in the normal position with head traction if necessary.
2. Fractures due to hyperextension associated with rupture of the anterior common ligament of the vertebræ.

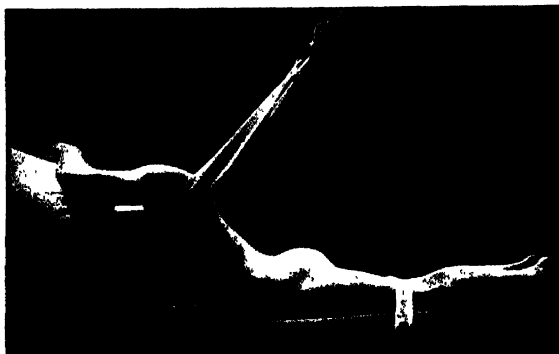


FIG. 472. — The spine is hyperextended by a band with block and tackle. The patient supports himself on a padded chair. The legs are strapped. The stockinette vest in this case has been omitted. (F. P. Fitzgerald, F.R.C.S.I., London.)

3. Fracture dislocation with locking of articular processes in the lumbar region. The superior processes of the lower vertebræ must be excised at a later stage.

Methods of immobilisation only protect the cord from further injury, they do not relieve the existing damage to the cord which may well be increased by unwise operative intervention, e.g. plating.

Nursing.—Symptomatic treatment is now instituted.

To prevent bed sore formation scrupulous drying and powdering of the skin is combined with regular turning of the patient every four hours to prevent local pressure, the patient being turned day and night in a Hey-Groves bed or on banks of pillows. Since plaster immobilisation increases the risk of sore formation plasters should only be kept on in cases of incomplete cord lesion in which there is prospect of neurological recovery. Once there is evidence that the cord is hopelessly damaged the plaster case should be removed to diminish risk of sore formation which delays the commencement of active rehabilitation. Attention to the bowels is necessary, and aperients or enemas are administered as required. If meteorism is allowed to occur upward displacement of the diaphragm causes pulmonary embarrassment. Hypostatic pneumonia is a grave danger, particularly if important accessory muscles of respiration, e.g., abdominal muscles, are paralysed, when coughing and expectoration are hampered. Contracture of muscles is prevented by frequent passive movements of the limbs.

Diet.—The patient should be given a high-protein diet to compensate for loss of serum from bed sores.

Care of the Bladder in Cord Injuries.—All cord injuries suffer from retention of urine in the initial stages. This may last for a few days to a year in recoverable lesions, or be permanent in complete lesions. Sixty per cent. of the mortality of spinal injury is attributable to ascending urinary infection producing pyelonephritis, and this infection results from instrumentation.

The Care of the Bladder is described in Chapter 46.

The Return of Reflex Activity.—Spinal shock should show some evidence of abatement at the end of 48 hours. If no sign of recovery is evident then the prognosis is gloomy for there is bound to be partial or complete contusion of the cord in such cases. The phase of spinal shock

and flaccid paralysis persists for days or weeks according to the severity of the cord lesion. When it passes off it will be found that lower motor neurone paralysis persists in a few muscle groups corresponding to the level of the lesion, owing to the crushing of the anterior horns at this level.

Partial cord lesions show spastic paralysis in extension with exaggerated reflexes and extensor plantar responses, and sometimes with uninhibited flexor spasms. There may, however, be some return of sensation. Retention of urine may persist for periods up to one year.

Complete cord lesions recover to a state of spastic paralysis in flexion with gross flexor spasms and the appearance of mass flexor reflexes of spinal automatism, which indicates complete lack of inhibition of the spinal flexor reflex arcs. In this reflex the slightest touch on the foot will produce flexion of the ankle, knee, and hip on the side of stimulation followed by a similar contraction of the opposite leg, sometimes associated with evacuation of the bladder and rectum. The sight of this reflex may be distressingly encouraging to the patient as it may be misinterpreted as evidence of recovering function, whereas it indicates that function can never recover. This mass reflex and the position of flexion may lead to the production of deep sores on the front of the thigh where this is cut into by the plaster shell. Since the cord is irreparably damaged the plaster case should be discarded once the diagnosis is established in order to keep the patient in as good a condition as possible for rehabilitation.

Cervical.—In high cervical lesions all the respiratory muscles are paralysed, including the diaphragm, which receives its motor supply from the phrenic nerve, mainly the C.4 segment. If the fifth segment is injured, the arms, trunk, and legs are all paralysed, and the patient only breathes with his diaphragm.

In lesions of the sixth cervical segment the only movement remaining is abduction of the shoulder by the deltoid muscles, the biceps and triceps and small muscles of the hand being totally paralysed. At the seventh segment flexion of the elbow is retained by the action of biceps and brachioradialis but the triceps is paralysed. Lesions at the first thoracic level produce paralysis of the small muscles of the hand with a Horner's syndrome from paralysis of the cervical sympathetic fibres which leave the cord at the first thoracic root (fig. 138). At lower levels a band of hyperæsthesia is usually detected encircling the trunk, one segment above the site of injury.

The lumbar enlargement corresponds to the twelfth dorsal and first lumbar vertebra and it contains the centre for the nervous control of the urinary bladder. In complete cord lesions above the tenth dorsal segment, after an initial phase of retention due to spinal shock, since the bladder centres are intact and uninhibited, an automatic bladder eventually develops, evacuation occurring when the intravesical pressure rises, the patient having to wear a receptacle.

Complete lesions involving the bladder centre at the lumbar enlargement produces permanent retention with overflow incontinence from paralysis of the detrusor muscle innervated by the third and fourth sacral nerves. But since muscles of the upper abdominal wall can be contracted voluntarily by the seventh to tenth dorsal segments, patients can be taught to evacuate the bladder by straining and by pressing their hands above the pelvis during the day. This is made easier if the resistance of the bladder neck is diminished by a transurethral 'V' resection of the internal sphincter (Chap. 47), but he must wear a receptacle at night.

The centre for defæcation is also situated in the lumbar enlargement and damage to this is followed by a patulous anus and incontinence.

Injuries to the nerves in the cauda equina gives rise to a degree of disability which corresponds to the actual nerves involved. Usually the injuries are in the lower lumbar region and spare the lumbar roots so that contraction of the muscles on

the front of thighs and legs are preserved. Involvement of the sacral roots however, produces anæsthesia in the back of the legs and saddle area of the peritoneum with urinary retention.

Rehabilitation.—If the patient has a partial or recoverable cord lesion, plaster immobilisation of the injured segment must be maintained for an adequate period of time of from four months, by which time stability is restored by the formation of massive areas of new bone in the anterior common ligament. If however, the cord is hopelessly destroyed, the plaster on the trunk should be discarded in order to minimise pressure-sore formation. The work of Guttman has revolutionised the prospects of the paraplegic patient, who can now be taught to work and earn a living, even with a complete lesion of the cord. The principles underlying Guttman's methods are:

- (1) To bring the patient into the best possible physical condition by the elimination of sepsis from bedsores and urinary tract.
- (2) To over-develop the muscles above the site of the injury so that these may move the pelvis.
- (3) To move the splinted legs by movement of the pelvis.

Toxic absorption can depress recovering activity in the cord. Once bedsores are established the plaster should be removed as soon as permissible in order to reduce pressure, the lesion then being treated by excision and skin grafting. The treatment of the established condition is described on p. 46.

Bladder training and the treatment of refractory cases or urinary infection is described in Chapter 46.

Rehabilitation of Muscular Activity and Locomotion.—Contractures are first relieved by physiotherapy, tenotomy, or partial neurectomy. If necessary, the lower portion of the completely divided cord is *destroyed* by intrathecal alcohol injection to abolish mass reflexes and flexor spasms which would prevent walking and which may be extremely painful if any sensory fibres persist. When the legs are straight, rope climbing and trapeze work is started to encourage enormous development of the muscles of the shoulder girdle. Lying in a sling, the patient then learns to swing the pelvis from the shoulder girdle using the long muscles, latissimus dorsi, erector spinae, and abdominal recti and obliques. Then, with his arms supported on parallel bars, he learns to move the pelvis like a hula-hula dancer, and when proficient in this art light aluminium splints with controllable knee-locks are fitted. Using sticks, the patient moves the pelvis from his shoulder and with the pelvis go the splinted legs, thus permitting the patient to walk slowly for short distances to a wheeled chair in which he may move about and engage in sport and training in specialised occupations, such as watch-making, lens-making, diamond-cutting, etc.

Complications.—Death may result from the following causes :

- (1) Shock, which may be immediately fatal.
- (2) Cessation of respiration from ascending cord oedema within hours or days, if the lesion is near the fifth cervical segment.
- (3) Hypostatic pneumonia within days or weeks, especially if the lesion is sufficiently high to cause paralysis of abdominal and intercostal muscles.
- (4) Infection is prone to spread to the kidneys, causing death from pyelonephritis (*syn.* surgical kidneys) within months or years. Necropsy reveals the renal cortex and pelvis studded with abscesses and areas of acute inflammation.
- (5) Urinary calculi may occur as immobilisation leads to decalcification, with increased renal calcium excretion, also recumbency encourages stagnation of urine in the kidney. Infection further increases the risk. Such calculi are usually painless, even though the kidney is both obstructed and infected, since the anæsthesia of spinal injury affects visceral as well as somatic structures.
- (6) Bedsores, which hasten death owing to toxic absorption.

Indications for Operation.

In the Acute Stage.—For the removal of a traumatic cervical disc protrusion causing cord compression without a fracture (p. 423) or for hæmatorraxis (p. 403). Apart from these rare conditions the indications for laminectomy are nil. The cord injury is irreversible and is made worse by meddling with indriven bone.

In the Late Stages.—1. Laminectomy is rarely required for the relief of cord compression produced by an arachnoid cyst which is impairing recovery of a partial lesion. Recovery occurs up to a point then deteriorates and lumbar puncture shows a block on Queckenstedt's test (p. 417).

2. Partial facetectomy may be required to reduce interlocked facets in the lumbar region.

3. Pain produced by root compression is treated by intrathecal phenol (p. 426).

Gunshot Wounds of Spine.—Portions of metal driven against the lamina or vertebral body and bouncing off away from the cord may nevertheless inflict an irreversible injury by exciting massive hæmorrhage in the cord substance. Direct penetration reduces the cord to a custard-like mass of diffused cord and bone chips.

INFECTIVE CONDITIONS

Meningitis.—Acute infective meningitis is likely to follow a penetrating wound, or may be a complication of septicæmia. Severe constitutional disturbances follow with local and referred pain, hyperæsthesia, muscular spasms, and increased reflexes. Extension to the basal meninges is usual, and the only hope lies in an adequate course of penicillin and chemotherapy.

Spinal Epidural Abscess.—Pus may collect in the extradural space of the cervical mid-dorsal or lumbar regions, to which infection has extended through the intervertebral foramina from adjacent sepsis, particularly from a perinephric abscess in the lumbar region. This produces a syndrome of progressive cord compression and meningeal irritation. The pus is evacuated by laminectomy.

SPINA BIFIDA

Embryology.—During the second week of intrauterine life a longitudinal furrow appears on the dorsum of the embryo, this groove being formed by infolding of the epiblast. The margins of the neural groove unite, so that it becomes converted into a tube from which the nervous system is developed. This epiblastic tube becomes separated from the surface by mesoblast, which grows over it from either side, and from which are developed the vertebræ, spinal muscles, membranes, etc. (fig. 473). In each segment bars of cartilage appear on either side of the neural tube, and during the fourth month they fuse with each other to form the vertebral arches. Failure of fusion of these arches gives rise to spina bifida, with which is frequently associated mal-development of the spinal cord and membranes. The incidence of spina bifida, excluding spina bifida occulta, is 0.1 per cent.

The types of spina bifida are as follows :

(1) *Spina bifida occulta* is due to failure of the neural arches to unite, but there is no protrusion of cord or membranes. Frequently only one vertebra is affected, most commonly in the lumbosacral region. A local patch of hair, a nævo-lipoma, or a depression in the skin are suggestive of underlying bony deficiency. A fibrous band, the *membrana reunens*, connects the skin to the spina theca. Growth of the body causes the membrana to pull on the theca and nerve roots. Foot-drop, nocturnal enuresis, or backache occur when the child is older or in adult life. Many cases are symptomless and are undiagnosed unless an X-ray is taken for some other reason.

(2) *Meningocele.*—This is a protrusion of meninges through a defect in the spino-laminar segment (fig. 474). It contains only cerebrospinal fluid.

(3) *Meningo-myelocele.*—The normally developed spinal cord or cauda

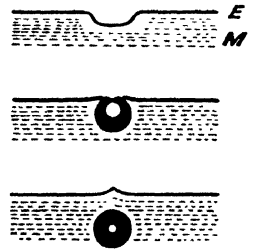


FIG. 473.—Development of the spinal cord. E = epiblast, M = mesoblast.



FIG. 474.—A typical meningocele which was excised successfully.

equina lies in the sac, and may be adherent to the posterior aspect. The cord or nerves can be seen as dark shadows on transillumination.

(4) *Syringo-myelocele*.—The rarest type of spina bifida, in which the central canal of the cord is dilated, and the cord lies within the sac together with the nerves arising from it.

(5) *Myelocele* results from arrest of development at the time of closure of the neural furrow. An elliptical raw surface is seen, which represents the ununited groove. At the upper end the central canal opens on the surface and discharges cerebrospinal fluid (fig. 475).

With the exception of spina bifida occulta, myelocele is the most common type of spina bifida, many cases are stillborn. If the child is born alive, death ensues within a few days from infection of the cord and meninges. Gross talipes is obvious.

Meningocele and meningo-myelocele are distinguishable on transillumination, a depression in the skin is sometimes produced by

FIG. 475. — Myelocele, with associated talipes.

adherent cord or nerves in a meningo-myelocele. Interference with the spinal cord or nerves may occur in either condition but is more severe in meningo-myelocele, in which condition bilateral talipes with trophic changes are common, and in advanced cases extensive paralysis of the legs and incontinence occur. In serious cases no surgical intervention is indicated.

Treatment.—Operation for meningocele or meningo-myelocele is advisable as soon as the surgeon is of the opinion that the child's strength and condition warrant the procedure (often within a few days of birth), otherwise the sac is liable to grow out of proportion to the growth of the child, and the overlying skin will become atrophic and ulcerate.

Operation is performed under local anæsthesia with the child tied face downwards on a splint. The child is kept with the head low to minimise escape of fluid, and



FIG. 476.—Spina bifida occulta with a tuft of hair, and wasting of the right leg due to traction on nerves in the spinal canal. Also right-sided Sprengel's shoulder.
(R. L. Benison, F.R.C.S., Wolverhampton.)

given a feeding-bottle during operation. The sac is opened and redundant membrane excised. If the cord or nerves are adherent, they are either freed by dissection, or separated with a strip of attached membrane and replaced in the vertebral canal. Membranes are sutured over the cord, spinal muscles are approximated, and the wound is reinforced with flaps of sheath from the erector spinæ muscles. Some cases subsequently develop hydrocephalus from the effect of associated congenital abnormalities at a higher level (p. 396).

Operation for spina bifida occulta is required when neurological symptoms develop later in life. These symptoms are only rarely produced by traction from the membrana reuniens alone but are more likely to be due to compression of the cord by intra- or extradural lipomas or the effect of disastemato myelia, a condition in which the cord is split in the mid-line by a bony spur which divides the neural canal beneath one laminæ into two lateral compartments. These abnormalities are almost always associated with the presence of a hairy tuft on the overlying skin, which is an important warning sign.

Myelography is required in all cases prior to operation.

At operation a tough fibrous band is encountered which leads down from the skin or deep fascia through a bony aperture to the dura mater. The dura is opened and the band excised together with any fibro-lipomatous tissue.

INFLAMMATORY DISEASES

Acute osteomyelitis occurs in the epiphysis of the body of a vertebra or, more rarely, one epiphysis of the neural arch. Severe constitutional disturbance is associated with local pain and tenderness. Pain may also be referred along an adjacent spinal nerve. If the disease commences in a neural arch, some local evidence of inflammation is often detected. Acute osteomyelitis of a vertebral body has been mistaken for spinal meningitis, acute appendicitis, acute pancreatitis, and other abdominal conditions.

As soon as the disease is suspected, penicillin is administered, supplemented by chemotherapy. X-rays assist in determining the progress of the disease, and evacuation of pus or sequestrectomy is performed if necessary.

Tuberculous Disease—p. 237

Spondylitis—p. 296

TUMOURS OF THE VERTEBRAL COLUMN

Innocent tumours are extremely rare and include chondromas, osteomas, and fibromas. Many cases previously described as chondromas were in reality protrusions of an intervertebral disc. These tumours produce a syndrome of spinal compression affecting the anterior surface of the cord (p. 423) associated with characteristic X-ray appearances.

Malignant tumours of the spine are primary or secondary. Primary sarcoma is most frequent in children and young adults. The neural arches are usually affected, a palpable swelling may be detected in the paravertebral muscles, or invading tumour is discovered at this site during laminectomy performed for spinal-cord compression. Operative removal is always incomplete. Deep X-ray therapy is seldom effective.

Secondary deposits in the spine are common and far outnumber primary spinal or spinal-cord tumours. The majority are produced by bone metastasis chiefly derived from the breast or prostate. Deposits (from broncho-carcinoma) may rarely occur in epidural fat or in the cord itself. Malignant ganglion neuroblastomas of the sympathetic chain may invade through the intervertebral foramina.

Clinical Features.—Bone deposits produce severe local pain which may be the only symptom for a variable period. Later, as nerves become involved, the pain becomes girdle in type and increases in intensity. It is aggravated by movement, so that the patient may remain crouched in a chair or huddled up in bed for hours at a time. If diagnosis is established

at this stage, deep X-ray therapy may prevent vertebral collapse and cord damage.

The sudden collapse of a vertebra in which a deposit has developed painlessly may lead to instantaneous paralysis and severe back pain as the first symptom. A similar disaster may occur if manipulation of the spine has been unwisely performed in an attempt to relieve the local pain produced by a vertebral deposit which has not been diagnosed. The diagnosis of secondary tumour should always be suspected when severe local pain is associated with a high erythrocyte sedimentation rate. Most commonly, a period of local pain is gradually followed by the onset of paraplegia and spasticity with increased reflexes, but once paraplegia is established it may become complete in a few weeks.

Treatment.—Secondary deposits from the prostate respond remarkably to treatment with stilbæstrol, and those from the breast may regress with hormone therapy, adrenalectomy or hypophysectomy. In other cases, treatment is limited to a support to the spine, analgesics, and deep X-ray therapy. Intrathecal phenol injections are a most satisfactory method of relieving agonising pain. Laminectomy is not advisable in the presence of vertebral collapse.

TUMOURS OF THE SPINAL CANAL

Tumours of the spinal cord and membranes are only one-tenth as common as cerebral tumours. On the other hand, at least 70 per cent. can be cured by operation.

Tumours of the spinal canal are extradural or intradural, the latter being either extramedullary or intramedullary.

Extradural tumours are relatively uncommon. Lipomas are described but are very rare. The majority consist of meningiomas or neurofibromas. Meningiomas form a plaque overlying several segments of the cord and compressing multiple nerve roots and the cord in the cervical and dorsal region.

Dumb-bell tumours are neurofibromas. Part of the tumour is extradural, and is connected with a larger paravertebral portion by a narrow isthmus which passes through an intervertebral foramen. Spinal cord compression eventually develops, necessitating laminectomy and excision. Erosion of an intervertebral foramen is a useful radiological aid to diagnosis.

Extramedullary tumours (75 per cent.) are neurofibromas and meningiomas.

Neurofibromas are commoner in males and usually arise from a posterior nerve root forming a fusiform tumour about 1 to 1½ inches (2.5 to 3.75 cm.) long present on the postero-lateral aspect of the cord. Occasionally there is an extradural dumb-bell extension through an enlarged intervertebral foramen.

Meningiomas occur almost exclusively in women and usually form a small globular tumour the size of a grape attached to the dura and indenting the cord.

Carpet-like tumours corresponding to the meningioma *en plaque* spread widely over the inner aspect of the dura in the cervical and lumbar regions, where there is considerable available space, and engulf the cervical cord or lumbar nerve roots before producing any symptoms of compression. In the author's experience, one tumour extended from the tenth dorsal vertebra to the bottom of the sacrum and measured 27.5 cm. in length.

Of all extramedullary tumours 12 per cent. occur in the cervical region,
60 per cent. in the dorsal region, and
28 per cent. appear in the lumbar region.

In relation to the cord, three-quarters are on the postero-lateral aspect and only a quarter on the anterior aspect. They are very easily accessible at operation and all

except a few diffuse meningiomas, or meningiomas invading bone, can be removed completely.

Intramedullary Tumours (25 per cent.).—Approximately half these growths are diffuse gliomas which cannot be removed. Others consist of ependymomas, a growth derived from the ependyma lining the central canal, forming either solid tumours like pencils which can be excised, or cysts which can be evacuated. Vascular malformations comprise 4 per cent., and for these little can be done. More than half the intramedullary tumours lie in the cervical cord.

Symptomatology

Spinal cord tumours produce symptoms :

1. By involvement of posterior nerve roots causing root pain and local sensory loss.
2. By involvement of anterior nerve roots and anterior horns causing lower motor neurone paralysis with wasting and loss of reflexes.
3. By involvement of long sensory tracts causing distant sensory loss below the level of the tumour.
4. By involvement of pyramidal tracts, causing upper motor neurone paralysis.
5. By involvement of both pyramidal tracts, causing disturbance of bladder function.

Extramedullary tumours are lesions of small size usually situated on the postero-lateral aspect of the cord in relation to posterior nerve roots, hence root pain is the earliest symptom which may precede any evidence of cord compression by a period of years. In the early stage it is often mis-diagnosed; when referred to the shoulders and arms, it may be regarded as arthritis or neuritis. In the thoracic region it is regarded as angina pectoris or pleurodynia. In the abdomen it may be thought to originate from the gall-bladder, appendix, or pelvic organs.

The spinal origin should be suspected because the pain is *increased by coughing*, which raises the intraspinal pressure and forces the nerve root against the obstruction. Secondly, because pain follows the distribution of a spinal root and is often bilateral. Owing to the overlap of adjacent sensory areas, local sensory loss is absent or slight. Since most muscles receive a double nerve supply, the lower motor neurone paralysis is not recognisable unless extremely important roots are involved, such as the first thoracic.

The distant sensory loss is produced by pressure on the cord, which affects the longest sensory fibres first, hence whatever the level of the tumour the sensory loss starts in the legs and gradually spreads up the body as shorter and shorter fibres become affected. It seldom corresponds to the level of the tumour and is usually below the tumour level except in the latest stages. Pyramidal involvement occurs later, and the bladder symptoms last of all.

Examination shows bilateral spastic paralysis with a transverse sensory level on the trunk below the level of the tumour. The site of the tumour is reliably indicated by the level of the root pain.

Intramedullary Tumours.—Fifty per cent. of intramedullary tumours occur in the cervical region of the cord. These tumours occupy a length of the centre of the cord. Root pain occurs last when the tumour presses against the walls of the spinal canal. Involvement of many posterior and anterior horns causes a wide belt

	<i>Extramedullary</i>	<i>Intramedullary</i>
Root Pain	Early	Late
Local Sensory Loss	Nil or slight	Wide
L.M.N. Paralysis	Absent except in C.8, T.1	Wide
Distant Sensory Loss	Ascending	Dissociated
U.M.N. Paralysis	Late	Early
Bladder	Late	Early

of local sensory loss and lower motor neurone paralysis, e.g. a dorsal tumour may cause extensive paralysis of the abdominal muscles. Distant sensory loss may be disassociated in type, as in syringomyelia, from involvement of spinothalamic tracts. Spastic paralysis and bladder symptoms from pyramidal involvement occur first of all.

Examination reveals a wide belt of local sensory loss and lower motor neurone paralysis associated with spastic paralysis in a patient who has only complained of increasing paralysis and bladder disturbance of a few weeks' duration.

Extramedullary Tumours of the Conus Medullaris and Cauda Equina.—All lumbar and sacral nerve roots leave the cord between the tenth thoracic and first lumbar vertebra. Tumours of the conus medullaris lie at the eleventh and twelfth dorsal and the first lumbar vertebrae, and press upon multiple lumbar nerve roots and the tip of the cord. The symptoms include root pain, sensory loss, and lower motor neurone paralysis in the distribution of the lumbar roots which supply the front of the thigh and legs. There is pain in the thighs, wasting of the quadriceps, foot-drop and reduction of the knee-jerks. Pressure on the cord produces spastic paralysis in the legs with increased ankle-jerk and extensor plantar response.

Reflexes :	K.-J.	—	—
	A.-J.	+++	+++
	P.-R.	↑	↑

Tumours of the cauda equina occupy a central position among the sacral nerve roots below the conus, producing root pain, sensory loss, and lower motor neurone paralysis in the distribution of the sacral roots which supply the back of the legs. There is sciatic pain and wasting of the hamstrings and calves, and sensory loss in the saddle area. The knee-jerks are increased owing to weakness of the opposing hamstrings; the ankle-jerks and plantar responses are absent owing to sensory disturbance :

K.-J.	+++	+++
A.-J.	—	—
P.-R.	—	—

The more important localising features are as follows :

Upper Cervical.—Tumours in this region are occasionally fatal from involvement of the phrenic nerve centres, and interference with the medulla oblongata sometimes causes cardiac irregularity and hyperpyrexia. Involvement of the oculo-pupillary fibres which pass down to their nucleus in the first dorsal segment results in myosis, narrowing of the palpebral fissure, hyperhidrosis and enophthalmos (Horner's syndrome). Neuralgic pains are likely to occur over the mastoid and occipital regions, the neck, supraclavicular or acromial areas, or down the arms.

Lower Cervical and Upper Dorsal.—Irritation or compression of the oculo-pupillary fibres is liable to occur if the tumour is situated at or above the first dorsal segment. Interference with the anterior horn cells from the fifth cervical to the first dorsal segments involve characteristic groups of muscles (p. 441).

Examination of reflexes may yield valuable information. If the supinator reflex is absent, the tumour is situated at the level of the fifth or sixth cervical segment, while absence of the triceps jerk indicates interference with the seventh segment. A tumour in the lower cervical or first dorsal area is likely to cause pain radiating down the arms. Below this level, intercostal neuralgia or girdle pains are common symptoms.

Lower Dorsal.—Interference with the cord between the eighth and twelfth dorsal segments is likely to cause loss of upper or lower abdominal reflexes. Paresis of muscles is sometimes present, and if unilateral, a striking bulge of one-half of the abdominal wall is noticeable when the patient coughs.

Irritation of the seventh to the twelfth posterior roots causes a variety of abdominal symptoms, usually indefinite but persistent, and is a fruitful source of diagnostic errors.

Lumbar Cord.—Disturbances of the rectal and bladder sphincters are common. If the tumour is situated at or below the level of the second lumbar segment, one or both knee-jerks are diminished or absent. The cremasteric reflex is abolished if the tumour involves the upper part of the lumbar cord. Involvement of centres from which the lumbar plexus is derived causes paralysis and wasting of corresponding muscles. Owing to the comparatively small size of the lumbar and sacral segments of the cord, sacral nerves are commonly affected in addition to the lower lumbar.

Cauda Equina.—Pain occurs in the back, and extends to the perineum, genitals, and backs of the thighs. Sensory loss usually occurs over the distribution of the sacral nerves (saddle-shaped anæsthesia), and the lower the tumour is situated the

smaller is the area of anaesthesia. Bladder and rectal disturbances are usual, and paresis of muscles depends upon the actual nerves involved.

Spinal arachnoiditis may produce simple cysts of the arachnoid, form cystic collections which compress the cord, or multiple diffuse adhesions which produce their effect by traction on blood-vessels. The cysts should be localised and removed in the same manner as spinal tumours.

Accessory Methods of Investigation

1. X-ray of the spine may indicate the level of the tumour by showing absorption of the pedicles of the vertebra, enlargement of an intervertebral

foramen on the lateral view, or erosion of the posterior aspect of a vertebral body (figs. 477 and 478).

2. Lumbar puncture may be required in early cases to differentiate

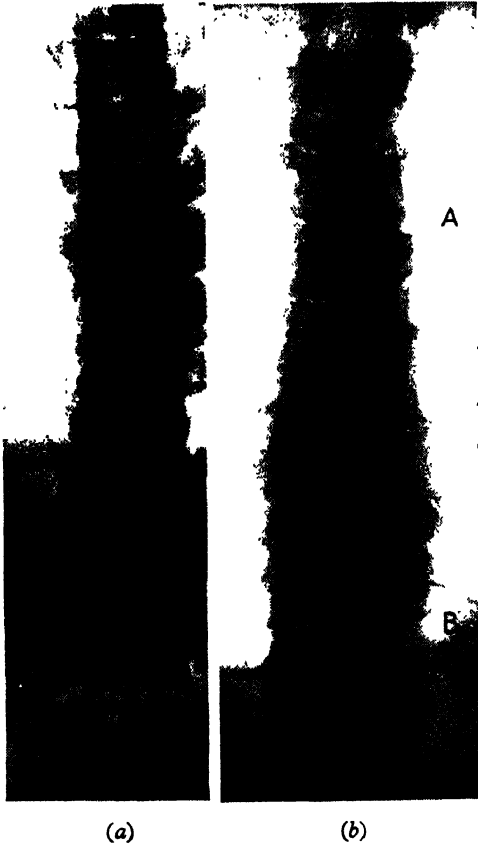


FIG. 477. — (a) Anteroposterior X-ray of spine showing normal pedicles. (b) Destruction of pedicles between sites A and B by pressure of a long fusiform intramedullary tumour.



FIG. 478. — Lateral X-ray showing marked hollowing out by pressure atrophy of the back of the bodies of the lumbar vertebra by a large tumour of the cauda equina extending throughout the lumbar theca. Similar appearances may also occur in the body of the sacrum.

such conditions as disseminated sclerosis. The presence of a tumour is indicated by an obstruction in the cerebrospinal-fluid circulation demonstrated by Queckenstedt's test. This is performed by charting the rate of rise and fall of the lumbar cerebrospinal-fluid pressure occurring during compression and release of the internal jugular veins. In normal circumstances, such compression causes a rapid rise in cerebrospinal-fluid pressure and a somewhat slower passive fall. The first effect of obstruction is to prolong

the passive fall, later there is a slight rise and fall, and finally no response at all.

3. Examination of the cerebrospinal fluid, including protein content.

In neurofibromas the cerebrospinal fluid protein is raised from the normal 20 to 40 mg. per cent. to figures of 200 to 400 mg. per cent. without any evidence of block. When block becomes complete, the cerebrospinal fluid in the lower portion of the theca is encysted and transudation occurs between

the blood-vessels and the cerebrospinal fluid with diffusion of protein and pigments. The fluid becomes yellow and may clot spontaneously (Froin's syndrome), and may contain as much as 5,000 mg. of protein per cent.

Cisternal Myelography. —

Cisternal myelography is essential in order to demonstrate the level of the upper border of the tumour before operation. The shape of the shadow produced at X-ray indicates the type of the tumour (figs. 479, 480, 481, and 483).

Cisternal puncture is performed by inserting a lumbar-puncture needle

in the mid-line at the level of the second cervical spine, the patient's head being flexed. The needle is advanced on a plane passing through the external auditory meatus to the outer margin of the eye. At a depth of 3 cm. the stylet is removed.



FIG. 479.—Descending cisternal myelogram showing complete obstruction of the down fall of myodil and the wavy outline produced by the upper border of a meningioma.



FIG. 480. — Descending myelogram showing complete obstruction and the smooth curved margin produced by the curved upper surface of a neurofibroma.

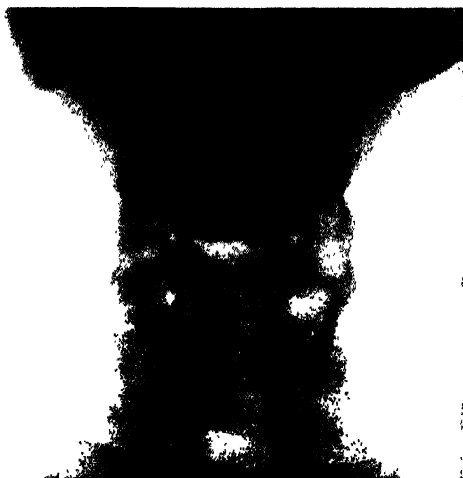


FIG. 481.—Descending myelogram showing the typical candle-grease appearance produced by myodil trickling down on each side of an expanded cord, an appearance characteristic of intramedullary tumour.

The needle is further advanced to a depth of 5 cm., when it pierces the occipito-atlantoid ligament, which feels like a sheet of brown paper; the moment this is pierced the operator should wait for fluid to flow out. It may not do so although the needle is in the cisterna magna, therefore the needle should be aspirated before it is advanced farther. If no fluid is obtained, it is put in a millimetre or two at a time until fluid escapes. Three ml. of Myodil is now injected and gravitates downwards until it is arrested by the tumour, the level of which is localised by examination on a tilting X-ray screen (fig. 482).

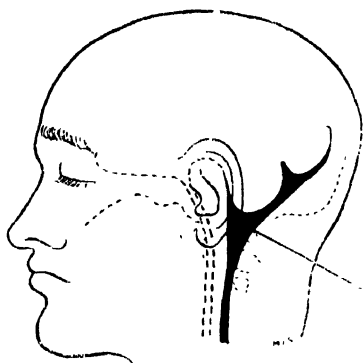


FIG. 482.—The technique of cisternal puncture.



FIG. 483.—Descending myelogram showing the characteristic curved lower border of the myodil shadow which is produced by the curved sac of arachnoid cyst.

Treatment is by laminectomy when the upper level of the tumour has been accurately localised by cisternal myelography. A meningioma is excised together with a portion of the dura from which it arises.

A neurofibroma is removed with a portion of the nerve sheath to which it is attached.

Intramedullary solid ependymomas are excised after incision of the cord strictly in the mid-line between the posterior columns.

Intramedullary cysts are emptied.

LAMINECTOMY

In estimating the level of operation, the discrepancy between the level of the affected segment of the cord and that of the corresponding vertebra must be considered. It must be remembered that owing to the disparity in the length of the spinal cord and vertebral column, a segment in the cervical region lies one vertebra above its corresponding vertebral body. In the upper dorsal region the tumour will be two vertebræ, and in the lower dorsal three vertebræ, above the corresponding vertebral body.

In estimating these levels by palpation of the spinous processes, it must not be forgotten that in the dorsal region the processes overlap the vertebra below. Thus, presuming a tumour is localised to the ninth dorsal segment, it should be opposite the tip of the fifth spinous process, i.e. three vertebræ higher on account of the shortness of the cord as compared with the spinal column, and one vertebra higher because of the obliquity of the spinous process.

The patient is usually placed in the prone position with sandbags under the sternum and pelvis to leave the respiratory excursion of the diaphragm unimpaired in order to minimise back pressure on the epidural veins. The paravertebral muscles are injected with 1 : 100,000 of adrenalin in saline. A mid-line incision is made, the

muscles are separated from the spinous processes by cutting diathermy, and raised from the neural arches with a raspator after tendinous attachments have been cut through with curved scissors. Pressure from hot saline packs produces a dry field after any major vessels have been secured with coagulation. Self-retaining retractors are inserted and spines and interspinous ligaments are excised with bone-cutting

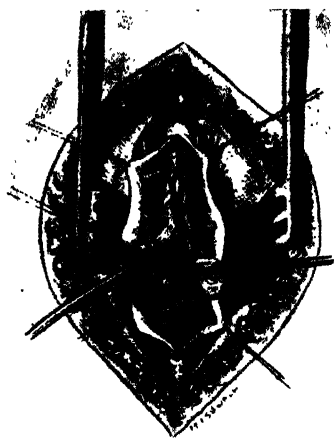


FIG. 484. — Removal of a meningioma indenting the dorsal cord.

forceps. The exposed raw edges of bone at the bases of the spines are now drilled through with a Hudson's burr to the level of the dura on each lamina, and finally the laminae are removed with bone-biting forceps, thus exposing the dura and the ligamenta subflava beneath the site of each lamina. The ligamenta subflava and epidural fat are now incised in the mid-line and the ligaments removed. The dura is then inspected for evidence of the tumour, usually shown by a slight bulge at the tumour site or a zone in which pulsation is absent. If any doubt exists as to the correct level, the dura is picked up on a fine hook and a small mid-line incision is made, and stay sutures of fine silk are inserted into the dural edge. A No. 3 fine rubber catheter is passed up or down inside the theca to estimate the site of obstruction. If the exposure is correct and no further bone removal is required, the dura is opened fully in the mid-line by extending the original small incision by slitting the dura with guarded scissors.

Several fine silk stay sutures are inserted into the dural edge to hold the dura back against the bone and prevent bleeding from the epidural veins. Strips of fine lintine cotton tissue are inserted against the muscle to absorb any minor oozing and to allow for suction on the cotton in order to keep the field dry and free from cerebrospinal fluid. The tumour is now removed in the manner previously described, following which the dura is sutured completely with fine silk, the muscles approximated with strong silk in layers, and the wound closed (fig. 484).

Additional Indications for Laminectomy include :

1. Traumatic. These are considered on p. 411.
2. Inflammatory paraplegia complicating Pott's disease (p. 238).
3. Division of posterior roots to relieve chronic pain resulting from (a) malignant metastasis in lymph nodes or post-irradiation fibrosis in the high cervical region following malignant disease of the tongue and jaw; (b) from post-herpetic neuralgia following herpes zoster in the thorax and abdomen. Owing to the sensory overlap of adjacent spinal segments, more than one nerve root must always be divided. To relieve pain in three thoracic root areas, five must be cut to include those above and below the affected zone. This renders the operation of posterior rhizotomy inapplicable to the relief of pain in the arms or legs, as extensive denervation will abolish position sense and thus result in a severe ataxia.
4. Cordotomy. Division of the spinothalamic tracts for the relief of pain due to such conditions as an irremovable new-growth or arthritis, *vide infra*.
5. The tremor of Parkinson's disease is relieved by division of the whole of the lateral tract at the second cervical segment on the side affected (Leslie Oliver). Most surgeons now prefer stereotaxic operations on the basal ganglia (p. 401).
6. Division of the anterior roots of C.2, 3 and 4, to paralyse the posterior rotator muscles in cases of spasmodic torticollis, this operation being combined with division of the contralateral spinal accessory nerve in the neck to paralyse the opposite sternomastoid.

SPINOTHALAMIC TRACTOTOMY

Intraspinal division of the spinothalamic tracts will produce loss of pain sensation with preservation of touch on the opposite side of the body. This operation can be employed unilaterally or bilaterally for the relief of chronic long-lasting pain resulting from arthritis, spondylitis, or intermittent claudication, and also be em-

William Henry Hudson, 1862-1917. Surgeon, Atlanta, Georgia.

James Parkinson, 1755-1824. English general practitioner who practised in Shoreditch, London.

Leslie Oliver, Contemporary. Neurosurgeon, Royal Northern and Charing Cross Hospitals, London.

ployed to relieve severe pain from incurable new-growths, but only if the general condition of the patient is good and survival is likely to be prolonged. In the terminal stages of cancer, however, cordotomy carries a higher risk of mortality from bronchopneumonia.

If pain is unilateral and not of visceral origin, the tract on the opposite side of the body only needs to be divided. For visceral pain bilateral divisions are required. If bilateral sections are to be made, they must be placed at levels two segments apart in the cord in order to avoid the risk of transverse degeneration and paraplegia.

In choosing the site of operation, allowance is made for the oblique crossing of the spinothalamic tracts. In the dorsal region the incision must be three segments above the level of the pain; in the cervical region, two segments. In the dorsal region the tract lies between the dentate ligaments and anterior roots, but fibres from the lower limbs may be displaced in front of the anterior root. Pelvic fibres will lie deep near the anterior horn. In the cervical region, recently entered arm fibres lie 2 mm. behind the dentate ligament. Section may be carried out as high as the fourth cervical segment.

The cord is exposed at the required level, the dentate ligament is identified, and divided and grasped with mosquito forceps, which are used to rotate the cord and thus expose the anterolateral aspect to view. An incision 4 mm. deep is now made from the line of the dentate ligament to the anterior root and carried into the anterior horn and beyond, if pelvic fibres are to be divided.

PROLAPSE OF INTERVERTEBRAL DISC

Intervertebral discs are interposed between the vertebral bodies, and serve not only as shock absorbers for the column but also provide the normal mobility between the adjacent vertebrae. Each disc consists of a soft central portion of spongy material, the nucleus pulposus, containing a remnant of the notochord, which is surrounded by a tough fibrous ring, the annulus fibrosus, which is attached to the adjacent vertebral bodies, the whole being enclosed between fibrocartilaginous plates above and below.

During normal flexion of the spine the disc is deformed and the annulus fibrosus and nucleus bulges backwards slightly into the neural canal.

Intervertebral disc protrusion is produced by the effect of flexion-forces acting upon the most mobile portions of the spine. A sudden strain with the spine in an unguarded position will rupture the tough annulus, allowing portions of the torn annulus and soft nucleus to escape into the spinal canal and form either a central protrusion in the midline under the posterior common ligament of the vertebrae, or a lateral protrusion at the side of the posterior common ligament adjacent to the intervertebral foramen (fig. 485).

In 80 per cent. of cases the protrusion is traumatic in origin and there is either a history of sudden severe strain, or the patient's occupation is one in which flexion strain must be resisted, such as a packer, fireman, porter, etc. The condition is therefore more common in males.

In 20 per cent. of cases the condition is degenerative in origin. There is no history of injury. A small portion of the nucleus pulposus herniates through a weak area in the annulus without tearing that structure.

Since the mechanism demands the combination of stress and mobility, protrusions are most common in the most mobile portions of the spine which are subject to the greatest stress, hence approximately :

19 per cent. occur in the cervical region at the mobile C.5/6 and C.6/7 levels.

1 to 2 per cent. occur in the immobile dorsal spine.

80 per cent. occur in the lumbar regions, particularly at the mobile L.4/5 and L.5/S1. levels.

Escape of disc material leads to :

1. The narrowing of the intervertebral joint space visible in X-ray in 50 per cent. of cases.

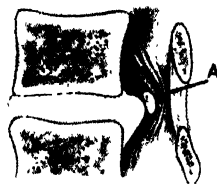


FIG. 485.—Intervertebral disc protrusion passing towards the intervertebral foramen, the posterior margin of which at A is constituted by the anterior edge of the subflavian ligament.

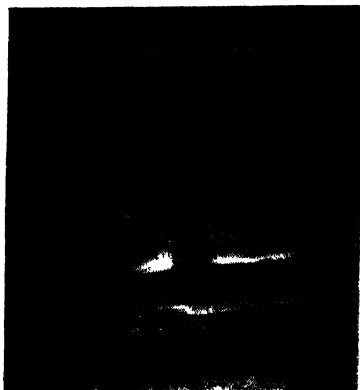


FIG. 486.—Schmorl's node in a lumbar vertebra (see text). Note also the narrowed disc space.

2. Slackening of the anterior common ligament of the vertebræ producing abnormal mobility between the vertebræ with local joint pain, and ultimately the development of intervertebral arthritis. Traction osteophytes form on the anterior aspect of the vertebræ and are visible in X-ray. Compensatory thickening of the ligamenta flava occurs in an attempt to check the abnormal mobility.

Massive protrusions may occur as a result of major cervical injuries (p. 423). Alternatively, when the spinal ligaments are softened at the end of pregnancy the strain of labour may force out a massive protrusion in the lumbar region, giving rise to one form of obstetric paralysis.

A *Schmorl's Node* (fig. 486) is a radiological manifestation of an extrusion of the nucleus pulposus into the body of a vertebra. In itself, it has no clinical significance. As time goes by, the new bone activity which surrounds it condenses into a shell.

Lumbar Disc Protrusion.—In the lumbar region the roots of the cauda equina run obliquely over a number of intervertebral joint spaces, hence a lateral protrusion may press on two roots. The nerve root that issues from the corresponding intervertebral foramen may be compressed against the lateral margin of the disc, whilst the nerve root going to the intervertebral foramen next below is caught against its inner margin.



FIG. 487.—Anteroposterior myelogram showing characteristic lateral filling defect produced by lumbar-disc protrusion.



FIG. 488.—Lumbar myelogram, lateral view, showing characteristic anterior pyramidal defect crossing to the joint space produced by disc protrusion. Myelography is not completely reliable as it fails to detect the small lesion.

Clinical Features.—In all cases there is an initial period of low back pain resulting from injury to the disc. Later the pain will radiate to the leg when nerve roots become compressed. There is then root pain, accentuated by coughing, in the distribution of the affected nerve, paræsthesiæ and pins and needles in the peripheral portion of that area, cramps, and tenderness in muscles supplied by the nerve, and a variable degree of sensory loss and motor weakness. Since the commonest roots to be involved are the first sacral and fifth lumbar, pain is usually in the back and side of the leg radiating to the sole of the foot and the big toe, and is called sciatica. Sensory loss is found on the sole and side of the foot and the ankle-jerk is lost. But in higher disc lesions the pain may be referred to the front of the thigh or leg.

<i>Root Involved</i>	<i>Pain and Sensory Loss</i>	<i>Motor Weakness</i>	<i>Reflex Change</i>	
1st sacral root	Back of leg. Sole and side of foot.	Gastrocnemius, weak plantar flexion.	Absent A.-J.	<div style="display: flex; align-items: center;"> <div style="font-size: 3em; margin-right: 5px;">}</div> <div> Often combined in L.4/5 disc. Often combined in L.5/S.1 disc. </div> </div>
5th lumbar	Back of thigh. Most of lateral aspect of leg. Dorsum of foot to big toe.	Anterior tibials. Weak dorsiflexion.	Nil.	
4th "	Side of thigh. Front of inner aspect of leg.	Quadriceps and anterior tibial, weak dorsiflexion and extension of knee.	Diminished K.-J.	
3rd "	Front of lower thigh.	Quadriceps.	Diminished K.-J.	
2nd "	Front of mid-thigh.	Quadriceps.	Diminished K.-J.	
1st "	Groin.	Nil.	Nil.	

In an endeavour to reduce pressure on the nerve root, the patient adopts a position of scoliosis and walks with a limping gait. Attempts to touch the toes or at straight-leg raising, or neck flexion with the legs raised, increases pain by pulling the nerve root against the protrusion.

Physical Signs.—Pain is produced by friction of the nerve against the protrusion. Small protrusions only compress the nerve root slightly and the nerve is free to rub against the protrusion. *Small protrusions* thus cause very severe pain because there is maximum friction without much loss of conduction in the nerve. Since there is little impairment of conduction, physical signs are slight and limited to a small patch of sensory loss in the periphery of the skin area involved where long sensory fibres have been affected. *Larger protrusions* cause less pain because they lock the root more firmly so that there is less friction, while the conduction of the nerve is diminished. Since conduction is diminished the physical signs are more marked and sensory loss is extensive owing to involvement of short sensory fibres. *Massive protrusions* completely fix the root and cause no pain whatsoever, but produce the maximum sensory loss and motor paralysis (obstetric paralysis).

Treatment consists of confinement to bed until symptoms abate, which is usually a matter of two to four weeks. A plaster jacket is applied for two to three months, after which a spinal brace is worn for a further period. The majority of cases are cured by this routine. Operation is indicated if symptoms persist, if severe pain recurs, or if motor weakness is developing.

The disc is removed by hemilaminectomy, removing the adjacent margins of two laminae and the subflavian ligament which obscures the view of the nerve root and disc protrusion until it has been excised (fig. 489). After removal of the subflavian ligament, the swollen root is seen overlying the protrusion by which it is displaced inwards. Loose disc material is then grasped with forceps and extracted.

A full laminectomy is contraindicated as it encourages further weakness of the spine. Even after satisfactory removal of a prolapsed disc, symptoms or physical signs are not always alleviated. Two conditions may persist. Prolonged compression of the nerve may have resulted in interstitial neuritis



FIG. 489.—A typical portion of protruded disc removed at operation.

producing sensory loss or motor weakness, or the intervertebral joint may be unstable or become affected by osteoarthritis. Intervertebral arthritis or instability is to be suspected when pain is not relieved by rest, but persists at night and wakes the patient when he turns over in bed. If this condition is present, spinal fusion should be performed when the disc is removed so as to afford complete rest to the affected segment of the spine. A paraspinal inlay of acrylic resin may be placed over the back of the intact laminae on the side opposite to that on which the hemi-laminectomy has been performed.

Cervical disc protrusions are relatively uncommon. In many cases a diagnosis of cervical disc lesion is made in patients who are really suffering from referred pain arising from a cervical strain which has affected the interspinous ligaments, or lateral articulations following a flexion strain, or who are suffering from cervical spondylosis. As true cervical disc protrusions are uncommon, this diagnosis must only be accepted when there is definite evidence of compression of the spinal cord or nerve roots, and when the presence of a disc protrusion has been confirmed by demonstrating a lateral filling defect at myelography. In the cervical region the nerve roots run transversely and come into relation with one intervertebral disc only. Each nerve issues above the level of the corresponding vertebra.

Lateral protrusions press on the corresponding nerve and half the spinal cord, producing cervical rhizalgia with half-cord compression, a Brown-Séquard syndrome. They are never situated sufficiently far laterally to press upon the nerve root alone. Symptoms of brachial rhizalgia without cord compression are produced by intra-foraminal osteophytes occurring in association with cervical spondylosis, which may, unlike a disc, affect multiple nerve roots. Each spinal root emerges above the corresponding vertebra, hence C.5/6 protrusion compresses the C.6 nerve root and half the cord (see Table below).

Mid-line protrusions compress the anterior part of the cord and affect the anterior spinal artery and the anterior spinal veins. Compression of the anterior spinal artery may affect the pyramidal and spinothalamic tracts and anterior horns. Compression of the veins which ascend on the front face of the cord produces stasis in the anterior horns below the level of the lesion.

The symptomatology varies on repeated examination as pressure on the vessels varies, and may simulate syringomyelia, disseminated sclerosis or primary lateral sclerosis. In time secondary atrophic changes produce an atrophy of the cord substance with permanent spastic paresis.

Root	Pain, Sensory Loss	Sensory Loss	Muscle Weakness and Reflex Change
C.6	Trapezius. Shoulder tip, outer border of upper arm. Dorsum of forearm.	Lateral border of upper arm. Dorsum of forearm and thumb.	Weakness of biceps, diminished biceps, and supinator jerk.
C.7	Trapezius. Shoulder tip, back of upper arm, and dorsum of forearm.	Dorsum of forearm and all fingers except thumb.	Weakness of trapezius and extensors of the fingers. Diminished triceps jerk.

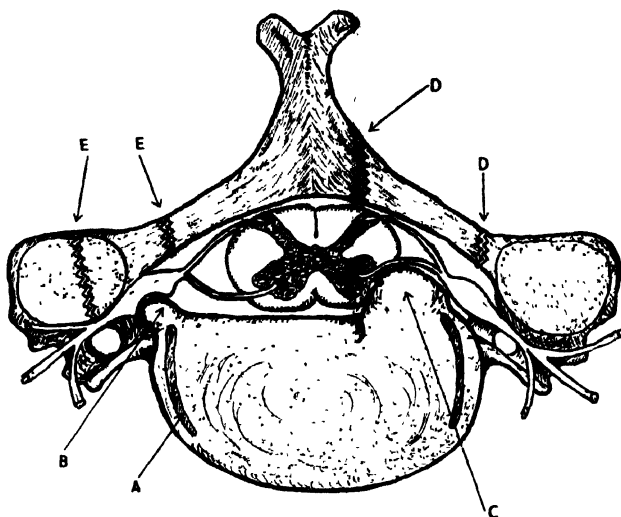
Acute protrusions occurring in cervical injuries may produce symptoms of partial or complete cord lesion without bone displacement.

Treatment.—Lateral protrusions are exposed by hemilaminectomy and removed by an extradural approach. Midline protrusions are treated by a full laminectomy of sufficient length to permit one to open the dura and divide the dentate ligaments for several segments above and below the protrusion in order to allow the cord to fall back and thus relieve pressure on the arteries and veins. Attempts to remove a mid-line protrusion may damage the anterior spinal artery and cause permanent paralysis.

Cervical Spondylosis is a degenerative condition characterised by (1) degeneration of the intervertebral discs with the formation of bony ridges running across the anterior surface of the neural canal and (2) the formation of osteophytes from the neurocentral joints of Luschka which project backwards into the intervertebral foramen (fig. 490). The condition may be quite symptomless or may cause neurological symptoms.

(a) Sudden strains inflicted on the affected joints as, for example, in the violent forward lurch sustained in road traffic accident, so called 'whip-lash' injuries, may give rise to referred pain in the occipital or post-auricular regions and in

FIG. 490. — Diagrammatic cross-section of the cervical spine and cord showing the position of (A) the neurocentral joint of Luschka, (B) an intraforaminal osteophyte developing from that joint compressing the nerve root within the foramen, (C) a lateral disc protrusion compressing the cord and nerve root. Projection into the intervertebral foramen is prevented by the position of the joint of Luschka. (D-D) the section of bone removed in hemi-laminectomy giving access to a lateral disc protrusion. (E-E) The section of bone removed in hemifacetectomy giving access to an intraforaminal osteophyte. (G. B. Northcroft, F.R.C.S., London.)



the upper portion of the trapezius, and between the shoulder blades. Persistent occipital and posterior auricular pain can be relieved by excision of the corresponding nerves. Since referred pain and muscle spasm in the lower cervical region is the result of movement in the painful joints, these symptoms are often relieved by wearing a collar, but if they become chronic they may be permanently relieved by the insertion of a paraspinal inlay to immobilise the affected joints. (b) Bony ridges on the anterior surface of the cord may compress arteries and veins with consequent neurological symptoms and spasticity and ultimate cord atrophy simulating a mid-line disc lesion. This should be treated by division of the dentate ligaments. (c) Intraforaminal compression by osteophytes may affect multiple nerve roots. The nerve roots may be decompressed by excision of the back of the intervertebral foramina by hemifacetectomy.

LUMBAR PUNCTURE

This procedure is adopted for the following reasons :

1. **Diagnostic**, e.g. meningitis, Wassermann reaction or gold test for syphilis; and to disclose intradural hæmorrhage. Also for the injection of pantopaque, or to assess the protein content, if a tumour is suspected. Manometric readings are essential to assess the intradural pressure (p. 417).

2. **Therapeutic**, in order to introduce some drug, e.g. penicillin for meningitis, or, the relief of pain.

Relief of Pain.—The intrathecal injection of phenol has now largely replaced the use of absolute alcohol, which was far more likely to produce undesirable sequelæ such as paralysis or retention of urine. Intrathecal alcohol injections are now given chiefly to destroy the severed lower portion of the spinal cord in complete and irrecoverable cord injuries, to obliterate flexor spasms and rigidity and facilitate rehabilitation (p. 410). The pain-conducting fibres passing from the posterior root ganglia to enter the spinal cord are either non-myelinated, or possess a much thinner myelin sheath than fibres conducting other forms of sensation. Since phenol acts upon the non-myelinated fibres, it will relieve certain forms of pain without affecting normal sensation or motor power. Slow pain travelling at a speed of approximately six metres a second is conducted by the finest non-medullated fibres of less than 10μ . Fast pain travelling at approximately sixty metres a second is conducted in larger medullated fibres of over 10μ . Since phenol affects the non-medullated fibres, it therefore abolishes the slow pain and is most effective in relieving the deep aching pain of cancer and secondary deposits, but is far less effective in the sharp pain of neuritic origin.

The injection of intrathecal phenol has now superseded the use of intrathecal alcohol which often produced rectal and urinary incontinence. A solution of 1 per cent. phenol in glycerine has proved to be unreliable and liable to produce incontinence, and a solution of 7.5 per cent. phenol in myodil is far safer. It appears that the phenol is in some way fixed in this solution. When used with radio-opaque myodil the phenol can be guided under X-ray control to the appropriate level. The myodil solution is heavy and therefore drops to a low level in the theca. To relieve pain from the bladder and pelvis the injection is made with the patient sitting up; at other levels, the patient lies on the affected side on a tilting X-ray couch, between 1 and 3 ml. of 7.5 per cent. phenol in myodil are injected by lumbar puncture, and the radio-opaque substance is then guided to the affected level by tilting the X-ray couch and checking the level of the myodil by observation on a fluorescent screen.

3. **Anæsthesia.**—Stovaine, Novocaine, Spinocaine, and Percaine; each has its individual supporters.

Spinocaine is composed of Novocaine (100 mg. in each ml.) and strychnine. The dose varies from 1 ml. for children under six years of age to 3 ml. for adults. A pressor substance, such as ephedrine gr. $\frac{1}{2}$ to $\frac{3}{4}$ (30 to 45 mg.) is usually given intramuscularly in order to maintain the blood pressure.

Percaine is used in a 1 : 1,500 solution in 0.5 per cent. saline. The specific gravity of this solution is 1.003, which is lighter than cerebrospinal fluid (sp. gr. 1.006). The average dose is 14 ml., which gives complete abdominal anæsthesia, but 10 ml. is usually sufficient for operations below the umbilicus. After injection the patient lies on his face for five minutes so that the posterior roots are chiefly affected. He is then turned on his back, and slight tilting downwards of the head of the table will help to fix the Percaine caudalwards and counteract any fall of blood pressure.

Catastrophes, such as incontinence of urine or damage to spinal nerves, occasionally occur. These calamities are possibly due to an invisible crack in a phial, which, if submerged in spirit or other antiseptic, may allow some of the fluid to percolate into the interior. *Phials should always be submerged in coloured fluid*, and any phial which contains coloured contents is rejected.

Some premedication, such as omnopon and scopolamine, is safe and humane, but if the intercostal muscles are paralysed, depression of the respiratory centre is dangerous.

Technique.—At birth the cord extends to the third lumbar vertebra. In the adult it terminates opposite the lower border of the body of the first lumbar vertebra, below this level the dural sac can be safely punctured. A space of 2.5 mm. in width exists between the caudal equina nerve roots on either side and hence, if the lumbar puncture needle is kept strictly in the mid-line, it will avoid contact with the roots. The patient lies on his side with the spine well flexed in order to open up the spaces between the neural arches. Scrupulous asepsis is required. The skin must be fully prepared and isolated with sterile towels, gloves and gown being worn in order

to perform the puncture. A line connecting the highest parts of the iliac crests passes over the fourth lumbar spine (fig. 491). The interspace above or below this spine is selected, and this area is infiltrated deeply by injecting local anæsthetic into the interspinous ligaments. The depth to which the needle will have to pass varies according to the build of the patient, from a few cm. in a small child to 10 cm. in a fat adult. In the case of some nervous children or patients afflicted with tetanus, general anæsthetic is necessary. The needle used should be of medium bore (about 18 to 20 gauge) as the aperture resulting from puncture with a wide needle will allow continuing escape of cerebrospinal fluid from the dura, with consequent persistent headache, produced by intracranial hypotension. The bevel of the needle must be short. As the puncture is commenced, the position of the needle should be checked, making sure that it is introduced at right angles to the skin and is not sloping to one side. This is best judged by looking along the skin of the patient's back; it should also be slanted slightly towards the patient's head so that it will enter obliquely beneath the lumbar spines. As the needle and stylet are introduced, the resistance of the tough ligamentum flavum is encountered, a little extra pressure perforates this structure, and on withdrawal of the stylet cerebrospinal fluid should flow from the needle. If only a few drops of blood escape, in all probability the point of the needle is lying in the spinal canal outside the dural sheath and the blood is escaping from the spinal veins, in which case the needle should be advanced slightly further. When the theca is entered, a manometer is attached to the needle in order to estimate the cerebrospinal fluid pressure, the normal being 120 mm. of water in the recumbent position. In cases of raised pressure only a small quantity of fluid must be removed for investigation, and the pressure should not be reduced, even as low as the normal 120 mm., owing to the risk of causing mid-brain pressure cone, or a medullary cone in the foramen magnum, with fatal consequence.

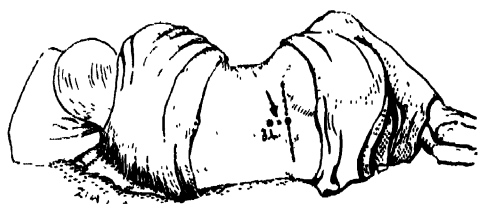


FIG. 491.—Lumbar puncture. (The site of puncture is usually between the third and fourth lumbar spines.)

SACRAL PUNCTURE (EPIDURAL)

The sacral epidural space may be entered through the large triangular opening on the dorsum of the bone, and sacral anæsthesia obtained by the injection into the space of 20 ml. of 1 per cent. Novocaine. Operations on the lower bowel and anal margin can then be performed painlessly, and some obstetricians practise this method in order to relieve the pains of child-birth, an additional advantage being that the infant is unaffected.

To perform sacral puncture the median crest of the sacrum is palpated until the depression caused by the sacral opening is recognised. A lumbar-puncture needle is introduced directly forwards until the ligaments covering the opening are pierced, after which the direction of the needle is changed so that it passes vertically upwards within the sacral canal (fig. 492). In order to ensure that the point of the needle is within the epidural space, it is wise to inject a few millilitres of normal saline; if the needle has not penetrated the canal, infiltration of the subcutaneous tissues will be evident, in which case it is withdrawn and a fresh attempt is made.

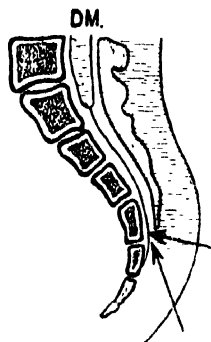


FIG. 492.—Indicating direction of needle.

CHAPTER 19

NERVES

GEOFFREY KNIGHT

CRANIAL NERVES

THE Olfactory Nerve may be injured by fractures of the cribriform plate, resulting in partial loss of smell (hyposmia) or anosmia on the corresponding side. Olfactory filaments may be ruptured as a result of anterior displacement following blows on the back of the head (*contre-coup*, p. 357).

The **Optic Nerve** may be damaged by fractures involving the optic foramen or by contusion against the margin of the foramen. Involvement by tumours or aneurysms is not uncommon. Primary optic atrophy with partial or complete blindness results in the affected eye, but contraction of the pupil will occur if the opposite retina is stimulated. The optic nerve is an outgrowth of the brain and gliomas arise in its substance particularly in young children.

The **Third Nerve** is involved by tumours, trauma, or aneurysm in the skull, sphenoidal fissure, or orbit. Pressure on the nerve above the tentorium occurs in the early stages of midbrain pressure cone formation causing a dilated pupil. Partial lesions may only produce a dilated pupil, but in complete lesions the following features are noted.

(a) Ptosis of the upper eyelid, owing to paralysis of the levator palpebræ superioris.

(b) Proptosis, owing to paralysis of the majority of the ocular muscles, which normally exercise traction on the eyeball; this will be increased if an intraorbital tumour is also present.

(c) Mydriasis, as the sympathetic fibres are unopposed, and cause unhampered dilatation of the pupil.

(d) Loss of accommodation, owing to paralysis of the ciliary muscle.

(e) Diplopia and external strabismus, with a slight downward inclination of the eyeball due to unopposed action of the external rectus and superior oblique muscles. Owing to their proximity, other nerves passing to the orbit are often affected.

The **Fourth Nerve** supplies the superior oblique muscle, and is rarely involved alone. Diplopia and deficient movement of the eye in a downward and outward direction may be noticed.

The **Fifth Nerve** or its branches are sometimes injured, and sensory disturbances follow (p. 185).

Trigeminal neuralgia or *tic douloureux* occurs most commonly in females. The cause is unknown but may be related to infection of the nerve by the virus of herpes simplex. Pain commences in the third or second

division and extends in time to the adjacent division, the ophthalmic division usually escaping. The pain is characteristically intermittent and extremely severe, but of brief duration, often described as being like red-hot needles searing the flesh. Spasms of pain are precipitated by external stimuli such as cold draughts, brushing the teeth, washing, speaking, eating, or drinking hot or cold substances. The patient often indicates 'trigger zones', stimulation of which produces an attack. Pain occurs in bouts of several weeks' duration with long periods of remission. The periods of remission, however, gradually become shorter and the attacks last longer, until eventually the patient is in almost continuous pain and may become suicidal. Pain may also be experienced in the trigeminal areas as a result of partial lesions either dental, nasal, or ocular, or from involvement of the trigeminal pathways, in disseminated sclerosis, eighth nerve tumours, or aneurysms but in such cases the pain is more continuous and occurs in association with physical signs of the causative lesion. Trigeminal neuralgia is to be distinguished by the fact that it is an intermittent pain of great severity which occurs in the absence of physical signs.

Treatment consists of a thorough search for any source of symptomatic pain, and the use of analgesics. Tegretal is effective in controlling pain in certain patients. If the condition persists, injection or operation is indicated.

Alcohol injection is made into the Gasserian ganglion. The needle is inserted 1 cm. below the zygomatic notch and passed upwards, backwards, and inwards to make contact with the third division at a depth of 5 to 6 cm. The needle is then advanced into the nerve, causing severe pain; a drop of local anæsthetic is immediately injected. Before alcohol is injected, the syringe is aspirated to make sure that no cerebrospinal fluid escapes. If the needle is well into the nerve, considerable resistance is offered to injection of local anæsthetic. The anæsthesia produced indicates the position of the needle point, which is now advanced into the ganglion, and further anæsthetic is injected until the requisite area of the face is rendered numb. 4 or 5 millilitres of absolute alcohol are now injected. Relief from pain may last from six months to two years, following which sensation usually returns and pain with it. Repeated injections are then necessary and are made with increasing difficulty owing to fibrosis in, and around, the nerve. If further injections are required in a young subject, it is better to proceed to the safe operation of sensory root section in the middle fossa.

Phenol Injection.—Recent experiments indicate that the injection of 7·5 per cent. phenol in myodil, introduced by injection into the ganglia under X-ray control, will produce relief of pain without loss of sensation.

Division of the Sensory Root.—Sensory root section is greatly facilitated by operation in the sitting-up position. This reduces pressure in the venous sinuses, minimises bleeding, and facilitates exposure, since any effused blood collects in the floor of the skull and is easily sucked away without obstructing the view.

The temporal muscle is exposed by a curved-hook incision and divided. An opening is made at the base of the skull in order to minimise the elevation of the dura. The dura mater is separated from the floor of the middle fossa until the middle meningeal artery is seen emerging from the foramen spinosum. A blunt hook is inserted into the foramen, the artery is coagulated with diathermy, and then divided with



FIG. 493.—Fractional section of the lower and outer two-thirds of the sensory root at the apex of the petrous. The motor root is visible behind the cut portion of the sensory root.

cutting current. The mandibular nerve is then identified as it passes back from the foramen ovale to the cave of Meckel. The dural sheath is now stripped up by blunt dissection with the point of the sucker, opening Meckel's cave in order to expose first the ganglion and then the sensory root which lies in a sheath of arachnoid farther backward on the apex of the petrous bone. A retractor is inserted into Meckel's cave and the sensory root is exposed and the sheath around it is opened. Cerebrospinal fluid escapes in considerable volume and is removed by suction. The sensory root is then distinguished by its loose texture and parallel fibres. The root is now divided on a blunt hook, avoiding the motor root which lies deep to the sensory fibres and crosses behind them at an angle of some 60 degrees. Fractional division of the lower and outer two-thirds of the root preserves sensation in the first division, the fibres of which lie in the upper and inner one-third of the root (fig. 493).

Temporary facial paralysis rarely follows operation as a result of traction on the great superficial petrosal nerve being transmitted to the geniculate ganglion of the facial nerve. This can be avoided by keeping the third division of the nerve and the ganglion between any dissection and the great superficial petrosal which passes under the ganglion.

Neuropathic keratitis also results from damage to the great superficial petrosal, which is the secreto-motor nerve to the lacrimal gland. In all cases tear secretion is reduced to 50 per cent. of normal for the first six weeks, the eye must therefore be kept covered for this length of time. The operative mortality is less than 0.5 per cent. The operation has been successfully performed in patients of up to eighty-four years of age.

Sjöqvist intramedullary tractotomy.—The descending root to the fifth nerve conveying pain fibres is divided by an incision 4 mm. wide and 4 mm. deep opposite the lowest vagal fibres in the medulla. This produces analgesia without loss of touch in the face, but is less satisfactory and more dangerous to the patient than sensory root section (fig. 494).



FIG. 494.—Sjöqvist intramedullary tractotomy. The left cerebellar tonsil is lifted up to expose the vagal fibres. The site of incision is shown by the arrow.

The **Sixth Nerve** is slender and has a long intracranial course. It may be affected by fractures of the base of the skull or displacement of intracranial structures. It may be involved in association with other ocular motor nerves by lesions in the cavernous sinus, sphenoid fissure, or orbit. The external rectus muscle is paralysed, and internal strabismus results.

The **Seventh Nerve** is involved by a variety of causes.

1. **Intracranial.**—Lesions within the brain are supranuclear, nuclear, or infranuclear. Supranuclear lesions involve only the lower half of the face, as the occipito-frontalis and orbicularis palpebrarum muscles enjoy bilateral innervation. In nuclear lesions the whole face and the sixth nerve on the same side are affected and also the opposite arm and leg, as the motor decussation takes place at a lower level. An infranuclear lesion occasionally results from pressure of a tumour, e.g. of the cerebello-pontine angle, in which case involvement of the auditory nerve is also evident.

2. **Cranial.**—The intraosseous portion of the facial nerve may be affected by fractures of the base (fig. 495) and middle ear disease. Following fracture

paralysis may appear immediately as a result of direct injury to the nerve or within hours or days from hæmorrhage within the nerve sheath, in which latter case recovery is possible. Paralysis occurring after some weeks is produced by the pressure of callus and is unlikely to recover. Facial paralysis is a complication of middle ear disease and may follow injury at operation on the mastoid antrum. Compression within the aqueduct of Fallopius occasionally follows chronic inflammation.

3. *Extracranial*.—The facial nerve or its branches may be injured outside the skull. The nerve is commonly involved by Bell's palsy, which is due to herpetic neuritis and follows exposure to cold or draught. Swelling within the sheath of the nerve extends into the stylomastoid foramen, and the nerve is compressed within the bony canal. Absorption of exudate usually occurs before the pressure has damaged the nerve permanently, but in about 3 per cent. of cases complete paralysis remains, and in 5 to 10 per cent. some degree of paralysis persists. Tetanus arising from a wound in the distribution of the facial nerve sometimes causes paralysis, the cause being similar to that described above. Malignant tumours of the parotid gland involve the facial nerve, and paralysis is an important diagnostic sign distinguishing simple from malignant tumours.

Branches of the facial nerve are injured either accidentally, e.g. by broken wind-screens, or by ill-placed operation incisions. For operating on the parotid, the main trunk of the nerve must first be located and then branches followed forward (p. 506).

The paralysed face is flat and expressionless. The eye cannot be closed, and attempts to do so result in the eyeball being turned upwards and outwards (fig. 495). Corneal ulceration may follow from exposure. Epiphora occurs owing to drooping of the lower eyelid. Whistling is impossible, as the cheek merely flaps, and food collects between the gums and cheek. Treatment is directed to the cause. To promote recovery the angle of the mouth is supported by a malleable rod covered with rubber tubing, which is bent like an 'S' so that the upper curve will hook around the ear and the lower will elevate the angle of the mouth. Small strips of adhesive strapping applied under tension also form a very convenient method of preventing over-stretching of the facial muscles. If considered advisable, an intra-oral splint can be fashioned from plastic material. Electrical treatment and massage are prescribed, and during recovery the patient should practise facial movements with the aid of a mirror. Hypoglossal anastomosis may be considered in otherwise hopeless cases. Repeated facial tic with spasmodic contracture of the facial muscles may be relieved by fractional division of the



FIG. 495.—Right-sided facial paralysis following fracture through the middle fossa.

branches of the facial nerves which supply the affected muscles, these branches being exposed through a curved incision anterior to the lobe of the ear.

In cases of Bell's palsy early decompression of the nerve by removal of part of the mastoid process reduces the risk of paralysis (Duel).

The **Eighth Nerve** may be involved in fractures of the middle fossa, or compressed by a tumour, e.g. of the auditory nerve sheath, causing unilateral deafness (p. 463). Vestibular functions are sometimes impaired.

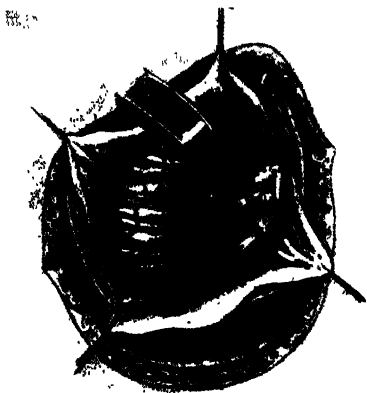


FIG. 496. — Unilateral cerebellar exposure. The cerebellum is drawn inwards and exposes the ninth, tenth, and eleventh nerves at the jugular foramen. Above this the eighth nerve has been divided as it lies behind the seventh at the internal auditory meatus.

Ménière's disease (p. 463) is a paroxysmal disturbance associated with giddiness, deafness, and vomiting. If medical measures fail, the vestibular portion of the nerve is exposed through the posterior fossa and divided as it enters the internal auditory meatus (fig. 496).

The **Ninth Nerve** is occasionally injured by a fractured base. Some dysphagia may occur from paresis of the constrictor muscles.

Glosso-pharyngeal neuralgia is characterised by severe explosions of pain either in the region of the tonsil or deeply in the ear. The 'trigger' zone is in the tonsillar area, and the diagnosis is clinched by the fact that cocaineisation of the zone temporarily relieves the condition. In genuine cases the nerve must be divided. It is approached either through the posterior fossa and severed as it enters the jugular foramen, or as it crosses the tonsillar fossa after tonsillectomy.

The **Tenth Nerve** may be damaged in association with a fractured base, or crushed by a ligature which includes it as well as the internal jugular vein or common carotid artery. Injury to one vagus nerve is unlikely to cause more than a temporary tachycardia. In doubtful cases examination of the laryngeal muscles will reveal recurrent laryngeal nerve involvement.

The recurrent laryngeal nerve may be damaged in operations on the thyroid gland (p. 637) or ductus-arteriosus by traction or post-operative extravasation. Complete paralysis may follow division or inclusion by a ligature at operation, infiltration by neoplasm of the thyroid gland or secondary lymph nodes from carcinoma of the lung, and on the left side by pressure of an aneurysm of the aortic arch. Partial involvement affects the abduction fibres, which are more susceptible than those which supply the adductor muscles, and thus if partial involvement is bilateral, stridor results, and tracheostomy may be required. Complete involvement results in paralysis of both abductors and adductors, and consequent paralysis of the corresponding vocal cord in the half-way or 'cadaveric' position. The opposite vocal cord increases its range of mobility, so that it reaches across the midline and closes the glottis. The voice is adequate but somewhat monotonous.

Hysterical aphonia is emotional in origin and is distinguished from true

paralysis by the fact that, although the patient can only whisper, coughing is readily performed on request.

The **Eleventh Nerve** is rarely damaged by fractures but is more commonly injured during high cervical dissections, particularly in the removal of tuberculous lymph nodes which often entirely surround it. The nerve passes downwards and backwards at right angles to the centre of a line connecting the angle of the jaw and the mastoid process, and emerges from the posterior border of the sternomastoid muscle at the junction of the upper third and lower two-thirds; it then passes across the posterior triangle, and disappears under cover of the trapezius muscle. Division of the nerve in the anterior triangle usually produces complete paralysis of the sternomastoid muscle, but the motor supply contributed from the second and third cervical roots is sufficient in a third of the cases to prevent a complete paralysis of the sternomastoid. Division will result in only a partial paralysis of the trapezius as the trapezius receives a sufficient additional supply from the third and fourth cervical nerves. If injury occurs in the posterior triangle, the trapezius alone is affected. On inspection, drooping of the shoulder is seen, and wasting of the trapezius is obvious (fig. 497). Contraction of the sternomastoid muscle is tested by placing a hand under the patient's chin and requesting him to flex his head. Palpation detects the rigid band of muscle if contraction is normal. Injury of the nerves to the trapezius results in inability to continue elevation of the arm after it is abducted to a right angle by the deltoid muscle. If the branches to the muscle from the third and fourth cervical nerves are intact, about 20 degrees elevation from the right-angled position is possible.

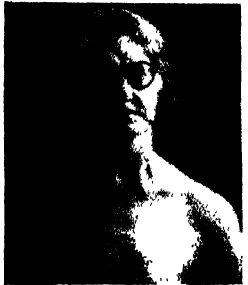


FIG. 497.—Drooping of the left shoulder and wasting of the trapezius muscle following division of the spinal accessory nerve. The swellings on forehead and face are soft fibromas—a common condition after middle age.



FIG. 498.—Hemiatrophy of the right side of the tongue following involvement of the hypoglossal nerve by syphilitic basal meningitis.

If division of the spinal accessory nerve is recognised at operation, primary suture should be performed. Secondary suture is not successful on account of retraction of the ends and difficulty in identifying them in scar tissue.

The **Twelfth Nerve** escapes in fractures of the base of the skull, as the anterior condyloid foramen is protected by a bony ridge which diverts a fissure towards the foramen magnum. It may be injured in submental operations, e.g. for removal of tuberculous lymph nodes. Hemiatrophy of the tongue occurs, the corresponding side of the tongue being shrivelled and wrinkled, the tongue being pushed towards the paralysed side on protrusion (fig. 498).

PERIPHERAL NERVES

Injuries of nerves are classified according to the extent of the damage to the nerve fibres and sheath, as neurapraxia, axonotmesis, and neurotmesis.

NEURAPRAXIA

is the equivalent of concussion. There is physiological paralysis of conduction in the intact nerve fibres as the result of stretching or distortion without any organic rupture. Neurapraxia is produced by minor stretch injuries, or by the concussion and vibratory effect of a high-velocity missile passing near a nerve without contacting it. Fibres remain intact within the intact sheath. It produces sensory loss, paræsthesiæ, and weakness of muscle groups lasting for days. During this time there is no reaction of degeneration in the muscles. There is no degeneration of the axons. When the power of conduction returns, all functions, motor and sensory, return together, sensation recovering in the whole limb within a period of hours. Recovery is complete.

Treatment consists of splinting the limb in a position of relaxation of the paralysed muscle groups until spontaneous recovery occurs.

AXONOTMESIS

consists of the intrathecal rupture of nerve fibres within an intact sheath. According to the cause, the nerve fibres are damaged to a variable degree (see below). Wallerian degeneration occurs in the distal portion of the broken axons, leaving an empty tubule. Intraneural fibrosis occurs at the sites of axonal rupture and minute intraneural hæmorrhages. Recovery takes place slowly by the proliferation and down-growth of axons into the distal tubules, the combined bulk of the proliferating axons and intraneural fibrosis producing a fusiform neuroma on the course of the nerve. There is some loss of nerve fibres owing to the blockage of the down-growing axons by the intraneural fibrosis, but since the relative position of axon and distal tubule is preserved by the intact sheath there is little maldistribution, hence the quality of regeneration is often good except in the case of progressive fibrosis. Recovery is delayed until the down-growing fibres reach their appropriate endings, and occurs first in the muscle groups nearest to the site of division and lastly in the peripheral skin areas, where the anæsthetic area begins to decrease steadily from the margin inwards. The length of time required for the recovery depends upon the level of the lesion. After an initial delay of approximately ten days, down-growing axons proceed distally at a rate of approximately 1 mm. a day, and on arrival at their endings there is a further delay of three weeks before the end organs become activated. Partial or complete intrathecal rupture of fibres may complicate a single stress occurring in association with fractures or dislocations, or excessive vigour during attempted reduction of such injuries; it also accompanies traction injuries at birth and may be produced by clutching for support when falling, as in brachial plexus lesions. It may follow contusion of a nerve with extensive hæmorrhage into its sheath or be the result of severe and acute compression by tourniquets, splints, or incorrect posture on the operating table.

Gradually increasing lesions are produced by progressive compression from crutches, splints, callus, and scar tissue or by repeated minor stretching of a nerve which becomes fixed near a mobile joint. The latter is seen when a nerve such as the external popliteal becomes fixed at the head of the fibula, or the ulnar nerve becomes fixed behind an arthritic elbow or stretched by cubitus valgus; in such cases movements of the joint pulling on the fixed nerve produce repeated minor ruptures at different levels, each of which is succeeded by a small degree of fibrosis, which gradually induces a progressive fibrosis and loss of function within the nerve.

Clinical Features.—Following a severe stretch, there is an initial picture of neurapraxia with widespread loss of sensation, tone, power, and reflex activity in the limb; this is followed by an incomplete recovery. The resolution of concussion will restore sensation and movement to certain areas of the limb to which the nerve fibres remain intact. The numbness and paralysis will persist in those areas to which the fibres have actually ruptured. Usually the total area affected is less than the known anatomical distribution of the nerve as a proportion of fibres have escaped.

Treatment of closed injuries of nerves consists in maintaining the nutrition of the limb and combating, by suitable means, the secondary pathological changes which would otherwise follow nerve injury, in order to maintain the muscles and joints in good working order pending the arrival of down-growing axons.

Secondary pathological changes accompanying nerve injury affect the skin, muscles, and joints. Impaired circulation consequent on disuse renders the part blue and cold. The skin becomes thin, the nails brittle, and so-called trophic changes occur as a consequence of minor unrecognised trauma. Those muscles which are paralysed and flaccid will be over-stretched by unopposed action of antagonist groups. Within three weeks the reaction of degeneration appears. The muscle fibres no longer respond to the rapid make-and-break of faradic stimulation owing to an increase in the duration of chronaxie which accompanies paralysis, but they will still respond to the slow make-and-break of galvanism. The polarity also changes so that A.C.C.¹ becomes greater than K.C.C.² If the muscle nutrition is not maintained by galvanic stimulation, muscle fibres degenerate and progressive fibrosis replaces inert fibres. Recovery is then impossible. Peri-articular adhesions eventually form around the immobile joints and fix them in a contracted position. To combat these effects the part must be protected by warm padding to prevent injury, and splinted in a position of relaxation of the paralysed groups. The electrical reactions of the muscles are recorded and muscle movement is maintained by regular galvanic stimulation of all paralysed groups. All joints are put through full passive movements daily to prevent contraction. In the case of the hands, joint movements and exercise of the paralysed muscles are best maintained by the use of elastic splints in which strands of catapult elastic are attached to leather thimbles, which are inserted over the finger-tips and mounted on wire frames

¹ A.C.C. = Anodal Closing Contraction.

² K.C.C. = Kathodal Closing Contraction.

in positions corresponding to the paralysed tendons, so as to provide an elastic counter-pull to replace the normal contraction of the paralysed muscles against which the opponents can act. This not only ensures full normal movement of all joints, but will also permit partial use of the hand during recovery.

Considerable encouragement must be given to the patient during the long period of time required for restoration of function. Progress is carefully checked by taking regular records of the electrical reactions and skin sensitivity. Electrical reactions change back to normal in the proximal groups as the downgrowth of fibres proceeds. These changes must be recorded, together with diagrams of the skin sensation, in order to determine whether normal recovery is taking place. Failure to recover, or regression after the initial recovery, is an indication for the local exploration of the injured site. It is occasionally necessary to deal with a perineural scar or remove an intraneural fibroma.

Movements produced by unaffected muscles must not mislead the observer. Thus, in a case of radial paralysis, the fingers can be extended by the interosseous and lumbrical muscles, provided that the hand is supported. Also vicarious movements may be performed by adjacent muscles, e.g. in the case of division of the median nerve, the adductors of the thumb, acting in conjunction with the extensor ossis metacarpi pollicis, can produce opposition of the thumb.

H. J. Seddon *et al.* have endeavoured to calculate the rates of nerve regeneration, basing their results on the following clinical data :

(1) Tinel's sign—the course of the nerve is lightly percussed with a patella hammer, from below upwards. A tingling sensation is experienced when the level of regeneration is reached.

(2) Measurement of the rate at which pain and touch sensibility return.

(3) Observation of the times at which the function of muscles returns at different levels from the injury.

It appears that regeneration occurs initially at about 2 mm. a day, but the rate diminishes as time passes, so that after about three months it has slowed down to about 1 mm. a day. (Other factors which influence the results of suture are discussed on p. 439.)

Compression.—The first symptoms usually consist of paræsthesiæ, numbness, and tingling along the distribution of the nerve, associated with neuralgic pain. Complete paralysis and wasting of muscles are uncommon unless the initial compression has been prolonged and severe, but when the compressing cause is progressive, paralysis can occur later and may then necessitate operative measures.

Treatment is by removal of the cause, usually a tight splint or plaster, but may necessitate exploration of the nerve and the excision of perineural scar tissue, or mobilisation of the nerve from callus, following which a new bed is prepared for the nerve in adjacent muscles, the nerve itself being wrapped in a layer of tantalum foil.

Sensory symptoms are satisfactorily relieved by these steps, but if intervention is delayed too long intraneural fibrosis may lead to persistent motor disability.

Progressive fibrosis produced by fixation of the nerve has to be treated by nerve transplantation (p. 446); recovery may be complete or incomplete, according to the degree of fibrosis.

NEUROTOMESIS

is usually produced by penetrating wounds and results in partial or complete division of the nerve sheath and fibres. Very rarely, complete rupture of nerves may occur in major brachial plexus injuries. In war and severe industrial injuries extensive soft tissue damage to muscle and skin, and associated fractures, greatly complicate the problems of repair.

Partial lesions produce a lateral neuroma of the nerve. Complete division produces a terminal neuroma on the end of the proximal segment. In the proximal portion of the divided axons, retrograde degeneration occurs as high as the first node of Ranvier. After an interval of ten days the axons begin to subdivide to produce an excess of end bulbs, which then commence to grow downwards. By this time, however, the gap between the divided nerve ends, which was at first filled with blood, has now been replaced by organising clot and fibrous tissue, which presents an almost impenetrable barrier to the down-growing axons. In the distal segment of the divided nerve, Wallerian degeneration of the axons occurs, but the cells of the sheaths of Schwann proliferate, forming a slight bulb at the commencement of the distal end from which sprouts of Schwann cells grow proximally into the plane of division, being drawn by chemotaxis towards the down-growing axons. A few axons may by this means succeed in entering the distal segment, producing there a deceptively encouraging Tinel's sign, but any spontaneous healing is minimal.

Even after accurate nerve suture the quality of regeneration is less perfect than in cases of axonotmesis owing to the wastage of axons in the scar tissue at the suture line, and mal-distribution of those fibres which reach the distal segment. The density of scar tissue at the suture line is increased by local sepsis and inflammation, and by tension at the suture site.

Mal-distribution of fibres is greatest in the case of mixed motor and sensory nerves, for motor fibres may then unite with sensory endings. It is also greatest in those motor nerves which supply a large number of small muscles; hence the quality of recovery is best in the case of a pure motor nerve, such as the radial, which supplies a few groups of large muscles concerned in coarse movement, and is worst in mixed sensory and motor nerves supplying a large number of small muscles concerned in fine movement, such as the ulnar or median nerves at the wrist.

Treatment is by suture under suitable conditions, in the absence of sepsis and in the absence of tension, when the nerve can be placed in a suitable bed. In war injuries where there has been considerable loss of surface tissue, preliminary plastic operations such as pedicle grafting must precede attempted repair of the nerve in order to provide a suitable covering and bed at the suture site.

Open Wounds.—Until recently it was considered that, unless infection was anticipated, immediate primary suture was the ideal treatment for a divided nerve. Surgeons with special experience of these injuries now recommend that the wound should be treated on accepted lines, i.e. excision and closure if possible, but that the nerve suture should be postponed until three or four

Louis Antoine Ranvier, 1835–1922. French histologist and pathologist.
Theodor Schwann, 1810–1882. Professor of Anatomy, Louvain, 1839–48, and thereafter at Liège. Original researches before the age of twenty-seven laid the foundation of Physiology of Nerve and Muscle. The first to deal with problems related to living matter on a purely physical and chemical basis, and to recognise the cell as the unit of living matter. Discoverer of pepsin, and rôle of living organisms in fermentation.

weeks after the injury. If a divided nerve is encountered during excision of the wound, the ends are approximated by one stitch of fine silk, which prevents retraction during the period of delay. On no account should an attempt be made to identify or scrutinise a nerve in the vicinity. If only one end of a nerve is seen, then a suture should be inserted so as to fix it to adjacent muscle or fascia.

The advantages of early secondary suture over immediate primary suture are as follows :

(i) Primary suture usually requires enlargement of the wound by further incisions, so as to mobilise the nerve in order to allow approximation of the ends without tension. As the wound is potentially infected, exposure of previously uncontaminated tissues should be avoided.

(ii) The normal nerve sheath is a delicate structure, which is easily torn by the slightest tension, and accurate suturing is essential for the success of the operation. In addition, the sheath is often further weakened by longitudinal slits or tears. After about three weeks of the injury, epineural fibrosis occurs and the sheath becomes thicker and tougher, consequently the insertion of sutures and accurate coaptation of the nerve ends are greatly facilitated.

NERVE SUTURE

The two ends of the nerve are identified, the incision being prolonged sufficiently to expose the nerve well above and below the seat of injury, in such a position that its normal anatomical relations are not obscured by scar tissue. The two ends of the nerve are freed and 'freshened' by means of a scalpel or a Bard-Parker knife. Scissors should not be used, as the nerve is crushed thereby. Slices are removed from the ends of the nerve until the projecting fibres are seen, and blood freely oozes from the cut surface. Apposition of the two ends is accomplished as follows:

(a) *Mobilisation*.—The two ends are dissected from surrounding structures, care being taken to preserve important motor branches. Branches can often be stripped from the parent nerve in order to facilitate mobilisation.

(b) *Posture*.—The limb being held in a suitable position.

(c) *Transposition*.—The radial nerve is brought in front of the humerus, or the ulnar nerve in front of the internal condyle (p. 446).

(d) *Nerve Anchoring*.—If it is obvious that the two ends of a divided nerve cannot be brought together, on account of excessive loss of tissue or retraction, then the

two untrimmed ends are approximated as closely as possible by tension stitches, the position of the limb being such that approximation is facilitated. Subsequently the nerve is stretched by gradually straightening the limb, and at a second operation the two ends are brought together. In some cases adequate suture can be performed while the limb is flexed, and extension is gradually regained.

(e) *Resection of Bone*.—This extensive procedure may be justifiable if nerve injury is associated with an ununited fracture, which also needs operative measures, e.g. in the case of the radial nerve and a fractured humerus.

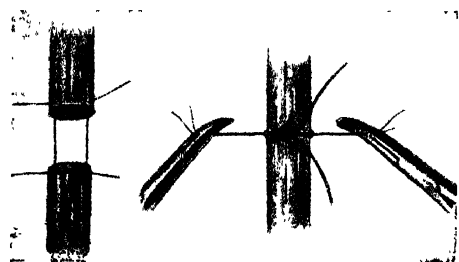


FIG. 499.—Primary nerve suture. Two sutures are passed through the sheath of the nerve in order to approximate the cut ends. The sheath is then sutured.

by one of the above procedures, sutures are introduced through the nerve sheath (fig. 499). Non-irritating material is used, such as fine black silk or tantalum

wire. Catgut encourages fibrosis of the nerve. The suture line can be further supported by painting with fibrin glue. Torsion of the nerve ends must be avoided, so that, as far as possible, proximal nerve fibres will join with their corresponding distal fibres. 'Shunting' is thus avoided, and delay due to re-education of groups of muscles is obviated. If adjacent tissues are fibrosed, a new path can be constructed for the sutured nerve by opening a muscle sheath, and embedding the nerve among the muscle fibres. Finally, a layer of tantalum foil is always wrapped round the nerve to prevent epineural fibrosis and the out-sprouting of axons from the suture line, which may produce a painful local lesion. The limb is placed in a suitable position to prevent any strain on the sutured nerve, and a plaster cast may be advisable for a few weeks to immobilise the limb.

RESULTS OF NERVE SUTURE

This depends on many factors:

1. **Pre-operative.**—(a) *The Nerve Affected.*—Mixed motor and sensory nerves, e.g. ulnar and median, are subject to a greater degree of maldistribution of down-growing fibres than purely motor nerves supplying a small number of large muscles, e.g. radial.

(b) *Infection* may cause delay in performing suture at the appropriate time and increases scar tissue formation following suture.

(c) *Time.*—Early secondary suture (p. 438) yields the best results, and further delay is detrimental.

(d) *Pre-operative Treatment.*—If muscles and tendons have been allowed to stretch or if the tone of the muscles has not been maintained, then the chances of recovery are correspondingly diminished.

2. **Operative.**—This consists of attention to the details already mentioned, e.g. hæmostasis, prevention of torsion and tension, preparation of suitable bed, and the use of non-irritating suture material.

3. **Post-operative.**—(a) *Absence of Infection.*—If the wound is already infected, or if infection supervenes, then little improvement is likely.

(b) *After-treatment* consists in continuance of the relaxation of paralysed muscles, massage, electrical treatment, and muscular effort gradually increased as muscles recover their power.

(c) *Co-operation of the Patient.*—This important factor must receive due consideration and the patient given every encouragement during his rehabilitation.

(d) *Vicarious Movements.*—Although the physiological results of nerve suture may be poor, yet adjacent muscles often take upon themselves some of the functions of those which are paralysed, e.g. if the hamstrings are paralysed and flexion of the knee thereby affected, the sartorius and gracilis muscles hypertrophy, and partially compensate for this deficiency. Thus the functional result of a nerve injury is often more satisfactory than the physiological recovery would suggest.

Irremediable Injury.—If suture is impossible on account of loss of tissue or wide separation of the ends of a divided nerve, the following procedures may be considered:

(a) *Nerve anastomosis*, e.g. part of the hypoglossal nerve is united to the distal end of the facial nerve. This method often results in improvement, but 'successful' cases are sometimes associated with uncontrolled grimaces on movement of the tongue.

(b) *Nerve grafting* has produced encouraging results in selected cases, e.g. a gap in the facial nerve within its bony channel may be bridged by insertion of an autograft from the external cutaneous nerve of the thigh. Grafting of peripheral nerves is disappointing.

(c) *Tendon transplantation*, e.g. in the case of radial paralysis, tendons and muscles of the forearm may be transplanted into the extensor group. However, if proper relaxation of the extensor muscles has been consistently maintained, drop wrist should not occur, as the extensor tendons will not be over-stretched.

(d) *Arthrodesis*, e.g. in the case of injury to the sciatic nerve, arthrodesis of the flail ankle joint will render it stable and rigid.

(e) *Amputation*, for persistent sores and ulcers on the foot, particularly if growth is impaired. Sympathetic ganglionectomy might first be tried (p. 140).

Incomplete division of a nerve gives rise to a central or lateral neuroma. Effects vary according to the extent of the injury. Fibres supplying certain muscles are often constant in position, and hence are more liable to injury if their position exposes them to trauma. Thus partial division of the great sciatic nerve affects the lateral popliteal portion nine times more commonly than the medial popliteal, which passes down on the inner and deeper aspect of the great sciatic nerve.

Partial lesion of the median or medial popliteal nerves, or injury to their branches, may give rise to the distressing condition of *causalgia* (p. 449).

Injection lesions are due to accidental injection of therapeutic agents. They are more common in tropical countries where amœbiasis, schistosomiasis, and malaria are treated by intramuscular injections. The sciatic and radial nerves are usually involved. Injections in the buttock should always be given into the upper and outer quadrant (fig. 4) and in the case of the arm into the upper half of the deltoid muscle.

SPINAL NERVES

Injuries of the **Cervical Plexus** are uncommon, although muscular branches, e.g. to the trapezius and sternomastoid muscles, are occasionally damaged (p. 433).

The **Phrenic Nerve**, which arises from the third, fourth, and fifth cervical nerves, may be crushed in order to cause a temporary diaphragmatic paralysis and so reduce the size of the pleural cavity in such conditions as bronchiectasis or tuberculosis, and also to diminish movement after repair of the diaphragm for a hernia. Should the temporary paralysis produced by crushing prove to be insufficient, it is not difficult to recrunch the nerve.



FIG. 500.—Elevation of the diaphragm following involvement of the right phrenic nerve by extension of a malignant tumour.

Paralysis of the diaphragm secondary to malignant disease in the thorax is equally frequent on the right and left sides and is often due to direct invasion of the phrenic nerve by adjacent growth (fig. 500). When phrenic paralysis is due to lymph-node metastases, it is almost always due to involvement of a node which lies alongside the phrenic nerve at the level of the pulmonary artery at the upper region of the hilum.

Brachial plexus lesions are either complete or partial (fig. 501).

Complete lesions are rare, as an injury of sufficient severity to damage all the roots of the plexus will probably inflict fatal injuries on adjacent important structures.

In the event of a complete lesion, anæsthesia of the upper limb occurs except over areas supplied by the supra-acromial branches of the cervical plexus and the intercosto-humeral nerve. Complete paralysis of arm and scapular muscles occurs, except that in the case of tears of the plexus the lesion is usually distal to the site at which the nerve of Bell and the nerve to the rhomboids arise, and consequently the serratus magnus and rhomboid muscles escape.

Incomplete lesions, if due to stabs or cuts, are liable to affect any of the roots, the clinical features depending on the nerves divided. The commonest

type of injury is due to traction or pressure, and affects either the upper or lower portions of the plexus.

Although the segmental innervation of the arm muscles is somewhat inconsistent, the following table summarises a distribution which is commonly accepted :

<i>Nerve</i>	<i>Muscles</i>
C.v . . .	Rhomboids, spinati, deltoid, teres minor, biceps, brachialis, brachio-radialis, supinator brevis.
C.vi . . .	Pectoralis major (clavicular head) and minor, subscapularis, coracobrachialis, latissimus dorsi, teres major, serratus anterior, triceps, pronator teres, pronator quadratus.
C.vii . . .	The extensors of the fingers, extensor carpi ulnaris, and sternal part of the pectoralis major.
C.viii . . .	The flexors of the wrist and fingers.
D.i . . .	The small muscles of the hand.

Upper Lesion (Erb-Duchenne, fig. 501 (1)). This injury is due to excessive displacement of the head, depression of the shoulder, or a combination of these two conditions.

It affects infants after a difficult confinement or adults following blows or falls on the shoulder. The fifth and sometimes the sixth cervical roots are involved. In the former case the muscles affected are the biceps, brachialis, brachio-radialis, supinator brevis, spinati and deltoid, and thus the limb, internally rotated by the unopposed subscapularis,

hangs by the side with the forearm pronated, in the - well

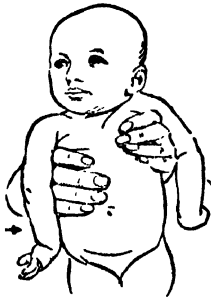


FIG. 502.—Erb's paralysis.

known 'tip' position (fig. 502). Sensory changes are absent if the fifth nerve only is involved, but if the sixth nerve also suffers, an area of anæsthesia is present over the outer side of the arm. Since the innervation of the hand is preserved, functional improvement is obtained by arthrodesis of the shoulder and elbow joints in suitable cases, together with transplantation of muscles

from the pectoral groups to the humerus or scapula.

Lower Lesion (Klumpké, fig. 501 (2)).—The lower nerve trunk or the inner cord are injured either above the clavicle, classically by inclusion with the subclavian artery in a ligature, or in the axilla, as by an unreduced dislocation of the humerus. In either case, the inner portion of the plexus is involved, and wasting of all the small muscles of the hand occurs, together with sensory loss along the inner side of the forearm and the inner three and

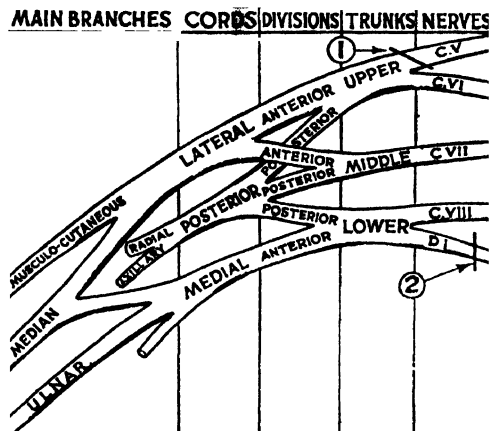


FIG. 501.—The brachial plexus, showing two classical sites for injury—see text.

a half fingers, owing to involvement of fibres passing to the ulnar and inner head of the median nerves.

Nerve roots may be avulsed from the spinal cord, as in the case of a falling person clutching at an object and hyper-abducting his arm, e.g. failing to obtain a foothold on a passing bus. The first dorsal root is usually affected. Paralysis of the intrinsic muscles of the hand results, with anæsthesia of the inner three and a half fingers in front, and inner one and a half behind. In addition, the oculopupillary fibres, which pass along the first dorsal nerve to the rami communicantes, and so to the cervical sympathetic, are also affected, and Horner's syndrome follows (fig. 138). Hæmorrhage sometimes occurs in the spinal cord following avulsion of the nerve root, and results in damage to the pyramidal tract and consequent spasticity of the leg on the same side.

Treatment.—Subcutaneous injuries are at first treated on expectant lines, paralysed muscles being relaxed. Thus, in the case of an Erb's paralysis, the arm should be fixed in a position of right-angled abduction and eversion, to relax the deltoid and spinati, the forearm being flexed and supinated in order to relax the biceps, brachialis, and supinators. In serious brachial plexus lesions, physiotherapy must be continued for two years before recovery is assessed.

Some minor improvement usually occurs during this time, especially if the initial loss of function has been due to neurapraxia and stretching rather

than rupture of nerve fibres. When fibres have been ruptured, a great deal of mal-distribution of down-growing fibres among the complex plexus branches is bound to result in considerable reduction of functional efficiency. Where the nerve tracts are torn across (neurotmesis) little recovery is to be expected, and such cases are rarely improved by exploration of the brachial plexus.

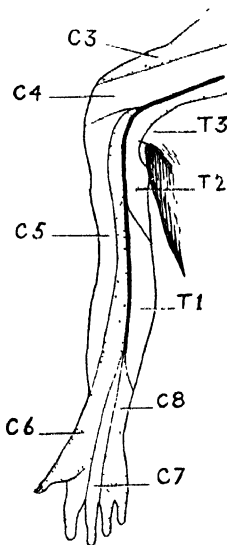


FIG. 503.—The dermatomes of the upper limb.

BRACHIAL NEURALGIA

This condition (*syn.* brachial neuritis) is comparable to sciatica in the leg. The cutaneous distribution of the cervical nerves is illustrated in fig. 503, and the muscular innervation is summarised on p. 441. Some of the more important causes of brachial neuralgia are as follows :

Spinal Tumour (p. 414).—Evidence of pressure on the cord is usually present, and Queckenstedt's test is commonly positive.

Prolapsed intervertebral disc (p. 421) is often difficult to distinguish from osteoarthritis, but localised pain in the neck, which often appears suddenly and which is aggravated by coughing, suggests a disc lesion.

Osteoarthritis of the cervical spine (*syn.* spondylitis) causes neuralgia either as a result of pressure on a spinal nerve by an osteophyte or from absorption of the disc and consequent compression of the spinal roots. The onset is gradual and symptoms intermittent, and radiography usually establishes the diagnosis.

Costo-clavicular syndromes are due to many conditions. Sagging of the shoulder girdle, especially in middle-aged females, is a common cause, quite apart from

cervical rib. Exercises and physiotherapy directed to improving the tone of muscles which elevate the shoulder may relieve the symptoms in such cases.

PERIPHERAL NERVES

The **Circumflex Nerve** passes through the quadrilateral space, and winds around the shaft of the humerus about one finger's-breadth below the centre of the deltoid muscle. It is sometimes injured by a direct blow, or involved by a fracture or dislocation of the humerus.

The deltoid muscle is paralysed and wastes rapidly, and a patch of anæsthesia over the outer side of the arm distinguishes this condition from a partial lesion of the fifth cervical nerve (fig. 504). Paralysis of the teres minor is unrecognisable clinically.

Recovery commences in a few weeks if the cause of the compression is removed, provided that the arm has been supported in right-angled abduction.

The **Nerve of Bell**, or external respiratory nerve, arises from the fifth, sixth, and seventh cervical nerve roots and supplies the serratus anterior muscle.

It may be injured by blows or carrying a heavy object on the shoulder, or during operations on the breast or chest wall, as it lies on the inner wall of the axilla. Paralysis of the serratus anterior allows 'winging' of the scapula, i.e. the vertebral border and inferior angle are unduly prominent (fig. 505). The 'lunge' stroke of fencing is dependent on the serratus anterior, and this and similar movements, such as pushing forward with the arm, are deficient. Owing to inability to rotate the scapula on the chest wall, difficulty is experienced in raising the arm above a right angle from a position in front of the body.



FIG. 504.—Wasting of the left deltoid muscle and area of anæsthesia due to bruising of the circumflex nerve.



FIG. 505.—Winging of the scapula; the patient is pushing against a wall.

Suture of the nerve is sometimes possible. Otherwise, if disability warrants such a procedure, the scapula is steadied by a slip of pectoralis major muscle, which is detached from the humerus and fixed to the inferior angle of the scapula.

The **Radial** or musculospiral nerve is commonly injured. The classical sites are the axilla and the radial groove.

1. Injury in the axilla follows:

(i) **Crutch palsy** : all crutches should have hand-grips, and their length should be carefully adjusted, especially if use is likely to be prolonged. Paresis has occurred after only four hours' use of crutches unsupplied with hand-grips.

(ii) Fractures and dislocations of the upper end of the humerus, or by attempts at their reduction.

(iii) Rarely by pressure of an aneurysm or new-growth.

Clinical Features.—(a) *Motor.*—The triceps and extensors of the wrist and fingers are paralysed, and consequently inability to extend the elbow, wrist, and fingers results, and wrist drop is present. If the hand is supported, as by supporting it upon the table, extension of the fingers can be produced by the action of the lumbricals and interossei, which are inserted into the extensor expansions. The supinator and brachio-radialis are also paralysed, but supination is ably performed by the biceps. The brachio-radialis muscle is tested readily by endeavouring to flex the semi-prone forearm against resistance. If the muscle is active, the contraction is visible, and the rigid muscle is easily palpable.

(b) *Sensory.*—Anæsthesia is present over the dorsum of the forearm and back of the hand where anæsthesia is reduced to a patch over the base of the thumb and first interosseous space owing to overlap of the musculocutaneous and other adjacent nerves (fig. 506). In peripheral nerve injury when sensory function is not restored, the final sensory loss is less extensive than the ana-

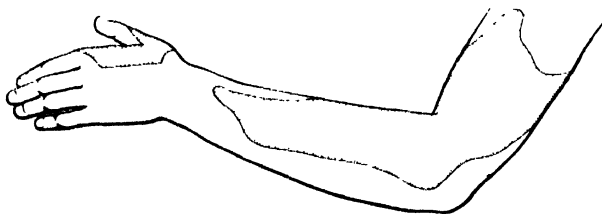


FIG. 506.—Areas of anæsthesia and anhidrosis following complete division of the radial nerve above the dorsal cutaneous nerve of the forearm and lower lateral cutaneous nerve of the arm. In lesions below the origin of the dorsal cutaneous nerve of the forearm the sensory loss is limited to the lower segment depicted over the dorsum of the base of the thumb and first interosseous space.

tomical distribution of the nerve owing to the overlap of adjacent sensory areas.

(c) *Trophic.*—These are usually trivial.

2. **Injury in the radial groove** is due to :

(i) Pressure, e.g. of the arm on the edge of the operating table, especially in Trendelenburg's position, or as in 'Saturday night' paralysis, due to the enjoyment of a heavy sleep with the arm over the sharp back of a kitchen chair. Prolonged application of a tourniquet is especially liable to compress the radial nerve, as it lies close to the bone, and possibly the median and ulnar nerves as well. For this reason a sphygmomanometer should always be used on the arm.

(ii) Fracture of the shaft of the humerus when immediate injury of the nerve occurs in about 8 per cent. of cases. It is often overlooked owing to the more obvious fracture overshadowing the nerve injury, in which case involvement in callus is usually blamed, rather than oversight on the part of the surgeon.

(iii) The nerve being overstretched during operations on the humerus, e.g. in dealing with an ununited fracture.

(iv) 'Intramuscular' injections of drugs being given into the radial nerve.

Clinical Features.—(a) *Motor.*—These are similar to those following injury in the axilla, except that the triceps and anconeus muscles escape.

(b) *Sensory*.—If the external cutaneous branch escapes, anæsthesia will be limited to a patch over the ball of the thumb. Division of the radial nerve in the upper third of the forearm is symptomless. Below this position the musculo-cutaneous nerve joins the radial, and division then causes anæsthesia over the ball of the thumb.

(c) *Trophic*.—These are slight.

The *posterior interosseous nerve* may be injured as a result of fracture or dislocation of the upper end of the radius, or in operations performed to deal with these conditions. Paralysis of the extensors of the wrist and fingers results. The upper end of the nerve has been sutured with excellent results. If nerve repair is impracticable, good results are obtained by a tendon and muscle transplant, e.g. the radial carpal flexor is transplanted to the extensor tendons of the thumb and fingers, and the pronator teres into the extensors carpi radialis longus and brevis.

The **Median Nerve** is classically injured at the elbow or wrist.

1. Injuries at the elbow are due to fractures of the lower end of the humerus or dislocations of the elbow joint. A tourniquet endangers the nerve at any level in the arm, in which case other nerves, particularly the radial, will also be involved.

Clinical Features.—(a) *Motor*.—The pronators of the forearm and flexors of the wrist and fingers, with the exception of the flexor carpi ulnaris and the inner part of the flexor profundus digitorum, will be paralysed. As a result of paralysis of the flexor carpi radialis, the hand deviates to the ulnar side when flexed against resistance. The index finger cannot be flexed at the phalangeal joints—the ‘pointing index’—but flexion of the other fingers is performed by that portion of the flexor profundus digitorum which is supplied by the ulnar nerve. Flexion of the terminal phalanx of the thumb is impossible, owing to paralysis of the flexor longus pollicis. The muscles of the thenar eminence are wasted and paralysed, and on inspection the eminence is flattened, so that the metacarpal bone of the thumb is apparently on the same plane as the other metacarpal bones—the so-called ‘simian’ or ‘ape-like’ hand. Paralysis of the two outer lumbricals is unrecognisable.

(b) *Sensory*.—Appreciation of touch is at first lost over the thumb and radial two and a half fingers in front, and posteriorly as far proximally as the middle of the proximal phalanges. Loss of response to pin-prick affects the terminal phalanges of the index and middle fingers, but sometimes a larger area is involved. Deep sensibility is lost over the terminal phalanges of the index and middle fingers. Later this area is reduced by overlap from adjacent nerves.

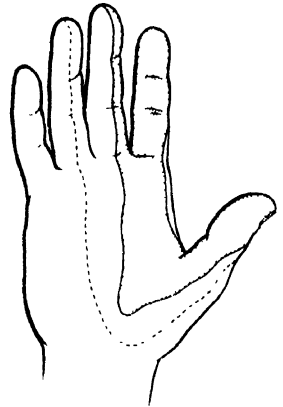


FIG. 507.—Area of persistent sensory loss in a complete median nerve injury above the wrist showing, in broken line, the distribution of the median nerve and, in stipple, the reduced area of anæsthesia, anhidrosis and analgesia finally persisting after injury owing to overlap of adjacent nerves. The ulnar nerve overlaps the ulnar side of the third finger.

(c) *Trophic*.—Obvious trophic changes are usually seen in the hand and affected fingers. Causalgia may complicate partial injuries (p. 449).

2. Injuries at the **wrist** are comparatively common, and are due to cuts from a variety of causes. Fractures of the lower end of the radius or dislocation of the semilunar bone sometimes cause injury to the median nerve.

Clinical Features.—(a) *Motor*.—The muscles of the thenar eminence are paralysed and wasted. The hand is 'simian', and abduction and opposition of the thumb are lost. Attempts to oppose the tip of the thumb to the tip of the little finger result in flexion and adduction of the thumb, as the patient is unable to swing it across the palm.

(b) *Sensory*.—Sensory losses resemble those following an injury at the elbow. Muscular sense is not impaired if tendons are not severed. Thus, as no striking muscular deficiency occurs, and as no part of the hand is completely anæsthetic, a divided median nerve at the wrist is readily overlooked, particularly in those who use refinements of sensation but little, e.g. a horny-handed labourer.

(c) *Trophic*.—These occur, as with an injury at the elbow.

The poor prognosis of a divided median nerve is rendered even more gloomy if tendons are also severed.

Median compression occasionally occurs as the nerve passes through the carpal tunnel. Paræsthesia, followed by wasting of the thenar muscles, suggests the diagnosis. A description is to be found on pp. 312 and 313.

The **Ulnar Nerve** is also classically injured at the elbow and wrist.

1. Injuries at the **elbow** are due to the following causes :

- (i) Fractures in the region of the internal condyle.
- (ii) Excision of the elbow joint.
- (iii) Cubitus valgus, due to old injury of the humerus and increase of the 'carrying angle' (fig. 235). Hence the nerve is unduly stretched, and friction occurs as the groove on the internal condyle becomes a pulley, and this continuous friction results in interstitial neuritis. This condition may occur many years after the original injury, and transposition of the nerve is required.

Anterior transposition of the ulnar nerve is sometimes required for friction (axonotmesis), following fracture of the internal condyle, pressure by an osteophyte, recurrent dislocation of the nerve, or injury which results in loss of substance, so that approximation is thus rendered possible. The nerve is exposed by a curved incision with the concavity forwards, and the humeral head of the flexor carpi ulnaris is divided. Careful dissection is necessary so as to avoid injury to motor branches, and the internal intermuscular septum should be divided or excised, otherwise the

nerve may be kinked by this structure when it is displaced forwards. A bed is then prepared in the flexor group of muscles and the nerve buried therein (fig. 508).

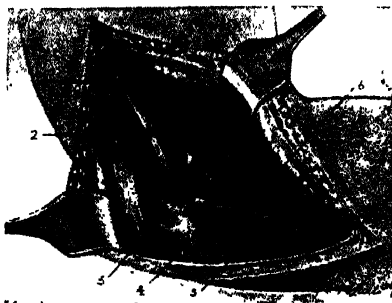


FIG. 508.—Anterior transposition of the left ulnar nerve.

1. Ulnar nerve.
2. Triceps.
3. Olecranon head of flexor carpi ulnaris.
4. Posterior ulnar recurrent artery.
5. Flexor profundus digitorum.
6. Superficial flexor muscles.
7. Inferior profunda artery.

(The dotted line represents the new course of the nerve.)

Clinical Features.—(a) *Motor.*—The flexor carpi ulnaris and inner portion of the flexor profundus digitorum are paralysed. Normally, on flexion of the wrist, the tendon of the flexor carpi ulnaris is readily palpable just above its insertion into the pisiform bone, but when the muscle is paralysed the tendon is impalpable, and wasting causes flattening of the inner border of the forearm. Weakness of the flexor profundus digitorum results in hyperextension of the little, ring, and slightly of the middle fingers at the metacarpophalangeal joints.

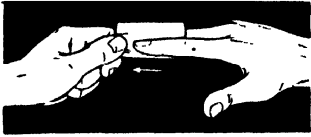


FIG. 509.—Test for weakness of the interosseous muscles.

Paralysis of the small muscles of the hand also results, with the exception of the thenar muscles and outer two lumbricals. Inability to abduct and adduct the fingers results, and the patient cannot grip a piece of paper placed between the fingers (fig. 509). If the patient pinches a piece of paper between his thumb and fingers the terminal phalanx of the thumb assumes a flexed position, as weakness of the adductor pollicis permits over-action of the long flexor of the thumb (Froment's sign) (fig. 510). Considerable wasting occurs, which is obvious in the interosseous

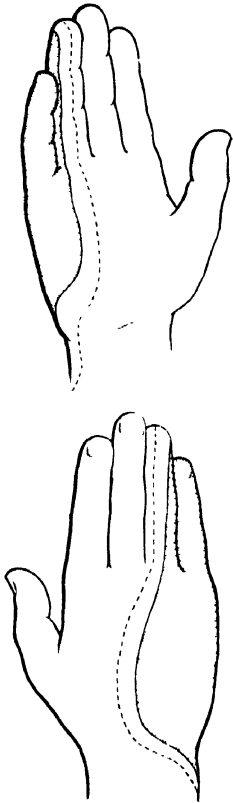


FIG. 511.—Persistent sensory loss in a complete ulnar nerve injury showing, in broken line, the anatomical distribution of the ulnar and, in stipple, the reduced area of persistent anaesthesia finally resulting.

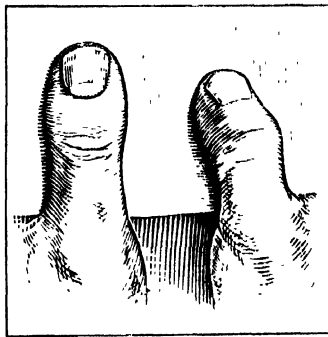


FIG. 510.—Froment's sign for right ulnar paresis.



FIG. 512.—Wasting of the hypothenar eminence following injury to the left ulnar nerve.

spaces and along the inner border of the hand, the normal curve being lost (fig. 512).

(b) *Sensory.*—The appreciation of light touch is at first lost over the inner one and a half fingers in front and behind. Response to pin-prick is lost over the little finger and ulnar border of the palm (fig. 511). Later this area will be reduced by overlap from adjacent nerves.

(c) *Trophic.*—These changes are usually well marked.

2. Injury at the wrist is due to the same causes as those enumerated in connection with the median nerve.

The ulnar nerve passes in front of the anterior annular ligament, and is damaged by more superficial injuries.

Clinical Features.—(a) *Motor*.—Paralysis and wasting of small muscles of the hand, as described above.

(b) *Sensory*.—The dorsal cutaneous branch of the ulnar nerve leaves the main trunk about $2\frac{1}{2}$ inches (6.3 cm.) above the styloid process of the ulna. Sensation is therefore lost only on the anterior aspect of the inner one and a half fingers.

(c) *Trophic*.—Correspond to the area of sensory loss.



FIG. 513.—An inguinal hernia which followed appendectomy with drainage through a gridiron incision one year previously.

The **Twelfth Dorsal Nerve**, as in the case of the intercostal nerves, is sometimes implicated by severe neuralgia, which may be associated with herpes zoster. More commonly the nerve is implicated by a suture during a kidney operation, or by subsequent scar tissue. Resulting pain is occasionally sufficiently severe to necessitate exposure and excision of part of the nerve.

The **Ilio-inguinal Nerve** may be damaged on the right side in a gridiron incision for appendectomy, although with care the nerve should be avoided. If drainage tubes are inserted through this incision, the resulting scar tissue may implicate the nerve. On the left side, injury to the nerve may follow iliac colostomy. Weakness of the conjoined tendon results, with consequent predisposition to the formation of an inguinal hernia (fig. 513).

The **External Cutaneous Nerve** is occasionally compressed as it passes through the deep fascia of the thigh, especially in muscular subjects, e.g. oarsmen. The condition is termed *meralgia paræsthetica*, and resection of part of the nerve is sometimes necessary to rid the patient of pain or paræsthesia.

The **Sciatic Nerve** is occasionally injured by wounds, fractures, or 'intramuscular' injection of drugs. The component nerves in the pelvis may be involved by fracture, tumour, or aneurysm. Injury in the upper part of the thigh sometimes complicates deep wounds or posterior dislocation of the hip joint. If the lesion is above the origin of branches to the hamstrings, the following features will be present:

(a) *Motor*.—The flexors of the knee are paralysed, but some degree of flexion is possible owing to the action of the sartorius and gracilis muscles. Complete paralysis exists below the knee, and the pull of gravity therefore causes foot drop.

(b) *Sensory*.—Complete loss below the knee, with the exception of the skin supplied by the long saphenous nerve, i.e. a strip along the inner side of the leg extending along the inner border of the foot to the ball of the big toe.

(c) *Trophic*.—Especially on the sole of the foot and toes.

(d) *Causalgia* may complicate partial lesions (p. 449).

Partial involvement of the sciatic nerve affects the lateral popliteal portion nine times as commonly as the medial popliteal.

The **Lateral Popliteal Nerve** is injured as follows :

(i) Subcutaneous tenotomy of the biceps tendon.

(ii) Fracture or excision of the upper end of the fibula.

(iii) Pressure from plasters or splints.

(iv) In operations for multiple ligation of varicose veins.

Clinical Features.—(a) *Motor*.—Complete paralysis of the extensor and peroneal groups of muscles, with resulting talipes equino-varus.

(b) *Sensory*.—Anæsthesia of the outer side of the leg in its lower two-thirds, and of the dorsal aspects of all the toes, with the exception of the outer side of the little toe, which is supplied by the external saphenous (sural) nerve, as one contributory branch—the sural communicating from the medial popliteal nerve—escapes.

(c) *Trophic*.—Corresponding to the sensory loss.

The **Medial Popliteal Nerve** is but rarely injured on account of its protected position. The calf muscles and muscles of the sole are paralysed, and talipes calcaneo-valgus may result. The sole is anæsthetic, and trophic changes are usually severe. Causalgia occasionally follows a partial injury of the nerve or injury to one of its branches.

AUTONOMIC NERVOUS SYSTEM

Indications for Operations.

1. **To Improve Circulation** (Sympathectomy, pp. 121, 136 and 139).

2. **To Relieve Secretory Disturbances**

Excessive sweating (*hyperhidrosis*) is sometimes so distressing to the patient that he is willing to undergo surgical measures in order to obtain relief. Excessive sweating of the face, which is sometimes associated with emotional stress, is abolished by removal of the superior cervical ganglion. Excessive sweating of the hands responds promptly to preganglionic cervico-dorsal sympathectomy below the third dorsal ganglion (p. 140). Sudden, offensive feet, the skin of which is cracked and painful, may be a genuine disability for which lumbar sympathectomy is justified.

A *parotid fistula*, which fails to respond to cautery or radium, is encouraged to heal by avulsion of the auriculo-temporal nerve, which is exposed in front of the ear in relation to the superficial temporal artery. The parasympathetic secretory fibres are ablated, and the fistula closes.

Vagotomy to decrease gastric secretion is described in Chapter 32.

3. **To Relieve Painful Conditions**

Causalgia is a condition in which paroxysmal attacks of pain follow an incomplete nerve injury, especially of the brachial plexus, sciatic or median nerves. In more than half the cases symptoms supervene immediately after the injury, in the remainder symptoms are deferred for any length of time up to two or three months. The incomplete lesion on the nerve, usually a lateral neuroma, gives rise to antidromic impulses which pass peripherally to the sensory nerve endings where stimuli give rise to the production of histamine-like substance (H substance) similar to that liberated by the posterior nerve root vasodilators. The accumulation of H substance causes vasodilation and renders the part red and engorged, the affected area also sweats profusely and is exquisitely painful and hyperæsthetic owing to the increased pressure. It is noticeable that the patient tends to keep the limb elevated and usually prefers to have the affected area out of bed at night in order to diminish vascular engorgement. The skin is often thin as the result of trophic changes. The pain is alleviated by cold (e.g. a wet sock or glove). Sooner or later the patient loses his morale and becomes introspective and unco-operative. Some cases of causalgia tend to recover gradually, but the condition is often so distressing that surgical alleviation is desirable.

Treatment.—Paravertebral block (either D.2 and 3, or L.1, 2 and 3) relieves the pain for a short time, and confirms the necessity for operation (p. 122). Thoracic sympathetic trunk section or lumbar sympathectomy is the treatment of choice. The increase in the rate of capillary blood flow resulting from the dilatation of arterioles washes away the H substance and this diminishes tissue tension which is the principle cause of pain. Sometimes the relief is incomplete although the area of pain involvement is reduced in superficial extent.

Visceral Pain.—Bladder.—The constant wearying pain of chronic cystitis is relieved by resection of the presacral nerve, which may be combined with division of the sympathetic cords. Carcinoma of the bladder (or uterus) is apt, sooner or later, to escape from the confines of the viscus and cause pain from invasion of the lumbo-sacral plexus and other structures, which is beyond the aid of sympathectomy.

Uterus.—Certain cases of spasmodic dysmenorrhœa which resist less drastic measures are cured or relieved by presacral neurectomy. No untoward effects on uterine (including parturition) or bladder function have been recorded:—

Presacral Neurectomy.—The patient is placed in the Trendelenburg position and the abdomen is opened by a subumbilical paramedian incision. The bifurcation of the aorta is exposed by a vertical incision through the overlying peritoneum, which is easily lifted from the vessel. The presacral nerves are usually represented by a plexus, and in order to ensure complete removal, all the nervous, fatty, and areolar tissues lying over the lower inch of the aorta and the sacral promontory are excised.

Renal Pain.—The diagnosis of sympathetico-tonus is confirmed if the pain is relieved by eserine. In these cases pyelography shows clubbing of the calyces. Stripping of the renal pedicle results in sympathetic denervation and restoration of neuromuscular balance.

Angina pectoris is relieved by removal of the upper five thoracic ganglia. In cases of difficulty the lower two, if out of reach, can be injected with alcohol. It was formerly alleged that to abolish pain was to remove a danger signal, but further experience disproves this. Equally good results appear to follow thyroidectomy.

CHAPTER 20

LIPS, PALATE, FACE, AND EAR

EMBRYOLOGY OF THE FACE

ABOUT the sixth week of foetal life a depression appears in front of the head. Around this depression, called the stomodæum or primitive mouth, five processes appear :

a single one at the cephalic end, the fronto nasal process, and on each side a maxillary and a mandibular process (fig. 514). Soon the frontonasal process becomes subdivided, by the appearance of the olfactory pits, into a solitary median nasal process and two lateral nasal processes. The median nasal process becomes bluntly bifurcated, forming the processus globularis. Maxillary mesoderm extensions grow medially beneath the olfactory pits to join in the mid-line, thereby forming the primitive palate and excluding the placodes (future nostrils) from the upper lip. The process of budding and cohesion is a rapid one, for it is commenced and completed in the brief space of three weeks. Thus every congenital deformity of the face has existed from the ninth week of foetal life. The pinna is developed from six tubercles situated around the posterior end of the first branchial cleft.

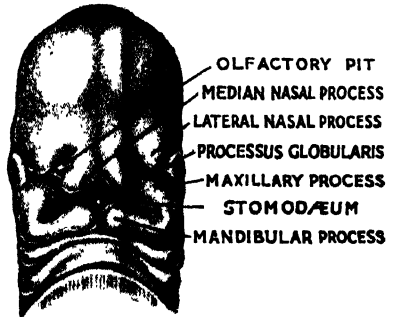


FIG. 514.—The head of an eight-week embryo.

The pinna is developed from six tubercles situated around the posterior end of the first branchial cleft.

CONGENITAL ABNORMALITIES

Cleft-lip and Cleft-palate

Classification.—Cleft-lip and cleft-palate are variations of one and the same congenital defect. Failure of coalescence of the developing processes (fig. 514) results in either (a) a cleft of the lip alone, (b) a cleft of the lip and

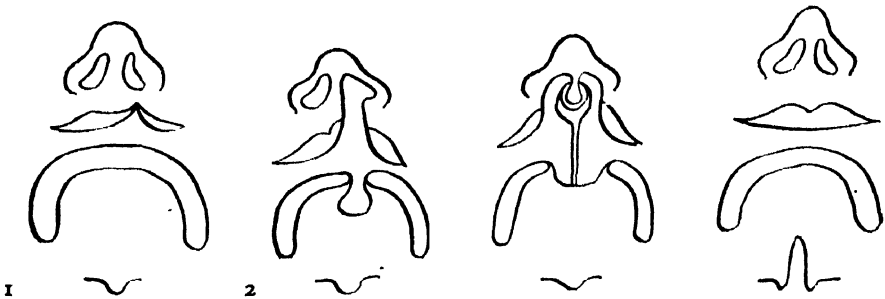


FIG. 515A.—1. Partial cleft-lip. 2. Complete cleft of lip and alveolus (ref. fig. 517). 3. Double cleft of lip and alveolus. 4. Cleft soft palate. (After Kernahan and Starke.)

the alveolus, (c) a cleft of the lip, alveolus, and palate, or (d) cleft-palate alone. The present adopted classification of cleft-lip and -palate is shown by figs. 515 A and B.

Normal union commences at the incisive foramen. (a) The extreme anterior portion of the Primary Palate and the alveolus and lip are formed in one process.

Failed formation may result in a cleft which involves part or all of the lip and may extend as far back as the incisive foramen. (b) The palate behind the incisive foramen—the Secondary Palate—is formed by the fusion of two shelves which grow medially from the maxillary arches. Failure of these to join results in a cleft-palate which may involve either the soft palate alone, or as far forward as the incisive foramen.

It can be seen, therefore, that a cleft may involve the lip and primary palate alone, or the Secondary palate alone, or a combination of these (Complete or Total Cleft-lip and -palate).

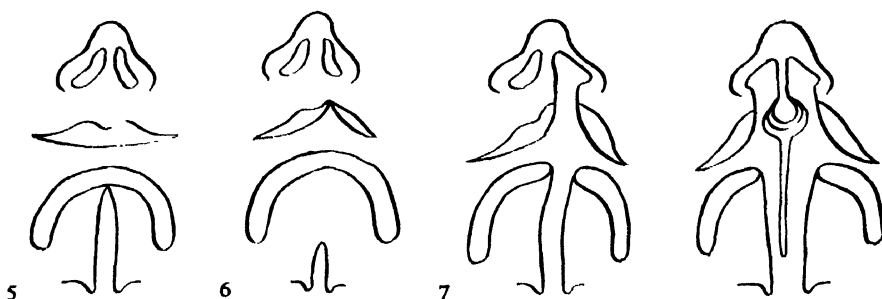


FIG. 515B.—5. Cleft soft and hard palate. 6. Partial cleft of palate and lip. 7. Complete cleft of lip, alveolus, and palate. 8. Double complete cleft of lip, alveolus, and palate (ref. fig. 521). (After Kernahan and Starke.)

In about 12 per cent. of cases the condition is familial, while in 10 per cent. the deformity is associated with other abnormalities of the head, such as hydrocephalus, congenital blindness and mental deficiency. Apart from the familial factor, exposure to X-rays, cortisone intoxication, vitamin deficiency, and certain virus infections (e.g. rubella) during the first trimester of pregnancy, are all associated with an increased incidence of these abnormalities in the foetus. For a reason that has never been explained, clefts on the left side greatly outnumber those on the right side and bilateral clefts.

Relative Distributions of the Various Types of Cleft.—Based on a study of the records of 703 Danish patients operated upon for one or other of these anomalies, P. Fogh-Andersen found that the incidence was as follows :

Cleft-lip alone	25 per cent. (60% males)
Cleft-palate alone	25 per cent. (59% females)
Cleft-lip and cleft-palate combined	50 per cent. (70% males)

In 75 per cent. of the patients the cleft was unilateral.

Effects on Function.—Swallowing, speech, dentition, nose development, and even hearing are affected.

Ability to Suck.—Cleft lip does *not* interfere with feeding (a special teat may be required for bottle feeding, but with breast feeding, a mother invariably acquires a technique to feed the baby adequately.

With cleft palate, however, the infant may be unable to suck properly as negative pressure cannot be established. In this case feeding may be assisted with special bottles and teats, or alternatively spoon feeding may be employed. In some cases an obturator is needed which facilitates sucking.

Speech.—A person with a cleft-palate is unable to make consonants and has a typical cleft-palate voice. To make consonants (e.g. B, D, K, P, T, and the hard G) air must not escape into the nose via the naso-pharyngeal sphincter (the levator sling pulls the soft palate backwards and into contact with the posterior wall of the pharynx). Eighty per cent. of patients may develop favourable speech, but usually require extensive speech therapy to achieve this.

Orthodontics.—The child will require very careful supervision of the growth of the alveolus and teeth, and corrective splinting where indicated.

The nose may remain misshapen (fig. 518), particularly if the upper lip does not develop to its full width and dimensions, and lack of development of the alveolar arch results in malpositioning of the teeth. Much depends upon the reconstitution of, and therefore the moulding action from, a properly functioning orbicularis oris muscle.

Hearing.—About 50 per cent. of children with cleft-palate have an audiometrically detectable hearing loss of about ten decibels, due largely to oedema of the orifice of the Eustachian tube consequent upon pharyngeal inflammation from regurgitated food.

Treatment

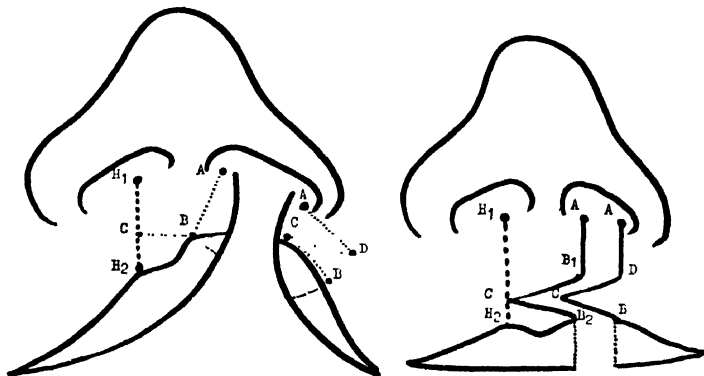
Aims of Treatment.—*Cleft-lip*—the aim is solely to improve appearances. (Cleft-lip does not interfere with feeding—see above.)

Cleft-palate—the aim is to achieve adequate speech and dentition. The length of the palate is not important, but mobility and muscularity are.

Timing of Treatment.—*Cleft-lip*—repair is usually undertaken at an age of three months or when the infant weighs twelve pounds (5–6 kg.).

Cleft-palate—repair is undertaken at the age of 1–1½ years.

FIG. 516.—Repair of cleft-lip (after *Tennison*). The main aim of the relaxing incisions is to provide an ample upper lip; since the scar does not grow with the child, deformity in adult life may be avoided.



Methods of Repair

Unilateral Cleft-lip: There are several different operations described for repair of the unilateral cleft-lip. Fig. 516 illustrates the method of repair described by *Tennison*.

Fig. 517 shows a cleft-lip before operation and fig. 518 the child at the age of four. Note the residual nasal defect.

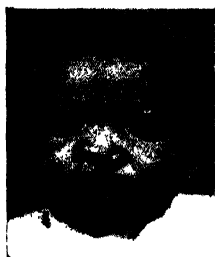


FIG. 517.—Cleft lip.



FIG. 518.—Same case as fig. 517 four years after repair.

Cleft-palate: The aim of repair of cleft-palate is to achieve adequate speech and dentition.

Operation.—Endotracheal anaesthesia is employed and the head is well extended. The principle of the repair is to achieve firm union of the muscle of the palate in the mid-line. In order to achieve this, incisions are made

laterally and the soft tissues extensively dissected to mobilise the palate. (This may involve fracturing of the hamulus). This repair is known as the V-Y repair

(fig. 519). The repair is effected by suturing the cleft in the mid-line in two layers (nasal and pharyngeal), leaving a raw area laterally which re-epithelialises.

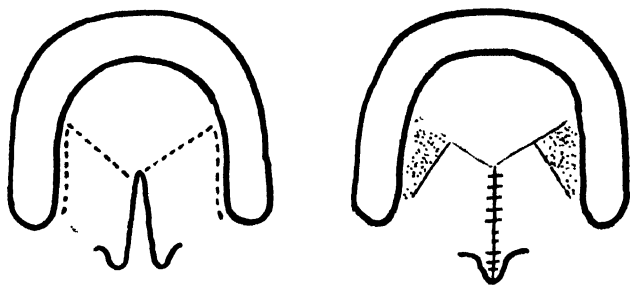


FIG. 519.—V-Y Repair of cleft-palate.

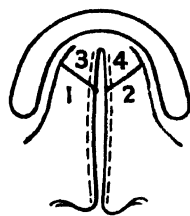


FIG. 520.—Four-flap method.

With clefts extending as far forward as the alveolar arch, the 'four-flap' repair is used (fig. 520).

When severe alveolar defects exist, bone-grafting is used after corrective orthodontic alignment.

Double Cleft-lip with an Unfused Premaxilla (fig. 521).—The premaxilla must not on any account be removed, for the subsequent concavity is most difficult to remedy with a prosthesis. The best method of treatment of these difficult cases is still open to question, but if the premaxilla juts out considerably the second of the following two procedures should be chosen.

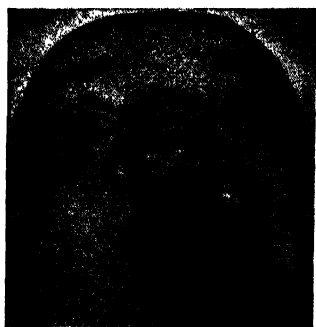


FIG. 521.—Bilateral cleft-lip associated with an unfused premaxilla and cleft-palate.

(a) The cleft on each side is united to the prolabium in one or two stages, and as a result of the continuous pull exerted by the reconstructed orbicularis oris, the premaxilla retreats between the halves of the maxilla.

(b) First the lip on one side is repaired, in order to ensure an adequate blood-supply of the adjacent bone. With this established, a few weeks later prevomerine bone can be resected safely and (at the same operation) the repair of the other side of the lip is performed (R. J. V. Battle). Again the reconstructed orbicularis

oris proves an admirable agent for retaining the replaced premaxilla.

Other Congenital Abnormalities

Preauricular Sinus.—Imperfect fusion of the six tubercles that form the pinna results in a preauricular sinus, the opening of which is usually found at the root of the helix or on the tragus. The track run downwards, and ends blindly. A preauricular sinus gives rise to no symptoms unless the tiny opening becomes occluded, when a cyst is prone to develop. If the cyst becomes infected and it bursts or is incised, a cutaneous preauricular ulcer may follow. This ulcer (fig. 522) refuses to heal, for infection is maintained from the sinus (Stammers). Often the condition is mistaken for a tuberculous sinus associated with a preauricular or parotid lymph node. The only curative treatment is complete excision of the sinus.

Periauricular dermoid cyst is due to inclusion of epithelium during fusion of two contiguous embryological aural tubercles. Usually the cyst is posterior to the pinna (fig. 523).

Congenital Short Frænum¹ of the Upper Lip.—Both lips have a frænum, that of the upper lip being better developed. Sometimes it is congenitally short and is

¹ Frænum—Latin = a bridle.

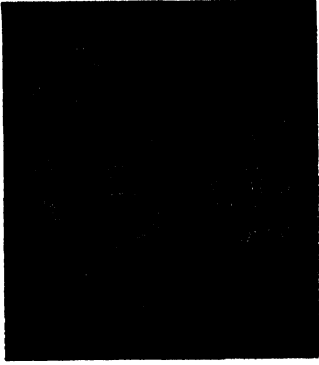


FIG. 522.—Preauricular sinus with ulcer. Note the sinus in the root of the helix. (*G. D. Adhia F.R.C.S., Bombay.*)



FIG. 523.—Post-auricular dermoid cyst.

then associated with a wide gap between the permanent incisor teeth (fig. 524). If dental malposition, and a sibilant lisp in the speech are to be prevented early in life, the frænum must be incised transversely, the mucosa on each side of the incision mobilised by undercutting, after which the cut edges are sutured longitudinally.

Congenital fistulæ of the lower lip, unexplained by embryology, occur in certain families. There are two blind pits, one on either side of the mid-line. They are, apparently, wide-open mucus secreting labial glands which are also found in certain quadrupeds. The treatment is excision.

Prolapse of the Mucous Membrane of the Lip (Bichelia).—The mucous membrane of the lip protrudes as an unsightly fold below the vermilion border. Usually congenital, it can be acquired by repeatedly sucking the lining of the lip through a gap between the natural teeth or an ill-fitting denture, in which case the prolapse is unilateral and mostly affects the lower lip. **Treatment** consists of excision of redundant mucosa. In acquired cases orthodontic corrections must also be made.

Micrognathia (fig. 525).—This is often associated with cleft palate. The infant is born with a mandible the

horizontal ramus of which is foreshortened. This renders the tongue unduly mobile, and results in tongue-swallowing with frequent attacks of dyspnœa, cyanosis, and feeding difficulties, especially if a cleft-palate is present as well. **Treatment**—is conservative with attention to nursing (face down), the maintenance of an airway, and feeding. In young adulthood, cosmetic bone-grafting may vastly improve the patient's appearance.

Mandibular Prognathism (fig. 526).—This occurs as a developmental abnormality and

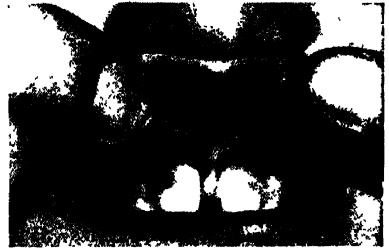


FIG. 524.—Congenitally short frænum labii. (*Patrick Clarkson, F.R.C.S., London.*)

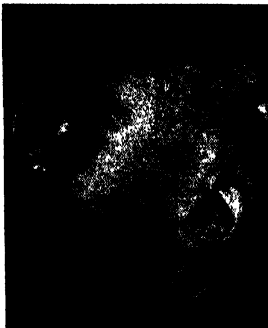


FIG. 525.—Micrognathia. An endotracheal tube has been passed. (*Sir Alan Moncrieff, London.*)



FIG. 526.—Mandibular prognathism. (*J.H. Howell, F.D.S., R.C.S., London.*)

may be genetically determined. The unopposed incisor teeth suffer early periodontal disease as a result of disuse. Faulty speech occurs on account of incorrect relationship of the tongue to the incisor teeth, and temporo-mandibular joint dysfunction is favoured. *Treatment*.—After freeing the periosteum from the bone, an open sliding osteotomy of the ascending ramus or the body, with preservation of nerves, and wire fixation of the fragments, gives satisfactory correction of the deformity. If the periosteum is not freed from the bone, muscle pull on the original attachment causes recurrence of the deformity.

LESIONS OF THE PALATE

Swellings of the Palate.—(a) A tumour of the palatal mucosa or bone: (b) carcinoma of the maxillary antrum extending downwards (p. 476); (c) an alveolar abscess of the incisors (p. 466); (d) chronic infections, e.g. gumma.



FIG. 527.—Ectopic salivary tumour.

considered as the differential diagnosis in fig. 527 since it starts as a rubbery rounded swelling which ulcerates in the centre, with the characteristic punched-out edge. Bone necrosis and sequestration result in the appearance shown in fig. 528.

Ectopic salivary tumour (fig. 527) is a special problem. Though it is a pleomorphic adenoma, it arises from ectopic salivary tissue, tends to recur after removal, and may be malignant. Unlike a squamous-cell carcinoma of this area which should primarily be amenable to radiotherapy, it requires surgical ablation. The excision must be adequate, and this usually means removal of adjacent palate and the subsequent use of an obturator (dental plate).



FIG. 528.—Perforation of the hard palate due to a gumma. Notice also the collapsed nose due to syphilitic destruction of the nasal septum.

A gumma is rare nowadays. It might be con-

Perforation of the Palate.—A hole in the *mid-line* of the hard palate may be due to friction from a malfitting dental plate. Rarely nowadays is it the result of a gumma (fig. 528). A hole *to one side of the mid-line* is due to the bursting of an empyema through the floor of the antrum, or a palatal antrostomy. *Treatment*.—A well-fitting dental plate is all that is required.



FIG. 529.—Primary chancre of the upper lip. (Dr. David Erskine, London.)

LESIONS OF THE LIPS

Pigmented Lips and Buccal Mucous Membrane.—Brown pigmented spots on the lips, inside the cheeks, and on the palate occur in familial intestinal polyposis (Peutz-Jeghers syndrome). Addison's disease must be excluded.

Macrocheilia.—True macrocheilia is due to a lymphangioma, akin to lymphangiomatous macroglossia (p. 494). Treatment is similar. Chronic inflammation also causes enlargement.

Cracked Lip.—Chapping is common, and a definite crack in the middle of the lower lip, which bleeds readily, is a frequent complaint in cold weather. *Treatment* includes the local application of glycerin. In chronic cases simple excision may be indicated.

Cracks at the corners of the mouth (p. 490).

Chancre of the Lip.—A chancre on a lip (fig. 529), usually the upper, is not a rarity. Unlike a similar lesion on the genitals, the neighbouring lymph nodes become greatly enlarged.

NEOPLASMS OF THE LIP

Lymphangioma (macrocheilia), and cavernous hæmangioma are innocent neoplasms (p. 104).

Ectopic Salivary Tumour.—Usually it is the upper lip of a young adult male which is affected, the tumour commencing on one or other side at the site of the right or left fusion lines (Kuber Dhar Sharma).

Carcinoma of the lip is usually seen in men between sixty and seventy years of age who have followed an outdoor occupation, and it has been known colloquially as 'countryman's lip'. Only comparatively recently has the importance of exposure to sunlight, wind and driving rain been stressed in the ætiology of this neoplasm. In particular, bright sunlight causes exfoliation of the lips—more especially the lower lip. Actinic cheilitis should be considered as a precarcinomatous condition, and 40 per cent. of patients with carcinoma of the lip give a history of recurrent blistering cheilitis.

This is a squamous-celled carcinoma (known for short as an 'S.C.C.' or an 'epithelioma') which presents as a shallow ulcer (fig. 37) with or without the typical everted edge. Thus, any ulcer of the lip which does not readily heal should be viewed with suspicion and a biopsy obtained. With small lesions, diagnosis and treatment can be combined as an excision-biopsy. The lower lip is affected in 93 per cent. of cases, the upper lip in 5 per cent., and 2 per cent. occur at one of the angles of the mouth. Rutherford Morison was the first to point out that a carcinoma occurring at the angle of the mouth and involving *both* lips, however slightly, is far more malignant than the more usual varieties. The reason for this is that the angle of the mouth has a double lymphatic drainage.

Carcinoma of the lip must be distinguished from Molluscum sebaceum (Kerato-acanthoma), described on p. 102.

Treatment.—In its typical form (fig. 530) the carcinoma is comparatively slow-growing and it is eminently curable by surgery or radiotherapy, the five-year survival being 70 per cent. If the lesion is advanced, or has recurred after radiotherapy, adequate surgical treatment necessitates plastic reconstruction.



FIG. 530.—Carcinoma of the lower lip.

Radiotherapy.—After confirmatory biopsy, conventional deep therapy voltage (250 kv.) is used. Ten doses are given over a period of two weeks to a total dose of 5,000 r, the mouth, alveolus, and tongue being protected by lead backing. Radium needles or a mould may be used for buccal or commissural lesions (7,000 r in seven days).

Treatment of Lymph Nodes.—Involvement of the lymph nodes (submental or deep cervical) is relatively late. If the nodes are mobile, the present practice is to perform a classical block dissection (p. 535). If the

nodes are fixed, or the patient is elderly and feeble, operation is contra-indicated. Telecobalt therapy may be of use in these circumstances.

Careful follow-up of cases is essential.

THE FACE

Wounds.—The face has an abundant blood-supply, so not only can the eight-hour limit for primary closure of wounds be extended to twenty-four hours or more, but debridement (excision of ragged edges, tags, and dead tissue) can be reduced to an absolute minimum.



Fig. 531.—Method of avoiding inversion of the skin edges. (After T. P. Kilner.)

The principles of the operation of repair include: (a) careful examination of the wound to find out which layers and structures have been damaged and (b) meticulous repair of each layer. Skin sutures alone, when muscle layers are divided, are *not* enough. The muscular layer is repaired with catgut sutures. The skin is sutured with 3/0 or 4/0 silk or nylon on an atraumatic needle. The skin sutures should be passed so that their points of entrance and exit are near the margins of the wound, but a broad bite of the subcutis is included (fig. 531), in order to avoid inversion of the skin edges.

Wounds of the face are not dressed (exposure treatment, p. 112).

Grazing of the skin with ingraining of dirt particles is also treated by exposure after careful removal of all dirt, using a nail brush if necessary.

Major wounds of the face associated with fractures (facio-maxillary injuries, p. 194) may appear at first sight to be in a hopeless state of mutilation. However, by taking time and trouble (and employing anaesthesia via an endotracheal tube or a tracheostomy), it is nearly always possible to effect a satisfactory repair of all layers—mucosa, muscle, and skin. Should a considerable portion of cheek or lip be lost, the skin is sewn to the mucous membrane, a plastic reconstruction being carried out subsequently.

SPECIAL INFECTIONS OF THE FACE

Boils and 'Pimples' in the region of the 'danger' (mask) area of the face (fig. 532) should never be squeezed, pricked, or incised, for by so doing the infection can reach the cavernous sinus by the venous connections (fig. 533) and cause cavernous sinus thrombosis (fig. 534), a fatal condition in the pre-antibiotic days. The local

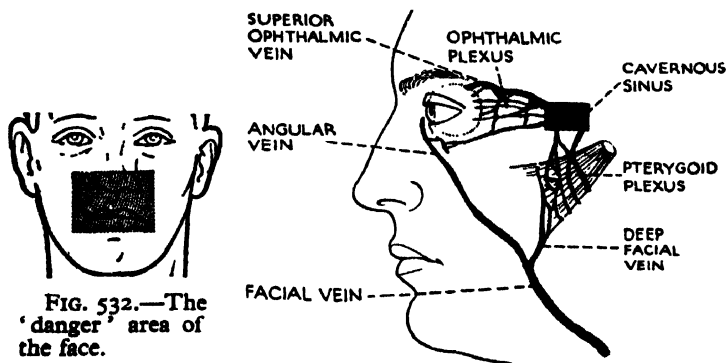


FIG. 532.—The 'danger' area of the face.

FIG. 533.—The cavernous sinus and its connections.



FIG. 534.—Thrombophlebitis of the cavernous sinus secondary to a furuncle of the naris. (P. Reading, F.R.C.S., London.)

treatment of boils and 'pimples' include dressings with magnesium sulphate and glycerin paste, or short-wave diathermy. A broad-spectrum antibiotic is given, with review following the culture of any pus. Massive doses of antibiotics (e.g. 25 mega

penicillin daily) are commenced immediately there are any local signs or general symptoms (rigors) suggestive of cavernous sinus thrombosis, and they are continued (in reduced dosage) for a prolonged period.

Infected Facial 'Cyst' of Dental Origin.—

Silent dental infection can be responsible for producing a chronic subcutaneous abscess of the face. Almost without exception this is diagnosed as an infected epidermoid cyst. What is even worse is that frequently the 'cyst' is excised, and a chronic discharging sinus results. When an example of such a condition as shown in fig. 535 presents, if there is a filled, apparently sound tooth in the vicinity, or a gap of a previous extraction (possibly a root fragment was not removed), the patient should be subjected to dental radiography. When a root abscess is the cause of the facial lesion, extraction of the offending tooth or the responsible remnant thereof will rapidly render other treatment of the facial lesion, other than perhaps a tiny incision, unnecessary.



FIG. 535.—Chronic infected facial 'cyst'. Inset, the causative lesion, showing a chronic abscess attached to a root. (D.C. Bodenham, F.R.C.S.(Edin.), Bristol.)

Anthrax (p. 11).

Lupus (p. 100).

Leprosy (p. 24).

NEOPLASMS OF THE FACE

Benign and malignant neoplasms of the skin of the face are considered in Chapter 7 (Skin), pp. 100 to 110.

THE EAR

Now follows a brief presentation of well-known afflictions of the ear. The reader is referred to the appropriate text-books for fuller accounts and descriptions of operative surgery.

PRETYMPANIC RECESS

Where water collects when bathing.

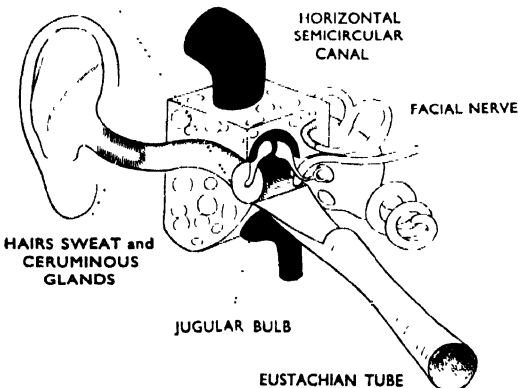


FIG. 536.—Diagram showing the surgical anatomy of the ear.

Surgical Anatomy (fig. 536).—

The ear consists of four parts: (1) Pinna, (2) External auditory canal, (3) Middle ear, (4) Inner ear and Eighth nerve track.

The pinna is cartilaginous, covered by stratified squamous epithelium. The external auditory canal is in two parts: the outer cartilaginous half, which is lined by similar epithelium, tapers to its narrowest part where it joins the bony external auditory canal. This is lined by a thin stratified epithelium containing no glands or hair follicles, and widens medially until closed off by the tympanic membrane at an angle of 60° to the vertical.

The middle ear is part of the mastoid bone. Above it is the attic leading to the mastoid antrum and variable air cells. Below, it communicates with the nasopharynx via the Eustachian tube. It contains (a) part of three ossicles: malleus (handle), incus (anvil), and stapes (stirrup); (b) two muscles—tensor tympani and stapedius; (c) two windows—oval and round; (d) one nerve—chorda tympani. The facial nerve

traverses the superior and posterior walls. The horizontal semicircular canal lies in the attic.

Congenital Abnormalities.—These are: partial or complete absence of pinna; partial or complete occlusion of external auditory canal; partial or complete absence of middle ear—the stapes is nearly always present. Treatment is surgical reconstruction.

EXTERNAL EAR

Wax.—This is best removed by syringing with warm water and bicarbonate of soda solution (1 per cent.) after olive oil has been introduced for the previous two nights (a coffee-spoonful of oil each night massaged into each ear by the patient). An aural syringe with a small nozzle must be used, and the patient's face watched for signs of pain. An intact drum is unlikely to rupture if these rules are followed.

Foreign bodies easily become wedged in the narrow part of the meatus. Beads in children can be removed manually by a loop (e.g. a hairpin) or by syringing.¹ A general anæsthetic may be required. Flies in the ears of motor-cyclists are removed by syringing.

Inflammation.—Inflammation of the external canal may be localised (furuncle) or widespread (external otitis).

Furuncles are staphylococcal in origin; they produce pain, pyrexia, purulent discharge, periauricular swelling, and a painful pinna on movement. Treatment is by local decongestants, e.g. aluminium acetate 7 per cent. (wick or drops). Short-wave diathermy helps. Antibiotics are relatively useless.

External otitis (*syn.* telephonist's ear, Singapore ear) is primarily fungal and secondary to dandruff or athlete's foot. Irritation, discharge, and minimal deafness are the symptoms—secondary infection introduced by scratching the ear leads to further discharge and pain. As vegetable oils inhibit fungi, olive oil drops with a mild antiseptic (e.g. Aurist. Hydrarg. Nit. Dil.²) are indicated. The primary cause, scalp or toes, must be treated at the same time and the ears kept dry. Mild sedation by Tab. Luminal 15–30 mg. (gr. $\frac{1}{4}$ – $\frac{1}{2}$) may be advisable.



FIG. 537.—Carcinoma of the pinna.

Carcinoma of the Pinna (fig.

537) is of the squamous-celled type, and the condition is often comparatively advanced before the patient seeks relief. However, if treated

early, favourable results accrue from excision of a part, or the whole of the pinna. Acrylic and rubber prostheses effectively replace the lost part.

Bony Exostoses.—Bony exostoses of the deep meatus produce a bizarre appearance (fig. 538). Only if the remaining chink past



FIG. 538.—Bony exostoses of the meatus, as seen through the auroscope.

¹ Celsus (A.D. 70) recommended tying the patient on a wooden plank with the affected ear downwards and hitting the plank with a hammer.

² Ung. Hydrarg. Nit. Dil. 3 G. and equal parts Ol. Olivæ and Ol. Amygdali to 30 ml.

them is very small is removal indicated to alleviate deafness. This is a difficult operation and has accounted for many facial palsies.

MIDDLE EAR

Acute otitis media is secondary to upper respiratory infection and presents as a sudden, painful, deaf ear. The temperature is raised and examination shows a red bulging drum.

Treatment.—In the very early cases, antibiotics are given. If they are used, they must be given in *full* doses for five days. Nasal decongestants (e.g. 1 per cent. Ephedrine spray) are given in all cases. If the drum is full and bulging, incision (myringotomy) is necessary (figs. 539 and 540). Otorrhœa is treated either by dry mopping under direct vision with the aid of a head-lamp, or by gentle syringing with sodium bicarbonate solution (1 per cent.), followed by 50 per cent. rectified spirit (S.V.R.) instilled freely forty-eight hours after myringotomy.

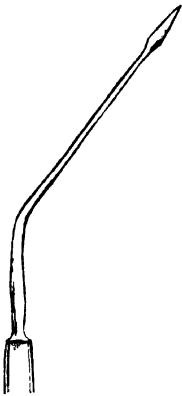


FIG. 539.—Myringotomy knife.

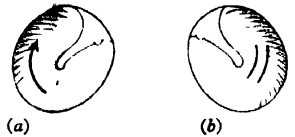


FIG. 540.—Myringotomy incision: (a) correct; (b) incorrect.

Complications.—Inadequate chemotherapy causes latent mastoiditis. Unresolved otitis media is likely to flare up a week or so later as acute mastoiditis.

Acute Mastoiditis.—The symptoms are pain deep in the ear, periauricular swelling, pyrexia. The pinna is not painful on movement (as distinct from a furuncle). Conduction deafness is marked.

Treatment.—Pus must be let out. Therefore a simple mastoidectomy is performed, with an adequate course of antibiotics in support.

The operation entails the removal of the infected air-cells through a posterior auricular incision. The middle ear is not disturbed. The wound is drained for twenty-four hours.

Complications (fig. 541) are lateral sinus thrombosis, extradural abscess, meningitis, cerebral or cerebellar abscess, facial palsy, and, rarely, labyrinthitis.

Chronic otitis media is of silent onset. First symptoms are a foul scanty otorrhœa with increasing deafness. Examination shows subtotal or

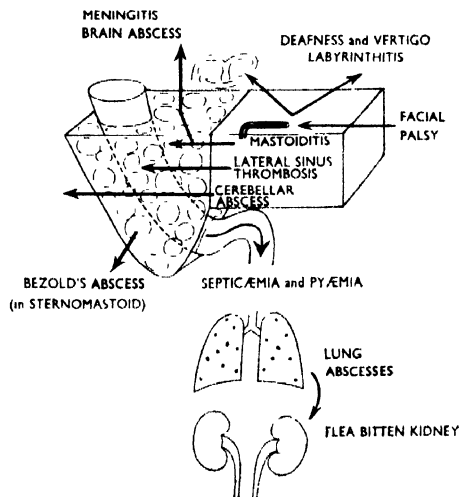


FIG. 541.—The complications of acute mastoiditis.

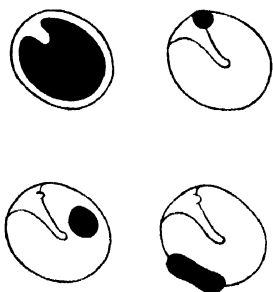


FIG. 542.—Types of perforation of tympanic membrane in chronic otitis media.

attic perforations (fig. 542) with scanty pus and marked conduction deafness.

Treatment centres upon radical mastoidectomy with preservation of the stapes, and reconstruction of the tympanum (tympanoplasty) (fig. 543).

Complications.—Conduction deafness with ossicular chain destruction, labyrinthitis (common), facial palsy, cerebral meningitis, cerebral and cerebellar abscess. A *cholesteatoma* (skinball), whose origin is de-

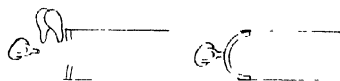


FIG. 543.—Tympanoplasty, using skin.

batable, produces otorrhœa and the above complications by pressure necrosis. **Treatment** is as above, with the removal of all the lining membranes.

Ruptured drum is due to trauma, e.g. hard slap on ear or syringing. There is sudden pain and deafness, with tinnitus and blood-stained discharge.

Treatment.—Do not touch. No eardrops. See the patient daily. No antibiotics required except if infection has been caused by syringing.

Otosclerosis produces bilateral conduction deafness. Common in females, it is often hereditary, and may be exacerbated by pregnancy.

Treatment by hearing aid or operation—stapedectomy with polythene (or wire) strut and vein (or fat) graft (fig. 544). This produces improvement permanently in 90 per cent. and it has replaced the fenestration operation.

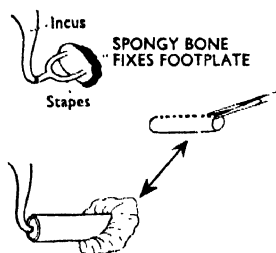


FIG. 544.—Otosclerosis, and stapedectomy, with polythene strut and vein graft.

Secretory otitis is a non-inflammatory fluid transudate into the middle ear, possibly allergic in origin. It is unilateral in adults. Myringotomy without anæsthetic releases the amber fluid and cures the deafness, though fluid may recollect. In children a sterile bilateral effusion may occur, but it can be a sequel of otitis media without myringotomy.

Malignant Tumours.—Excluding the pinna (fig. 537), *squamous-celled carcinoma*, though uncommon, can arise in the middle ear or mastoid cells. There is pain and otorrhœa (which may be long-standing), becoming blood-stained. Deafness is variable. Biopsy will confirm. Treatment is by combined surgery and radiotherapy, but the prognosis is poor.

Glomus jugulare tumours are rare but of special interest as they resemble carotid body tumours. Slow-growing and locally invasive, they are highly vascular, with nests of epithelioid cells and fibrous tissue. Early signs are seventh nerve palsy, pulsatile tinnitus, and free bleeding. Radiotherapy diminishes both size and vascularity.

THE INNER EAR

Presbycusis.—High-tone bilateral perception deafness gradually affecting lower tones with advancing years, often associated with atherosclerosis. Treatment is by a hearing aid.

Cochlear Concussion.—Hearing at 4,000 cycles per second is commonly affected, but if concussion is repeated daily (e.g. gunfire, road-driller, or aircraft pilot not using ear defender), the loss spreads up and down. Failure to hear the telephone bell is often the first symptom.

Ménière's syndrome is unilateral perception deafness, intermittent true rotational vertigo associated with nausea, continuous tinnitus, and headache. It may be associated with hyper- or hypo-tension, intracranial lesions, syphilis, or disseminated sclerosis. But if all these are excluded, and labyrinthine function tests show loss of function on one side, it is true Ménière's disease.

Treatment may be (a) medical—Tab. nicotinic acid 100 mg., t.d.s., Avomine, Stemetil, or (b) surgical—destruction of labyrinth by ablation or ultrasonics.

Eighth nerve tumour causes perception deafness (unilateral), tinnitus, vertigo, severe headache, pain in malar region, and loss of the corneal reflex. A neurosurgeon can remove this via a posterior fossa craniotomy (fig. 496).

Congenital Deafness.—This is due to intrauterine viral toxins in first three months of pregnancy (e.g. rubella, or influenza, etc.).

Prosper Ménière, 1799–1862. Physician, Institute for the Deaf and Dumb, Paris.

CHAPTER 21

THE TEETH AND GUMS. JAWS. NOSE. ORBIT

Congenitally Absent and Supernumerary Teeth.—The third molars and upper second incisors may be absent. Supernumerary teeth sometimes occur in clusters.

Characteristic misshapen teeth that are of diagnostic importance are :

(a) Notched, peg-shaped incisors of Hutchinson, and turret molars of Moon in congenital syphilis (pp. 30 and 236).

(b) Transversely-ridged teeth are not uncommon as a result of deprivation of vitamins C and D while enamel deposition is in progress.

(c) Teeth bespattered with black pits occur in persons who have habitually drunk water containing an excess of fluoride during childhood.

IMPACTION OF A TOOTH

The tooth most often to be affected in this way is the third lower molar (the 'wisdom' tooth). It is prevented from normal eruption by an adjacent tooth. The unerupted portion of the crown is overhung by a pocket or flap of the gum, known as an operculum, viz.

where food debris collects, resulting in infection, which tends to spread to the cheek and sometimes to the neck. Trismus¹ is often present.

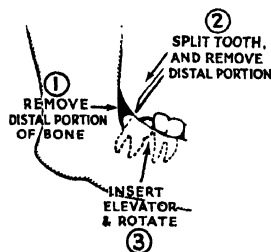


FIG. 545.—Steps in extracting a vertically impacted mandibular molar tooth. (After Professor H. H. Stokes.)

Treatment.—If trismus is severe the patient should be given a general anæsthetic to release the spasm of the muscles of mastication and permit the insertion of a mouth-prop. If an abscess is present beneath the operculum, it is drained. When the acute symptoms have subsided, the impacted tooth should be extracted (fig. 545), unless one of the molar teeth anterior to it is carious. Extraction of



FIG. 546.—Radiograph showing horizontal impaction of third molar. (Dr. Sydney Blackman, London.)

the carious tooth may then afford room for the impacted molar to erupt.

In horizontal impaction (fig. 546), the second molar may be extracted first and the third impacted molar removed by (a) chiselling away the overlying alveolus; (b) dividing the tooth; (c) removing the fragments with an elevator, viz.

HÆMORRHAGE AFTER TOOTH EXTRACTION

Especially in males, it is necessary to exclude hæmophilia (p. 76). As a rule, the bleeding is not due to this cause. Often within the socket there is an open artery that in more accessible situations would have been ligated in the first instance. Quite often the bony alveolus has been fractured, and a loose fragment or a spicule of bone is present, either of which prevents firm clotting within the socket.

Treatment.—Provided the blood pressure is satisfactory the patient should sit bolt upright. A mouth gag is placed in position, the edge of the socket is everted with a pair of dissecting forceps, and with another pair the cavity is mopped with suit-

¹ Trismus—Greek *τρίσμος* = clenching.

able pledgets of cotton-wool. Loose bone or spicules, if present, are removed. Firm pressure with a pledget for a few minutes may stop the hæmorrhage. Should the hæmorrhage come from the gum margin, the flow can be arrested by a stitch passed through the gum on either side of the tooth socket. When the hæmorrhage comes from the depths of the socket many dental surgeons use liquor ferri perchlor. as a styptic, and having packed the cavity, a small roll of gauze, upon which the patient bites, is placed over the socket; a firm four-tailed bandage is applied. Snake venom (Stryphen) is also a good local application.

An absorbable gauze or gelatine sponge can be used in place of the wool pledget. A special gelatine sponge for dental use is manufactured by Allen & Hanburys Ltd. ; it is impregnated with 0.1 per cent. dequadin chloride.

In severe or recurrent cases suture of the gum should be carried out under general anæsthesia. In order that the sutures do not cut out, the sharp edges of the bony alveolus must be removed with nibbling forceps.

ORO-ANTRAL FISTULA

A hole between mouth and antrum via a tooth socket is attributed to the special use of narrow elevators for removing the apex of a tooth (Fickling). A small piece of bone will be seen attached to the apex, air will escape when the patient blows his nose, or a sinogram will confirm the diagnosis.

Treatment.—(1) Immediate suture of the socket, and cover (not pack) by ribbon gauze. (2) If seen later with sinusitis give saline irrigations and parenteral antibiotics. (3) Failure to heal in six to eight weeks requires the operation of inkwell inversion of freed circularly incised mucosa (fig. 547 inset) and cover by a long mucoperiosteal flap (fig. 547).



FIG. 547.—Method of closing an oro-antral fistula. (B. W. Fickling, F.R.C.S., London.)

DENTAL CARIES

Ætiology.—**Diet.**—The teeth of those Esquimaux who live exclusively on fish, flesh, and fowl, and of African pygmies who consume uncooked foods only, are almost free from dental caries. The incidence rises steeply among civilised people whose diet includes carbohydrates in highly refined forms, e.g. white bread.

Acid-producing and Proteolytic Oral Bacteria.—Carbohydrate material clinging to the teeth is subjected to the activity of enzymes liberated by amylolytic oral bacteria: *Lactobacillus acidophilus* is found in association with caries, but the disease has been produced artificially by a streptococcus. A principal end-product of the breakdown of carbohydrates is lactic acid, which is capable of dissolving dental enamel. Once the enamel shell has been penetrated, proteolytic enzymes, generated by other bacteria, disintegrate dentine, which forms the bulk of the tooth (fig. 548).

Minor enamel hypoplasia resulting from lack of vitamins, and possibly from fluoride deficiency in domestic water supplies, is a factor in producing an increased incidence of caries in children (Mellanby).

Clinico-pathological Features.—Dental caries is rife before the age of twenty-five, particularly in early childhood because the deciduous teeth are vulnerable to decay. (Children in England on reaching school age, have an average

of five decayed teeth.) In 90 per cent. of patients the caries starts in the enamel, the hardest tissue in the body. In older people with receding gums, the caries begins in exposed cementum.

The Stages of Dental Decay

Stage 1.—*Erosion of the enamel* is symptomless, and will almost certainly be overlooked unless each tooth is examined systematically with a dental probe.

Stage 2.—*Dissolution of dentine* is also painless until it has progressed to near the

Benjamin William Fickling, Contemporary. Dental Surgeon, St. George's Hospital, London.
Lady May Mellanby, Contemporary. Investigator for the Medical Research Council, London.

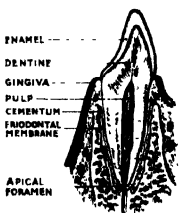


FIG. 548.—Sagittal section through an incisor tooth *in situ*.

pulp, when thermal changes (heat, and especially cold) and mastication of sweets may bring on the familiar pain (toothache). Tapping the tooth causes no pain.

Stage 3.—Part of the crown collapses, due to undermining. The cavity is now more easily discernible on visual examination. In all other respects the symptoms and the signs are the same as those of stage 2, with the addition that pressure of debris in the cavity causes pain; such pain may be referred to a sound tooth on the same side.

Stage 4.—Acute Pulpitis.—Pulpitis may be acute or chronic. In acute cases the pain is throbbing and intermittent. Except in the early stages, thermal changes cause pain. Tapping the tooth is painless, but pressure of a piece of cotton-wool inserted into the cavity is painful. Pulpitis terminates in disintegration of the pulp.

Untreated, acute pulpitis is liable to lead to an alveolar abscess, whereas chronic pulpitis is the harbinger of a periapical abscess.

Dental Caries and the Surgical Patient.—The culmination of caries, a mouth full of broken teeth, stumps and tartar, increases the risk of morbidity from surgical procedures. Pre- and post-operatively there is a likelihood of stomatitis, and, in conditions leading to dehydration (e.g. intestinal obstruction), of acute parotitis. In other cases, mastication at mealtimes will massage bacteria from infected roots into the blood stream, so causing recurrent bacteriæmia which may be detrimental to recovery from any operation.

Thus, special attention must be given to oral hygiene in the pre- and post-operative periods. A dental opinion and the help of a dental hygienist is invaluable, and in non-emergencies dental extraction prior to surgery may be considered. This tends to delay operation by four to six weeks in order that the gums should be soundly healed, so dispersing all chances of bacteriæmia.

During anæsthesia, special care must be taken to prevent loosening or breaking of a tooth, which may be inhaled to cause collapse of a segment or lobe of a lung and then a lung abscess. Thus, prior to anæsthesia a note should be made of the state and numbers of teeth and, if possible, the patient should be warned that a loose tooth is in jeopardy and may require removal for safety. In case of a missing tooth, believed inhaled, an X-ray of the chest, and possibly bronchoscopy, will be required.

ALVEOLAR ABSCESS

While an acute alveolar abscess can occur at any age, it does so most often during childhood and early adult life. Abscesses in the first dentition can

occur in either jaw, while those connected with the second dentition concern particularly the teeth of the lower jaw, more especially a molar tooth.

Pathology.—As a result of acute pulpitis, the putrifying pulp generates gases that exert pressure through the root canal into the osseous tissue around the apex of the tooth. Localised osteitis and abscess formation (gumboil) ensue. Almost always the abscess

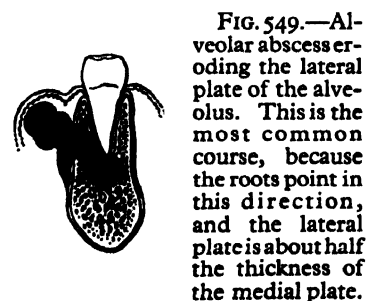


FIG. 549.—Alveolar abscess eroding the lateral plate of the alveolus. This is the most common course, because the roots point in this direction, and the lateral plate is about half the thickness of the medial plate.

points on the labial aspect (fig. 549). One abscess to point lingually is that in relation to the upper lateral incisor, which gives rise to a swelling on the

palate (p. 456), and an abscess from an impacted wisdom tooth can burst through the medial wall of the alveolus and cause the dangerous Ludwig's angina (submandibular cellulitis, p. 527).

If early resolution is not brought about by antibiotic therapy, or if the abscess is not drained promptly and effectively, osteomyelitis of the jaw is to be expected (p. 471).

Clinical Features.—

The pain is dull and constant. Swelling of the cheek (fig. 550) and redness and œdema of the gum in the neighbourhood of the culpable tooth are characteristic signs. The general reaction to the infective process may be considerable, and a tender enlargement of the regional lymph nodes is usual.

Diagnosis and Radiography.—Every swelling of the jaw demands a careful dental and radiological examination. For alveolar abscess a dental cause must be found—caries, an impacted 'wisdom', retained roots, or a dead tooth (p. 468). Only when sufficient time (ten days or more) has elapsed for resorption of bone to occur will rarefaction around the apex of the affected tooth become apparent on X-ray.

Treatment. Drainage and Antibiotics.—Surgical principles must be followed. (1) An abscess must be drained. (2) Antibiotics are not used in place of a drainage operation: they are of value early, before pus has formed, i.e. in the stage of indurated cellulitis, and they are used in support of the drainage operation. Here penicillin is usually employed, to be changed if necessary when the antibiotic sensitivities of the organisms grown from the pus are known.

Physical Agents.—Cold applications externally and hot intra-oral irrigations are useful adjuncts. Hot external poultices are avoided as they promote external pointing which, cosmetically, is to be avoided (fig. 551).

Drainage of an Alveolar Abscess:

(a) *Into the Mouth.*—When a cystic swelling in relation to the gum becomes apparent, the abscess should be opened into the mouth by incising the periosteum (fig. 552). If an alveolar abscess has been opened, ten days should elapse before the dental extraction.

(b) *Drainage by Extraction of the Offending Tooth.*—In the case of the primary dentition this is an accepted and generally successful procedure. In the case of the second dentition gentle and proper dentistry is vital. *General anaesthesia* must be used. Rough dentistry and local anaesthesia engender osteomyelitis, in spite of antibiotic cover.

(c) *Extra-oral Drainage* (seldom required).—When the abscess is pointing externally an external incision must be made, when possible in the shadow of the jaw



FIG. 550.—Extensive alveolar abscess.



FIG. 551.—Sinus following bursting of an alveolar abscess externally.



FIG. 552.—Incising a dento-alveolar subperiosteal abscess.



FIG. 553.—Incision for draining a large mandibular dental abscess.

(fig. 553), by making it $\frac{1}{2}$ inch (1.3 cm.) below and parallel to the lower border of the mandible.

ROOT ABSCESSSES

While an alveolar abscess is usually acute, a root abscess is nearly always chronic. It arises by extension of infection from the pulp through the apical canal, the organisms being for the most part staphylococci and non-hæmolytic streptococci. As a rule there are no symptoms, and no visible signs of a root abscess, which is only revealed by a dental radiograph (fig. 554). The absence of pain, or sensory response to hot or cold stimuli means a *dead tooth*. If so, it will look grey-blue and has an altered percussion note.



FIG. 554.—Dental radiograph showing root abscess of the left lower lateral incisor tooth.

Occasionally a chronic root abscess breaks its confines, and burrows to present externally in the form of a medial mental abscess, sinus (p. 472), or a facial 'cyst' (p. 459), which respond to drainage by extraction of the affected tooth.

THE GUMS

GINGIVITIS AND PYORRHŒA ALVEOLARIS

The periodontal membrane is the periosteum lining the tooth socket. Its fibres are arranged to form a sling for the root (or roots) of a tooth in its bed of bone.

Clinico-pathological Features.—The fundamental predisposing cause of these conditions is gum recession and an excessive deposit of tartar. The tartar pushes the gingival margin away from the teeth with the result that (a) the periodontal membrane is broken, (b) the gum recedes, and (c) particles of food accumulate between the gum and the teeth. Thus inflammation (gingivitis) and suppuration (pyorrhœa) ensues. In *gingivitis*, the gums are swollen, spongy, and bleed readily. In *pyorrhœa*, pus can be expressed from the gingival pockets. Fetor oris may be extreme. The teeth become loose and fall out.

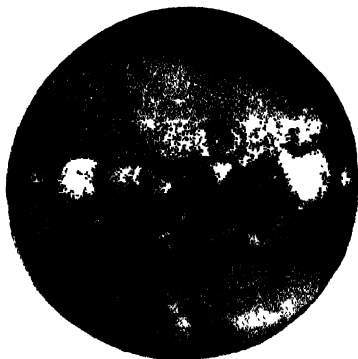


FIG. 555.—The lead line. The patient was a painter, and he was sent to the out-patient department complaining of attacks of colicky abdominal pain.

Gingivitis may be associated with general stomatitis (p. 486), Vincent's stomatitis (p. 488), or be in evidence in scurvy and in chronic mercurial poisoning. In chronic lead, and bismuth poisoning, there is a characteristic narrow line of blue-black dots on the gums near the dental margin (fig. 555).

Treatment.—Comparatively early cases are often benefited by regular scaling, daily massage of the gums with the fingers, removing debris with a wooden toothpick and the frequent use of an astringent mouth-wash, (e.g. ferric

chloride). If this treatment is unsuccessful and the majority of the remaining teeth are sound, gingivectomy (see below) is advised in order to eradicate the periodontal pockets.

When the condition is fully established, cure is unlikely, if not impossible, without extracting the teeth implicated.

Dangers of Dental Extraction in *Pyorrhæa Alveolaris*.—Clearing the jaws of teeth is not to be undertaken lightly in cases of *pyorrhæa alveolaris*. The bacteriæmia that follows is liable to set up broncho-pneumonia, and also may infect any existing endocardial valvular lesion; frequently the onset of subacute bacterial endocarditis dates from dental extraction. Therefore extraction in cases of *pyorrhæa alveolaris* should be carried out only under full antibiotic cover, and the antibiotic, usually penicillin, must be continued for not less than three days. It is advisable for the patient to be admitted to hospital for this period. As a rule, the teeth should be removed a few at a time, and if a major surgical operation—for instance, gastrectomy for peptic ulcer—is contemplated, the operation, when possible, should be postponed until the gums have healed.

HYPERTROPHY OF THE GUMS

Hyperplastic Gingivitis.—Usually first noticed in childhood, the condition persists and often progresses until adequately treated. The patient is a mouth breather, and usually has protuberant front teeth. The gums around the labial aspect of the upper incisors and their inter-dental papillæ are affected most (fig. 556). Bleeding is frequent. On pressure, pus usually oozes from the gingival pockets. In some long-standing cases the hypertrophied gum almost buries the teeth.



FIG. 556.—Hyperplasia of the gums in pregnancy. (Prof. Sir Ernest Bradlaw, Hon. F.R.C.S., London.)

Gingivitis Gravidarum.—Consisting of granulation tissue, the hyperplastic 'tumours' may grow as large as currants and they may require cauterisation or diathermy.

The administration of sodium phenytoin (e.g. Epanutin (Parke Davis)) in the treatment of epilepsy, leads to hyperplasia of the gums in susceptible individuals.

Treatment is gingivectomy or, if a radiograph shows the bone to be hypertrophied, alveolectomy in addition. In advanced cases, post-operative radiotherapy should be given to prevent recurrence.

Localised hypertrophy of the gum due to pressure of an ill-fitting denture is common. Clinically, the condition is indistinguishable from a fibrous epulis. If removal of the cause does not remedy matters in a fortnight, the hypertrophied area should be excised and examined histologically.

GINGIVECTOMY

Indications.—(1) Hypertrophy of the gums; (2) to remove periodontal pockets around otherwise sound teeth.



FIG. 557.—Gingivectomy. The obliquity of the resection promotes drainage and prevents recurrence.

Operation.—Under local anæsthesia, the excess of gum is excised *obliquely* (fig. 557) on both the labial and the lingual sides, together with the interdental papillæ. A cautery arrests bleeding, and a paste (zinc oxide and eugenol¹) is applied to the raw surface and left for a week to prevent granulation and to allow epithelialisation. If

both sides are affected there are three (fortnightly) stages of removal: (a) both left sides as far as the canine teeth; (b) both right sides similarly; (c) the remainder.

¹ Eugenol—an active principle extracted from oil of cloves.

ALVEOLECTOMY

Indications.—(a) Hypertrophy of gums with hypertrophy of alveolus before fitting dentures. (b) Irregularities of alveolus, e.g. after extractions. (c) Sometimes in cases of fibrous epulis.

Operation.—Any remaining teeth are extracted. With a stout scalpel, an incision is made right down

With a stout scalpel, an incision is made right down the bone. Employing a periosteal elevator, a flap is raised on the labial aspect, and a similar one on the palatal aspect. The irregularities of the bone are removed with a hammer and chisel. Any excess of mucosa is trimmed before suturing the cut edges over the new level alveolus (fig. 558).



FIG. 558.—Alveolectomy.

TUMOURS OF THE ALVEOLUS (EPULIS)

'Epulis' is an ancient term with no pathological significance; it merely indicates that there is a solid swelling 'situated on the gum'. There are four varieties of epulides:

1. Granulo-

matous Epu-

lis.—A mass of granulomatous tissue forms around a carious tooth, or at the site of irritation by a denture, requiring extraction of the tooth and scraping away the granulations. A portion must be examined histologically. The granulomatous epulis of pregnancy (*Gingivitis Gravidarum*) is referred to on p. 469.

2. **Fibrous (fibroid) Epulis.**—The commonest form. It is almost always a simple whorled fibroma arising from the periodontal membrane, present under the gum. Rarely a soft, bluish-red, bleeding, and rapidly growing fibrosarcomatous type occurs (fig. 559). They all require removal of the adjacent tooth or teeth and resection of a wedge of bone, including the portion of the gum containing the growth.

3. **Giant-celled ('myeloid') epulis** is an osteoclastoma, arising peripherally in the jaw and presenting under the gum. It is soft, and the overlying gum is sessile and plum-coloured from vascularity.

Ulceration and serious hæmorrhage can occur. X-rays show bone destruction with ridging of the walls (pseudo-trabeculation).

Treatment.—Curettage for small tumours, but radical excision (even requiring a graft for a mandible) for large tumours.

4. **Carcinomatous Epulis.**—An epithelioma of the gum may start around a tooth, or in a socket, causing a painful, infected, and infiltrating lesion which later becomes frankly fungating and ulcerating, invading bone and spreading to cervical lymph nodes. A biopsy must be performed to confirm the diagnosis. Treatment depends upon adequate resection (grafting for mandible, excision of maxilla). Radiotherapy may be used.



FIG. 559.—Fibrosarcomatous epulis.

THE JAWS

A CLASSIFICATION OF JAW SWELLINGS

This is merely for guidance. The epulides, by definition 'swellings of the gum,' are excluded.

- | | | |
|---|-------------------------------|---|
| 1. INFLAMMATORY. | 2. ODONTOMES
(epithelial). | 3. BONE TUMOURS
Benign. |
| (a) Alveolar abscess. | (a) Dental cyst. | (a) The fibro-osseous group. |
| (b) Osteomyelitis. | (b) Dentigerous cyst. | (b) Osteoclastoma, and the
giant-celled reparative
granuloma. |
| (c) Actinomycosis.
(<i>Not specifically of
the jaw but related
to it.</i>) | (c) Adamantinoma. | Malignant.
(a) Osteosarcoma.
(b) Carcinoma of the max-
illary antrum (destroying
bone). |

1. INFLAMMATORY SWELLINGS

(a) **Alveolar Abscess.**—This has been described (p. 466).

(b) **Osteomyelitis of the Jaws.**

This can be acute, subacute, and chronic.

Acute osteomyelitis is rare. In infants it may be a complication of acute specific fevers (measles or scarlet fever), and either the maxilla or mandible may be affected. In other cases infection may spread from the antrum, lachrymal sac, or a dental sac of the first molar tooth. If the maxilla is affected, puffiness of the eyelids occurs (fig. 560). The lateral aspect of the alveolus will be found swollen. A discharging sinus may be seen and pressure over the maxilla will cause pus to run from the nostril and sinus. Radiographs are of no value (Cavanagh). The condition is often mistaken for dacryocystitis.

Treatment.—Though the antrum is small, a polythene tube is introduced by trocar and cannula and the cavity is irrigated with normal saline twice daily, the tube being strapped to the cheek. Appropriate antibiotics are given for seven to ten days.

Subacute Osteomyelitis.—The commonest type, it affects adults and is caused by the spread of apical dental infection or an alveolar abscess. The organism is usually the *Staphylococcus aureus*. For practical purposes the mandible is usually affected, probably because it has a single, rather tenuous blood supply running in the long axis of the bone, which is easily obstructed by infection or trauma. (The condition may follow a fractured jaw, which is almost invariably compound into the mouth.) Obstruction to the blood supply causes bone necrosis, which is the essence of osteomyelitis of the jaw. The maxilla is better protected by a series of vertical arteries with anastomoses. Injudicious extractions performed under local anæsthetic, and a poor general condition (anæmia and under-nutrition), are local and general factors (Wass).

Thus osteomyelitis must be suspected if there is pain, swelling, tenderness after dental extraction or a treated alveolar abscess. There may be a discharging internal or external sinus; this may relieve tension within the bone and so pain is relieved. Increased tension in the dental canal compresses the inferior dental nerve (as well as the vessels) so numbness of the chin in

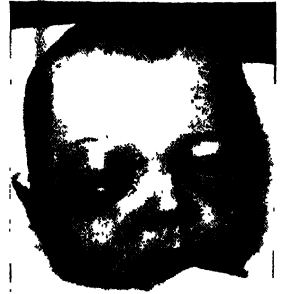


FIG. 560.—Acute osteomyelitis of the maxilla. (Florence Cavanagh, F.R.C.S.(Edin.), Manchester.)

the distribution of the mental nerve is diagnostic. Radiological evidence of bone necrosis takes three weeks or longer to become evident.



FIG. 561.—Osteomyelitis of the lower jaw shortly after a very large sequestrum had been removed.

Differential Diagnosis.—Osteomyelitis must be distinguished from the two other jaw infections—alveolar abscess (p. 466) and, in particular, actinomycosis (p. 22). Sarcoma, though clinically similar, has distinct X-ray appearances.

Treatment.—If osteomyelitis is suspected clinically but not confirmed radiologically, *weekly X-rays* must be taken. As soon as bone necrosis is seen, *operation* is required to relieve tension and remove diseased and dead bone (sequestrum) (fig. 561). *Antibiotics* are given: (a) during the waiting period, providing one is not thereby lulled into a false sense of security (weekly X-rays must continue); (b) as an aid to operative surgery.

Bone recovery (involucrum) is poor. It occurs in young patients, but after middle life there is hardly any regeneration and pathological fracture may occur.

Chronic Osteomyelitis.—Again the mandible is affected more often than the maxilla, and necrosis occurs in a number of ways.

(i) **After Apical Dental Infection, Alveolar Abscess, or Peridental Suppuration.**—Most patients are over fifty years of age. In 50 per cent. of cases dental extraction precedes the onset of symptoms. Many months may elapse before the patient seeks advice. X-ray will show local osteitis with sequestrum formation or a localised abscess (a Brodie's abscess similar to chronic osteomyelitis in long bones).

(ii) **After fractures.**

(iii) **Radiation and Chemical Necrosis.**—Radium and irradiation necrosis, though seemingly non-bacterial, has a large infective element, probably due to the spread of apical dental infection after the vascular thrombosis caused by the irradiation of tumours in the area. There is also a danger of necrosis if extractions are performed after radiation. 'Phossy-jaw' is necrosis due to phosphorus poisoning, rarely seen nowadays, since the crude element is no longer used in the manufacture of matches.

Tuberculous, syphilitic, and actinomycotic necrosis are rare. A hole in the hard palate following a gumma is an example of syphilitic necrosis (p. 456).

Treatment.—Dependent drainage gives the best results. A suitable incision is made beneath the mandible, and a trough is chiselled in the bone, any sequestrum present being removed. The wound is left open and packed lightly with petroleum jelly gauze. Appropriate antibiotics are given.

Median mental sinus (fig. 562) is a clinical entity which is often diagnosed and treated incorrectly. It is produced by a periodontal abscess when pus has tracked between the two halves of the lower jaw to the point of the chin. The patient has a discharging sinus on, or less frequently just below the chin, but always in the mid-line. He usually states that it has been scraped and

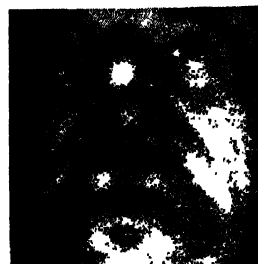


FIG. 562.—Median mental sinus.

packed many times. A radiograph of the bone in the immediate neighbourhood reveals no abnormality, but a radiograph of the lower incisor teeth, which on clinical examination often appear to be sound, shows areas of rarefaction around the roots. After extraction of the affected teeth, the sinus heals within a fortnight.

(c) Actinomycosis

The cervico-facial type of actinomycosis (swelling of the soft tissues, p. 22) has clinical similarities to osteomyelitis of the mandible and must be considered in the differential diagnosis of inflammatory swellings of the jaw. There is swelling of the soft tissues with sinuses, with or without pain. The swelling is more brawny than in osteomyelitis, and the skin is bluish. External sinuses are common, being uncommon in osteomyelitis. 'Sulphur granules' may be seen in the discharge from the sinuses, but their absence, and the inability to grow the streptothrix actinomyces does not rule out this diagnosis. In fact, such a swelling with persistent negative X-ray appearances of the jaw, is considered to be actinomycosis and treated accordingly (p. 23).

2. THE ODONTOMES

Odontomes are cysts, tooth malformations, or tumours arising from the epithelial or mesothelial elements of the teeth, or sometimes from both. Only the three epithelial odontomes are common and important: (a) Dental cyst, (b) Dentigerous cyst, (c) Adamantinoma.

(a) **Dental cyst** occurs in connection with the root of a normally erupted, but chronically infected, usually pulpless, tooth, viz.:—
Epithelial cells, believed to be derived from the enamel-organ, proliferate and degenerate, forming a cyst. The cyst enlarges,

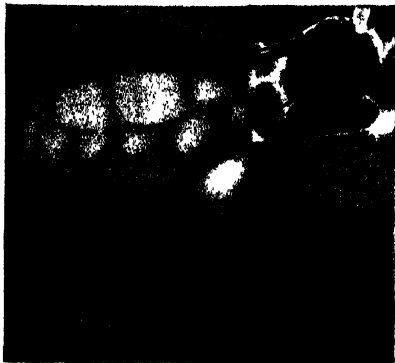


FIG. 563.—Dental (root) cyst expanding the alveolus. Inset, the radiograph.

causing expansion of the alveolus (fig. 563), and eventually most of the epithelial lining disappears. These cysts, which can appear at any age, are more frequent in the upper jaw, and when they attain a large size they encroach upon the antra or the nasal fossæ, but rarely open into these cavities. Usually the fluid in the cyst is clear, and it often contains cholesterol crystals; as a rule it is sterile, but secondary infection can occur. When not infected, the condition is painless.

(b) **Dentigerous cyst** (*syn.* follicular odontome) arises, usually in young adults, in connection with a non-erupted permanent tooth. The swelling consists of a cyst containing a tooth, most commonly an upper or a lower third molar, often well developed except for a truncated root, lying obliquely in a cavity filled with viscid fluid, viz.:—



These cysts expand the outer table of the jaw even to causing egg-shell crackling. The stronger inner table prevents pathological fracture. Ridges of bone on the side walls cause a pseudo-trabecular or a soap-bubble appearance on X-ray.

Treatment of Dental and Dentigerous Cysts.—The essential point in the operative treatment is the excision of the whole epithelial lining of the cyst. After pre-operative dental hygiene, treatment of caries etc., cysts in the upper jaw or small cysts in the lower jaw are exposed through an intra-oral approach to the outer table by raising a muco-periosteal flap. An external approach is used for a large mandibular cyst. After excision the bone cavity is obliterated by a soft tissue 'push-in', or with bone chips, and the wound is sutured.



FIG. 564.—Naso-palatine cyst.

Naso-palatine Cyst (fig. 564).—A rare cyst, arising in connection with the nasopalatine canal and *not* with incisor teeth, which need not be sacrificed (Roper-Hall).

(c) **Adamantinoma** (*syn. multilocular cystic disease; ameloblastoma*) is an epithelial tumour possibly arising from ameloblasts (enamel-forming cells). It is more common in coloured races. It behaves pathologically and looks histologically like a basal-celled carcinoma (p. 106). (Willis believes it arises from the ectodermal epithelium of the stomadæum.) Unlike the other two odontomes just described, it is a locally invasive solid tumour, but undergoing multicentric cystic degeneration. Some of the loculi are separated by true bony trabeculae. An adamantinoma will recur if simply curetted, but it does not metastasise to lymph nodes. It usually occurs in the third decade, nearly always affecting the mandible causing expansion, mostly of the outer table in the region of the molar teeth (fig. 565). It rarely causes pain. The cystic degeneration leads to areas

of softening or egg-shell crackling.

An X-ray shows large loculi, but also has a fine honey-comb appearance which is characteristic and distinguishes it from some dentigerous cysts or an osteoclastoma (fig. 566).

Treatment.—

Evacuation and curettage of the cysts is followed invariably by recurrence. X-ray or radium therapy is without permanent value. Therefore resection of that portion of the jaw bearing the tumour, together with a margin of healthy bone, is essential. Before this is undertaken provision should be made for retention of the remaining portions of the bone in normal relationship by means of dental fixation. Provided no recurrence takes place, after an interval of several months the mandibular defect can be bridged with a bone graft.



FIG. 566.—X-ray of adamantinoma (large loculi and small honey-combing). (H. S. Wass, F.R.C.S., London.)



FIG. 565.—Adamantinoma of the mandible. (A. H. Lendon, F.R.C.S. Adelaide.)

3. BONE TUMOURS OF THE JAWS (BENIGN)

(a). **The Fibro-osseous Group.**—The jaws are membrane bones; there is no cartilage. The 'tumours' of this group are composed of varying proportions of fibrous tissue and bone, being hard if mainly bone, softer, if fibrous tissue, while myxomatous degeneration causes softening in parts. The condition can be localised or diffuse. Thus the variety of types and their descriptive names are legion. For example, a mainly fibrous tumour is called *Monostotic fibrous dysplasia* if localised, or *Polyostotic fibrous dysplasia* if diffuse. If solely of bone and localised, it is an *Ivory osteoma*

(fig. 567) or *Leontiasis ossea* (p. 249) if diffuse. Radiological appearances vary between uniform sclerosis and irregular mottling.

The cause is obscure. Dental sepsis was once thought responsible (*Leontiasis Ossea* being due to a creeping periostitis). The behaviour of the 'tumours' is unlike that of a true neoplasm, for they appear in the first or second decade and usually stop growing when the individual is fully grown. Thus Wass believes that they are aberrations of ossification in membrane. When growth has finished, the unsightly swellings of small local lesions can be removed under antibiotic cover.



FIG. 568.—Paget's disease of the maxillæ. After partial resection of both upper jaws the patient emigrated to New Zealand.



FIG. 567.—Ivory osteoma of the left maxilla. The tumour has been growing slowly for eight years.

Paget's Disease of the Jaws (fig. 568).—This condition can be part of a generalised Paget's disease, or confined mainly to the jaws. It may be confused with that diffuse bony variety of the fibro-osseous group, *leontiasis ossea*, but it occurs in older people, pain is often present, other bones are affected, and the radiological appearances of the teeth (if there are any) show apical club-shaped swellings (cementosis). As the need arises the alveolus and, if necessary, the anterior surface of the maxilla, is reduced in size by chiselling away the excessive bone formation.

Complication.—Sarcoma develops more often than is the case in other bones affected with Paget's disease.

(b) **Osteoclastoma and Giant-celled Reparative Granuloma.**—Both these conditions are rare. *Osteoclastoma*, besides occurring under the gum as a myeloid epulis (p. 257), can

expand bone, though Wass encountered only one instance (in the maxilla) out of 350 jaw tumours.

Giant-celled reparative granuloma is often mistaken for an osteoclastoma (Jaffe). The lesion appears to be related to the occurrence of a hæmorrhage within the bone marrow. Somewhat rare, it occurs more frequently in females than in males, and nearly always between the ages of ten and twenty-five years, whereas a *bona fide* giant-celled tumour appears in patients between twenty-five and forty years of age. The lesion causes a painless swelling of the jaw, situated much more frequently in the mandible than in the maxilla.

Radiography.—There is a rounded or oval area of radio-translucency (fig. 569). The granuloma thins and expands, but does not perforate the cortex.

Pathology.—Multinuclear cells are small, sparse, and distributed unevenly. Histologically it is difficult or impossible to distinguish this lesion from the so-called brown tumour of hyperparathyroidism.

Differential Diagnosis.—Hyperparathyroidism should be excluded by ascertaining the serum calcium level and by radiography of other parts of the skeleton (p. 571). It is also important to exclude an adamantinoma (p. 474), for the treatment of the latter condition must be more radical. At operation a giant-celled granuloma will be found to consist of opaque, semi-solid, dark red material, whereas an adamantinoma is filled with transparent fluid or jelly.

Treatment.—The treatment of choice is thorough curettage through an external incision without opening the bone cavity into the mouth. Should the condition recur, hyperparathyroidism must again be excluded.



FIG. 569.—Radiograph showing a giant-celled reparative granuloma of the mandible. (Dr. H. L. Jaffe, New York.)

3. TUMOURS OF THE JAWS (MALIGNANT)

Malignant Neoplasms of the Maxilla.—The clinical diagnosis of 'malignant upper jaw' embraces a number of pathological conditions.

(a) **Osteogenic sarcoma** is usually of the round-celled variety; rarely it is a highly differentiated fibro-myxochondro-sarcoma.

(b) **Columnar-celled carcinoma**—of the maxillary antrum (fig. 571).

(c) **Squamous-celled carcinoma**—derived from the epithelium overlying the hard palate, from a tooth socket, or from the gum.

(d) **Invasion of the maxilla by a sarcoma of the ethmoid.**

(e) **Malignant Lymphoma of Africa (Burkitt Tumour)** (p. 158).

(a) **Osteogenic Sarcoma.**—While neither sex nor any age is exempt, this disease has a curious predilection for women about the age of forty (fig. 570). When of the periosteal variety—and this is the more usual—it is the *anterior* aspect of the jaw which is maximally affected, but the condition soon shows itself on the inferior or palatal wall as well. Pain, nasal obstruction, and epiphora¹ usually occur late in the disease.

(b) **Carcinoma commencing in the maxillary antrum** is much more frequent than the foregoing, and the age incidence of this group is similar to that of carcinomas elsewhere. These neoplasms are divided about equally between the sexes, and most cases are associated with secondary infection of the nasal sinuses. Growths that originate on the floor of the antrum frequently cause pain in the teeth, and a dental surgeon is consulted, but without relief of the pain; however, pain usually results in a comparatively early diagnosis. A foul, purulent and blood-stained discharge from the nose is common. Free bleeding associated with antral puncture should always

suggest the possibility of carcinoma of the antrum. The main clinical features vary in accordance with the direction in which the tumour spreads:

If posteriorly, there is little if any alteration in contour of the face.

If the medial wall is implicated, there will be nasal obstruction and epiphora.

If the antero-lateral wall is involved, there will be evident swelling of the face (fig. 571).

If the floor of the antrum is transgressed, the hard palate will bulge.

If the roof is invaded, there will be proptosis and diplopia.

Metastasis occurs relatively late, the upper jugular lymph nodes being the first to be affected, giving rise to a swelling beneath the angle of the jaw. A third of the cases have metastases when they are first seen.

Radiography.—In the early stages there is opacity and increase in size of the antrum. Later decalcification of the bony walls is visible.

¹ Epiphora—Greek, *ἐπιψορά* = overflow. An abnormal overflow of tears down the cheek usually due to obstruction of the lachrymal duct.

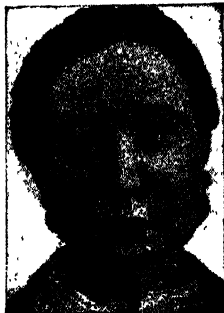


FIG. 570.—Sarcoma of the left maxilla. (The late J. A. C. Macewen, Glasgow.)



FIG. 571.—Carcinoma commencing in the maxillary antrum.

Biopsy.—All nasal polypi should be submitted to microscopical examination. Unfortunately cases in which polypi are present are comparatively few. Fragments of the neoplasm may be washed out by antral lavage and solid matter, after centrifuging, should be examined microscopically. As a rule, a diagnostic Caldwell-Luc operation (p. 479) is necessary to obtain biopsy material, and as histological examination is a cardinal step in diagnosis, no effort should be spared in obtaining a representative specimen.

Treatment.—Collaboration between surgeon and radiotherapist is essential.

Combined Irradiation and Conservative Surgery for Carcinoma of the Maxilla.—In several clinics the following plan is adopted:

(a) A course of high-voltage irradiation or gamma rays from a teleradium unit is given.

(b) Six to eight weeks after the conclusion of the irradiation, by which time the inflammatory reaction has abated, palatal antrostomy (fig. 572) is performed. The purpose is to remove any residual irradiated growth and to discover whether and where any viable malignant tissue remains by taking a series of geographic biopsies. It also allows easy follow-up inspection of the cavity.

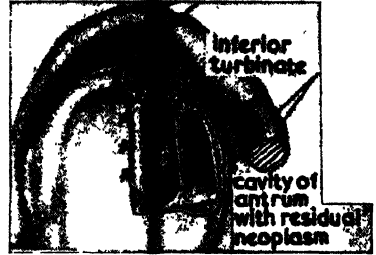


FIG. 572.—Palatal antrostomy.
(After C. P. Wilson, F.R.C.S., London.)

The plate of bone forming the antro-nasal wall is nibbled away. The inferior and middle turbinate bones and the tissue involved in the growth are removed with a diathermy needle, if possible in one piece; doubtful areas are coagulated. A pack or plastic obturator is inserted. The patient is given antibiotic therapy for a week.



FIG. 573.—Trotter's incision for removal of the upper jaw. Oedema of the lower eyelid is avoided by carrying the incision through the eyelid just outside the punctum lacrymale, along the orbital margin inside the lid, and thence as shown avoiding the outer canthus.

(c) In 30 per cent. of cases the tissue removed shows no evidence of malignancy, and nothing more is required. When the tissue removed at operation shows evidence of active malignancy a hollow plastic applicator, made by a dental surgeon, is filled with wax and drilled so as to take radium tubes. In this way further irradiation is given. If the post-ethmoidal or sphenoidal regions are involved, radium needles are inserted directly into those areas.

(d) A block dissection of the neck is undertaken if lymph nodes are palpable (p. 535).

(e) A permanent obturator bearing teeth is fitted at least three months after operation.



FIG. 574.—Fergusson's lion forceps.

Excision of the Maxilla.—Even in the pre-anæsthetic days complete excision of the upper jaw (fig. 573) was practised. It was for this purpose that Fergusson invented lion forceps (fig. 574). Since the introduction of intratracheal anæsthesia the operative mortality

Wilfred Trotter, 1872-1939. Surgeon, University College Hospital, London.
Sir William Fergusson, 1808-1877. Surgeon, Royal Infirmary, Edinburgh, later Professor of Surgery, King's College Hospital, London. Performed 134 cleft-palate operations with only five failures.

is low. There is surprisingly little deformity after this formidable operation, especially if a prosthesis is constructed by a dental expert as soon as the wound has granulated. Such treatment offers hope of a cure (fig. 575). Whether surgical excision should precede or follow irradiation is still undecided, as comparable results are obtained by either method (Maxwell Ellis).



FIG. 575.—Patient two years after excision of the upper jaw and the eyeball for a carcinoma arising from the antrum and invading the orbit. Three years later he was at work as a night watchman.

If recurrences occur they may be treated by diathermy excision, intracavity irradiation or infusion with cytotoxic drugs (p. 631). The overall five-year survival rate is 35 to 50 per cent.

MALIGNANT NEOPLASMS OF THE MANDIBLE

The mandible is rarely the site of a primary malignant neoplasm or of a metastasis. However, rather frequently it becomes directly involved by an advancing primary carcinoma of the tongue or of the floor of the mouth. Sometimes an advanced carcinoma of the lip spreads to this bone through the mental foramen. A third method of neoplastic involvement is occasioned by the relation of the facial lymph node which lies in juxtaposition to the mandible near the groove on this bone for the facial artery. Should this inconstant node be the seat of a secondary deposit, it tends to invade the mandible.

Treatment.—So regularly does irradiation cause necrosis of the mandible, that when the bone is involved by growth, it is usually necessary to resect a portion or perform hemi-mandibulectomy. If the chin itself can be spared, gross deformity is avoided.

SARCOMA OF THE ETHMOID

Sarcoma of the ethmoid is rare. As it expands it widens the space between the orbits and flattens the nasal bones, producing that clinical entity, the 'frog-faced man'. Still later the superior maxillæ are invaded.

Treatment.—The treatment by combined irradiation and operation does not differ in principle from that described for carcinoma of the maxillary antrum. When the air sinus is infected, drainage is essential before irradiation is commenced.

INFECTION OF THE MAXILLARY ANTRUM

Surgical Anatomy.—The maxillary antrum (of Highmore) is rudimentary at birth and attains full development by the age of twelve years. Lined with ciliated epithelium, it communicates with the middle meatus of the nose by a small ostium situated high on its medial wall (fig. 576). The apices of the roots of the second premolar and the first and second molar teeth are in close apposition to the floor of the antrum being separated only by periosteum and mucous membrane. Rarely, the first premolar and the canine teeth are related similarly.

Maxillary sinusitis may be unilateral or, much less frequently, bilateral. Infection occurs as an extension from the nose. One epidemic of acute respiratory infection (common 'cold') brings as an aftermath many cases of infection

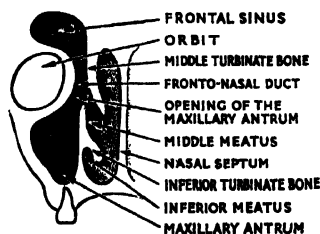


FIG. 576.—Coronal section of the maxillary antrum.

of the accessory nasal sinuses, of which the maxillary antrum always heads the list ; while another epidemic, in all other respects similar, is free from this complication. A less frequent cause is penetration of the floor of the antrum by bacteria from a periodontal abscess connected with a carious tooth of the anatomical variety described above.

Acute Empyema Antri.—As a rule the general symptoms are severe, especially when the pus is confined by occlusion of the natural ostium. Pain and tenderness are present over the affected maxilla ; sometimes the pain is referred along one of the divisions of the trigeminal nerve. Frequently the affected side of the face is swollen and the lower eyelid is suffused. Breathing through the nostril on the side of the lesion is impaired and often obstructed. If the ostium is patent, which is unusual in acute cases, a flow of pus can be obtained when the head is held downwards and forwards with the affected side uppermost.

Transillumination of the antrum and a radiograph (fig. 577) are likely to reveal a relative opacity of the affected antrum.

Treatment.—This includes bed rest, analgesics, Ephedrine 1 per cent. in saline by a nebulizer, menthol inhalations, and systemic chemotherapy if the temperature is over 100°F. Puncturing the antrum is for diagnostic and therapeutic purposes. An antral trocar and cannula punctures the antrum

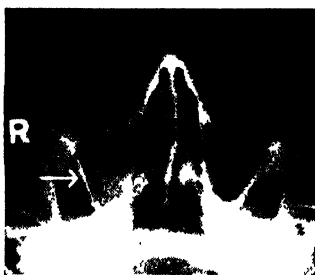


FIG. 577.—Radiograph showing pus in the right antrum of Highmore.



FIG. 578.—Pus exuding from beneath the middle turbinate.

beneath the inferior turbinate bone after application of surface anæsthesia. Pus is aspirated and a washout performed with normal saline. Ultra-shortwave diathermy is helpful in some cases.

Chronic Empyema Antri.—Pain and swelling are often absent, and frequently the only symptom is an intermittent flow of pus from the nostril on the affected side. When the ostium is patent, the information gained by an examination with a nasal speculum may be diagnostic (fig. 578).

Treatment.—Purulent maxillary sinusitis not responding to three weeks' trial of irrigation warrants adequate drainage:

Caldwell-Luc Operation.—This is a good standard procedure, though not advocated in children as dentition may be affected. Under general anæsthesia, with oral intubation and packing, an intra-oral incision one inch long is made in the buccolabial sulcus, centred over the canine fossa. The muco-periosteum is elevated and a gouge is used to make an opening about half an inch in size through the anterior wall of the antrum. All the mucous membrane is removed with Luc's forceps (fig. 579)

George Walter Caldwell, 1834–1918. Practised otorhinolaryngology in New York, San Francisco and Los Angeles. Henri Luc, 1855–1926. Otolaryngologist, Paris. His clinic was visited by otorhinolaryngologists from all countries.

and any diseased ethmoid cells can be exenterated. An intranasal antrostomy completes the procedure, allowing post-operative washouts.

Intranasal Antrostomy is performed in children in order to avoid affecting dentition, and as an operation of compromise in some patients with recurrent sinusitis. Under a general anæsthetic, a piece of the medial wall of the antrum is removed to allow drainage into the nose.

Nasal Polypi.—Nasal polypi (œdematous ethmoid sinus mucosa) are usually multiple and are found prolapsing from the ethmoid cells in the region of the middle turbinate (fig. 576), and are recognised by their glistening gelatinous appearance when light is focused upon them.

They are practically confined to adults, and patients complain of nasal obstruction, nasal discharge, and some loss of smell. Many of the sufferers are allergic to dusts and pollens that initiate in them attacks of sneezing and acute rhinitis.

Treatment.—Nasal polypi together with pieces of carious ethmoid are removed under surface anæsthesia with Luc's forceps (fig. 579). If sinusitis is present the underlying cause must be eradicated also.

Antihistamines should be used after removal, since recurrence is common.

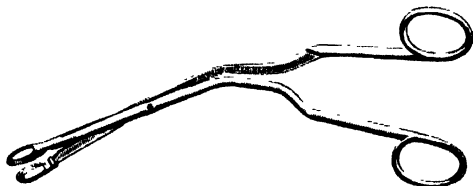


FIG. 579.—Luc's ethmoidal forceps.

times varicose) situated in Little's area (fig. 580) on the septum. The most frequent cause is nose-picking—epistaxis digitorum. Other causes include trauma, nasal infection, neoplasms, hypertension, blood dyscrasias, acute specific fevers, nephritis and uræmia.

Treatment.—Sit the patient up so that blood does not run down the throat. Blow nose to remove clots. Pinch the nose for ten minutes. An ice-pack applied to the nose may be helpful.

Sedation with morphine and admission to hospital are necessary if bleeding is uncontrolled. Blood transfusion may be required. If the patient is hypertensive, some loss of blood is beneficial.

Cauterising the bleeding-point with trichloroacetic acid, 50 per cent., or electric cautery, is undertaken if the bleeding-point can be seen.

Packing: Anterior Packing.—After anæsthetising the mucous membrane with 4 per cent. xylocaine, ribbon gauze saturated in liquid paraffin is inserted so as to fill the nasal cavity, and if the bleeding is from the septum the nasal cavity of the opposite side is packed also. Penicillin is administered to combat infection.



FIG. 581.—Method of inserting a posterior nasal tampon (Dickie's method).

Posterior Packing.—The insertion of a cone-shaped gauze tampon (fig. 581), moistened with liquid paraffin is the most satisfactory method because it minimises complications (e.g. hæmotympanum). The pack can be left in for five days if an antibiotic is administered.

Arterial Ligation.—When the above methods fail, or profuse hæmorrhage recurs and it is likely that the source of the bleeding is more posterior, ligation of the

EPISTAXIS

Source and Cause of the Bleeding.—The bleeding may be arterial or venous. In 90 per cent. of cases it comes from a plexus of veins (some-

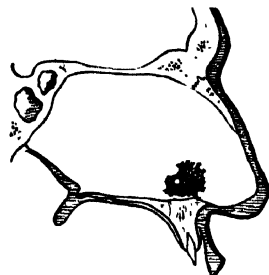


FIG. 580.—Little's area.

anterior ethmoidal artery in the orbit is indicated, particularly in traumatic cases (fig. 582). Trans-antral ligation of the internal maxillary artery, and ligation of the external carotid artery are other methods which are sometimes called upon.

DEFLECTED NASAL SEPTUM

Although the majority of adults do not have a completely straight septum, it is only the grosser deflections causing symptoms which require correction. Septal deformities are caused either by trauma or by disproportionate growth during development. They may produce nasal obstruction or headaches in the region of the nasion.

The submucous resection (S.M.R.) operation consists of the removal of cartilaginous and bony spurs from between the two coverings of mucoperichondrium and mucoperiosteum. This allows the septum to take a more mid-line position.

Nasal Bone Fractures (p. 195).



FIG. 582.—Incision for the approach to the anterior ethmoidal artery.

CYSTS ABOUT THE ORBIT

From the point of view of clinical surgery it is convenient to group together certain conditions which have but one point in common—they form cystic swellings in the neighbourhood of the orbital margin. Passing latero-medially the following cystic swellings can be recognised :



FIG. 583.—External angular dermoid.

1. **External Angular Dermoid.**—This is perhaps the most common situation for a dermoid cyst (fig. 583), and its position is so constant that it allows the diagnosis to be made with irrefutable accuracy. In the course of time, by constant pressure, the cyst causes a depression in the bone on which it lies. Some external angular dermoid cysts are dumb-bell-shaped, with the deeper component lying within the orbit. Like the superficial, the

deeper component causes decalcification of the bone on which it lies (the orbital plate of the frontal bone), so that this thin plate becomes eroded and the cyst comes to lie against the dura, to which it becomes varying adherent. The treatment is excision.

2. **Meibomian cyst** usually occurs in the upper eyelid (fig. 584). It is due to a staphylococcal infection of a Meibomian gland, the swelling mainly being retained Meibomian secretion. It must be distinguished from a



FIG. 584.—A Meibomian cyst.



FIG. 585.—Hordeolum (stye).

hordeolum (stye) (fig. 585), which is due to infection of an eyelash follicle. The treatment consists in making an incision into the cyst from its conjunctival aspect, at right angles to the margin of the lid, and scraping out the sac wall with a small sharp curette.

Heinrich Meibom, 1638–1700. Professor of Medicine, Helmstädt. Resigned the Chair for that of History and Poetic Art.

3. **Mucocele of the lachrymal sac** (fig. 586) is the result of lachrymal obstruction with distension of the sac and secondary infection (dacryocystitis). The treatment consists in washing out the sac by means of a lachrymal syringe and removing the cause of the obstruction.



FIG. 586.—A mucocele of the lachrymal sac. (Dr. Peter Hansell, London.)

4. **Cyst over the Root of the Nose.**—If the swelling is beneath the skin and does not empty with pressure, it is almost certainly a dermoid cyst (fig. 587). If it can be made to empty, a meningocele is probable, but a sinus pericranii, connected with one of the intracranial venous sinuses, must also receive due diagnostic consideration. Especially when the swelling is not strictly median, the possibility of a mucocele of the frontal sinus or ethmoidal cells should be borne in mind.



FIG. 587. — Dermoid cyst at the root of the

TUMOURS OF THE ORBIT

Tumours of the orbit are primary or secondary. The principal primary tumours are :

1. **Osteoma.**—It will be recalled that ivory osteomata grow from membrane bones; in the orbit particularly from the lachrymal bone.
2. **Mixed Tumour** (occasionally a carcinoma) of the lachrymal gland (fig. 588).



FIG. 588.—Mixed tumour of the lachrymal gland. (Professor Charles Saint, Cape Town.)



FIG. 589.—Glioma of the optic chiasma. (J. Minnion, F.R.C.S., London.)



FIG. 590.—Sarcoma of the orbit. (Professor A. A. McConnell, Dublin.)

3. **Glioma** (Astrocytoma) of the optic chiasma (fig. 589) or, rarely, of the optic nerve.

4. **Sarcoma** (fig. 590).

Clinical Features.—Proptosis is the cardinal symptom. The eye is seldom pushed directly forward except when the tumour is growing from the optic nerve or its sheath. Visual disturbances are complained of, and the lids are prone to become oedematous. In advanced cases the cornea becomes inflamed (keratitis) or ulcerated because it is unduly exposed.

Treatment is excision when practicable, followed, in the case of the malignant tumours, by irradiation.

Secondary tumours of the orbit are uncommon. A well-known example is a metastasis from a neuroblastoma of the adrenal gland (p. 584). Frequently the orbit is invaded by a primary carcinoma of the antrum, by a very advanced rodent ulcer, and also by a carcinoma of the ethmoid.

INTRA-OCULAR TUMOURS $\left\{ \begin{array}{l} \text{Occurring in Children} = \text{Retinoblastoma} \\ \text{Occurring in Adults} = \text{Melanoma} \end{array} \right.$

Retinoblastoma originates in foetal undifferentiated retinal cells that tend, in the tumour, to imitate rods and cones. Most retinoblastomas are unilateral and sporadic, but in 4 per cent. of cases the affliction is transmitted to the offspring. When retinoblastoma is transmitted by a parent with one healthy eye, both eyes of the offspring commonly show the lesions (Sorsby). Doubtless present in very early infancy, a retinoblastoma remains latent until the age of about two years, when it grows rapidly; very few cases occur after the age of six. In 25 per cent. of cases the tumour is bilateral. As a rule the neoplasm arises in the posterior third of the retina, and tends to grow into the optic nerve. Metastases occur, but not so early as in cases of intra-ocular melanoma; usually they are located in the cranial bones and the lungs. The parents may bring the child for advice because they notice the baby has a dilated pupil and peculiar 'cat's-eye' reflections in the affected eye. Later proptosis occurs, and early blindness is the rule. Untreated, this highly malignant tumour soon breaks down and bleeds. When increased intra-ocular pressure occurs in a child, the pressure is distributed equally in all directions and the sclera and cornea bulge, producing what is known as 'buphthalmos' (ox eye). If the diagnosis can be made reasonably early, the treatment is excision of the eye with as much of the optic nerve as possible, followed by deep X-ray therapy. Because of intracranial extension along the optic nerve, recurrence after enucleation occurs in about 50 per cent. of cases, in which event death results usually within a year. If the patient is free from recurrence for four years, a permanent cure is practically certain.

Melanoma is the most important intra-ocular tumour of adults. Usually it originates in the posterior part of the choroid, but it may arise in the ciliary body or the iris. Always unilateral, it grows as a disc-like mass until it breaks through the elastic membrane covering the choroid, when it spreads beneath the retina in a mushroom-like manner, often causing retinal detachment. Very occasionally a melanoma of the eye remains small and seemingly benign, but most of these tumours are highly malignant. Malignant intra-ocular melanoma shows comparatively little tendency to spread into the orbit, but metastases are carried especially to the liver (fig. 591) where they sometimes grow to an immense size, even weighing 20 lb. (9 Kg.). The patient is nearly always between forty-five and sixty years of age. When present, a characteristic early symptom is a fixed black spot in the visual field, but the most frequent symptoms are those of retinal detachment. There is no pain until secondary glaucoma has set in, by which time the tumour has reached a considerable size. If the globe is removed early in the course of the disease, dissemination may be limited, but rarely is the expectation of life more than three years. Sometimes, after early diagnosis and excision of the globe, the metastasis is delayed for many years, and then arises a classical pitfall for the unwary diagnostician. It has been remarked, with much wisdom, that the

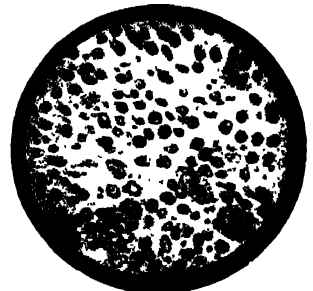


FIG. 591.—Section of liver showing secondary melanoma. The primary growth was intra-ocular.

clinician should "beware the patient with a large liver and a glass eye" (fig. 592). A curious unexplained fact is that the five-year survival rate after excision of the eye is higher in women than in men.



FIG. 592.—Patient with a greatly enlarged liver, who for many years had worn a glass eye.

PULSATING PROPTOSIS

Unilateral pulsating proptosis is a rare condition that always excites clinical interest. The principal causes are as follows:

1. An arterio-venous aneurism between the internal carotid artery and the cavernous sinus (fig. 593); usually traumatic in origin.
2. An aneurism of the ophthalmic artery.
3. A cirroid aneurism involving the orbit.
4. Thrombosis of the cavernous sinus. However, pulsation is not a usual feature of this condition.
5. A rapidly growing vascular intra-orbital neoplasm.

Clinical Features.—In the first three conditions the patient complains of impaired or blurred vision, always on the side of the lesion, and a buzzing noise in the head. Retro-ocular pain and headache are also common manifestations. The signs include proptosis, bruit, congestion of the retinal veins, pulsation of the globe, conjunctival chemosis, and venous dilatation. Glaucoma is a common complication, and results from venous back pressure.



FIG. 593.—Pulsating proptosis due to an intracranial arterio-venous aneurism.

Investigations.—In addition to neurological and ophthalmological examinations, X-rays of the skull are necessary to seek a fracture involving the cavernous sinus area. Bilateral carotid angiography may be helpful.

Treatment.—The first variety, which is the most common, is the most amenable to treatment. Ligation of the orbital veins is often successful. Adson recommended two small incisions, one over the inner canthus (angular vein) and one under the inner end of the eyebrow (superior ophthalmic vein). Loops of the veins are freed and segments of them are resected between ligatures. When this fails, ligation of the common carotid artery is resorted to. These combined measures cure about two-thirds of the patients belonging to the first three groups. Should this fail, the advisability of ligation of the internal carotid artery must receive full consideration (p. 129).

ORBITAL CELLULITIS

Cellulitis of the orbit (fig. 594) gives rise to proptosis, œdema of the eyelids, and œdema of the conjunctiva (chemosis). Much the most frequent cause of the condition is a spread of infection from one of the paranasal sinuses. The constitutional symptoms are often severe and there are two

outstanding dangers. Firstly, thrombophlebitis of the cavernous sinus may follow via the ophthalmic plexus of veins, and, secondly, the globe of the eye may become infected.

Treatment.—In early cases, antibiotic therapy, which should be instituted at once, sometimes results in resolution of the infection. Unless full response is undoubted, an incision along the inferior orbital margin followed by blunt-nosed forceps passed into the intra-orbital fat should not be delayed; this will relieve tension in the orbit and provide drainage.

INJURIES OF THE EYE

Injuries of the eye belong properly to the domain of ophthalmology. It is necessary here to call attention to a peculiar danger of perforating wounds involving the lens. Leakage of the protein of the lens, hitherto isolated from the body by the lens capsule since foetal life, sets up an auto-immune reaction in the sound eye (*sympathetic ophthalmitis*). When this occurs, the sight of both eyes may be lost. The only certain way of avoiding sympathetic ophthalmitis is to excise at once a wounded eye in cases where there is no perception to light.



FIG. 594.—Orbital cellulitis.

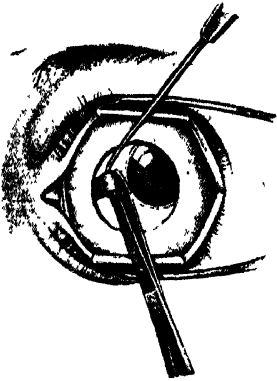


FIG. 595.—Excision of an eyeball. Medial rectus tendon being divided, aided by a strabismus hook.

Excision of an Eyeball: *Indications include:*

Trauma, Iridocyclitis, Neoplasms, Anterior staphylococcal infection, Irremediable glaucoma.

The Operation.—The speculum is introduced between the lids, and opened. The conjunctiva is picked up with toothed forceps and divided completely all round as near as possible to the cornea. Tenon's capsule is entered, and each of the rectus tendons is hooked up on a strabismus hook and divided close to the sclerotic (fig. 595). The speculum is now pressed backwards and the eyeball projects forward. Blunt scissors, curved on the flat, are insinuated on the inner side of the globe, and these are used to sever the optic nerve. The eyeball can now be drawn forward with the fingers, and the oblique muscles, together with any other strands of tissue which are still attaching the globe to the orbit, are divided. A swab moistened with 1 : 1,000 adrenalin and pressed into the orbit will control the hæmorrhage.

Evisceration of the Eyeball.—Owing to the danger of opening up lymphatic spaces at the back of the globe and thus favouring meningitis, evisceration is to be preferred to excision in panophthalmitis. The sclera is transfixated with a pointed knife a little behind the corneo-sclerotic junction, and the cornea is removed entirely by completing the encircling incision in the sclera. The contents of the globe are then removed with a curette, care being exercised to remove all the uveal tract. At the end of the operation the interior must appear perfectly white.

CHAPTER 22

THE MOUTH, THE TONGUE, THE CHEEK,
AND THE SALIVARY GLANDS

INFECTIONS

Stomatitis is a general term that embraces all forms of diffuse inflammation of the mouth.

The inflammation may be caused by an injury that is mechanical, thermal, chemical, or by radiotherapy. Secondary to this injury, or sometimes when no obvious injury can be detected, infection occurs. The mucosa may suffer damage by malnutrition of many different types, in which case it usually becomes atrophic. This, although not strictly speaking an inflammation, is often described in such terms as stomatitis, angular stomatitis, glossitis, etc. The atrophic and mal-nourished mucosa less readily withstands trauma and infection. Such conditions are seen in the Vitamin B deficiencies, in sprue, in protein malnutrition (kwashiorkor¹), in pernicious anæmia and in the iron deficiency anæmias.

The Infecting Organisms.—Numerous organisms may be found in the mouths of healthy persons and in disease. These can be divided into the following categories:

1. *True Saprophytes*.—These are not pathogenic, and are found in man's normal environment—in the air, in plants, in dust, etc., and arrive in his mouth as it were by accident and are never the cause of disease.

2. *True Pathogens*.—These cause human diseases.

3. *Facultative Pathogens*.—There are a very large number of organisms indigenous to man not found in his mineral or vegetable environment. They are transmitted from man to man, and may in some cases be found in animals. Such organisms often inhabit healthy mouths but may give rise to, or be found in, many human pathological lesions.

Quite clearly then, the varieties and variations of stomatitis are innumerable, and only a few of the more important conditions are described below.

Traumatic Stomatitis.—A simple cut or burn of the mouth usually heals rapidly. It is quickly covered by a thin grey glistening coagulum. Salivation is increased, but because the mouth is painful, movements are restricted and fur collects on the tongue. Because of the stagnation the breath becomes foetid. In healthy persons no treatment is required, but frequent rinsing of the mouth with a warm carbolic mouth wash (1:1000) or glycerin of thymol may give some relief. A simple cut should be epithelialized within one week.

Traumatic lesions are commonly produced by too vigorous use of a hard toothbrush, by ill-fitting or unclean dentures, and by jagged teeth. The typical non-specific ulcer of the tongue is described on p. 499. Burns of the mouth are not uncommon in children, and they may be especially extensive in those children who put their mouths to the spouts of kettles or teapots in order to 'taste the steam'.

¹ The name means "the red boy" or "the disease the child gets when the next baby is born" in the Ga language of Ghana.

Aphthous¹ Stomatitis.—This name has been given to many conditions. The three main varieties, which are quite distinct, are as follows:

1. *Monilial stomatitis* (thrush) of infants occurs usually in the first few weeks of life, most often in maternity hospitals and crèches. It is due to *Candida albicans* (p. 12), a fungus found in one-third of the normal population and especially in pregnant women and nursing mothers. The disease appears as spots on the buccal mucous membrane, varying in size from a pin's head to a pea. They are at first red and later become coloured with a white exudate composed of desquamated epithelial cells entangled in the mycelium. Pain and salivation are constant accompaniments. The condition subsides gradually without treatment, but the mouth should be kept clean by the use of glycerin of thymol applied on a swab stick. Nystatin is a specific anti-fungal antibiotic and should be used in the more severe cases. Precautions must be taken to ensure that the milk is fresh and that utensils in which it is served are scrupulously clean.

The condition is not entirely confined to this age group and is sometimes seen in diabetics, in debilitated patients, occasionally in those with leukæmia, and in the 'denture-sore' mouth.

2. *A virus infection* causes another form of aphthous stomatitis in children which occurs usually below the age of six. It is an infectious disease. An attack confers some immunity. The aphthæ or mouth ulcers are usually numerous, small, regular ulcers in a raised, furred, swollen area of mucosa, usually at the anterior part of the mouth.

Treatment is merely to improve general oral hygiene with frequent mouth washes. The children usually recover without incident and without scar, in about ten to sixteen days.

3. *Recurrent aphthous ulceration* is not uncommon in adults. (Mikulicz' aphthæ²). An example of this is shown in fig. 596. The lesions usually occur on the inner side of the lips and cheeks, and the under-surface of the tongue. These ulcers recur in different parts of the mouth over the course of many years. They are not contagious. There is a familial predisposition. They are twice as common in women as in men. In 50 per cent. of the female patients but only in 2 per cent. of the male are concomitant lesions seen on the external genitalia (Sircus). The ulcers are usually single but may occur in twos and threes, though seldom more. They are found on the outer surface of the tongue, in the sulci, and on the inner side of the cheek. They are very painful but heal in between seven and fourteen days without a scar. The treatment is to keep the mouth clean, and if the ulcers are very painful



FIG. 596.—Aphthous ulcer of the tongue.

¹ Aphthous—Greek, *ἄφθαι* = mouth ulcer.

² Known in the U.S.A. as canker sores, and in Dutch-speaking medical circles as sprouw, because it is the first arresting symptom in tropical sprue.

'cauterisation' with tincture of iodine or silver nitrate after local surface anaesthesia is justifiable. Various drug firms prepare adhesive oral pastes. These are useful in treatment if applied to the lesion in an early stage. Some incorporate a topical corticosteroid. The administration of cortisone systemically, which has been recommended, is an unnecessary and dangerous treatment. A culture should be taken from the mouth, and if this shows a preponderance of a pathogenic organism, the appropriate antibiotic may be given.

Vincent's angina (*syn.* *ulcero-membranous stomatitis* or *acute ulcerative gingivitis*) is another form of stomatitis.

Ætiology.—*Borrelia vincenti* is an anaerobic spirochaete with three to five loose spirals. Seen in a wet smear by dark-ground illumination it is actively motile.

Fusiformis fusiformis is a Gram negative anaerobic, spindle-shaped rod. Both these organisms may be found in normal mouths but are present in large numbers in association with lesions of Vincent's stomatitis (fig. 597). Whether they are the

cause of the disease or secondary invaders is debatable. Patients fail to show a significant rise in antibody titre to either organism and each organism alone or both together fail to produce lesions when artificially introduced into the tissues. The incidence of this disease in service personnel and civilians reached almost epidemic proportions during both world wars. Vitamin deficiency in war-time diet was held responsible for lowering resistance against infection. This disease is not communicable. (Findings of the Research Commission of the American Dental Association.)



FIG. 597.—*Borrelia vincenti* and *F. fusiform* (from Gillies & Dodds: 'Bacteriology Illustrated.' E. & S. Livingstone, Edinburgh.)

Clinical Features.—The disease is one of early adult life; rarely does it occur after the age of thirty-five years, and almost never in the edentulous.

In acute cases the prodromal symptoms are general malaise, pyrexia, and increased salivation. About thirty-six hours after the onset the patient complains of dull, generalised toothache, and notices that the gums are bleeding. On examination there is overpowering foetor. The gums, especially the interdental papillæ of the incisors and the gum around the third molars, are red and inflamed, and later covered by yellowish-white pseudo-membrane. If a portion of this membrane is peeled away and the gum wiped free from blood, small ulcers are revealed. Untreated, the ulceration often spreads to the cheeks, palate, fauces, and pharynx, but rarely to the tongue. The regional lymph nodes become enlarged and tender. When the fauces are involved, the condition must be distinguished from diphtheria and secondary syphilis. In subacute and chronic varieties the gingivæ are dark red and swollen, and there is gradual destruction of periodontal tissue with pus-pocket formation.

Treatment.—The mouth should be cleansed with mouth washes and the pseudo-membrane on the surface and the cellular debris between the teeth swabbed away with pledgets of cotton wool soaked in hydrogen peroxide.

Penicillin should be given systemically. When the acute attack has subsided, dental treatment for the chronic disease of the periodontal tissues and carious teeth will be required.

Acute Pyogenic Stomatitis.—Acute stomatitis can be caused by staphylococcal, streptococcal, or gonococcal infections. A concrete diagnosis can only be made by bacteriological examination. Treatment consists of systemic antibiotic therapy and local mouth washes.

THE ORAL MUCOSA AND NUTRITION

Associated with a large variety of nutritional deficiencies, the mucosa of the tongue, mouth and pharynx undergoes atrophy. The atrophic process may also affect other parts of the alimentary tract lower down. The variations of the pattern of this atrophy are very great. In some the condition can be rapidly reversed by treatment; some respond slowly and some not at all. The thinning of the mucosa makes the mouth more susceptible to trauma, and hot drinks and spices may be so painful that they cannot be tolerated by the patient.

Vitamin B (nicotinic acid, the pellagra-preventing vitamin). Pellagra means a rough skin. Disease occurs in areas with a deficient diet, largely consisting of maize. In the acute stages the mouth is painful and becomes a fiery red. Salivation is profuse and the epithelium of the tongue desquamates over wide areas. The skin as a whole becomes thick and irregular and patchy desquamation may occur, leaving unpigmented areas. This is a dangerous condition, but, if diagnosed, is rapidly cured by giving nicotinic acid or nicotinamide.

Vitamin B₂ (Riboflavine) deficiency (fig. 598) gives rise to a diffuse atrophy of the lingual papillæ and is accompanied by a slight magenta tint to the redness of the tongue. On giving Vitamin B₂ improvement takes place in a few weeks. This condition is hardly ever seen in its pure form.

Vitamin C deficiency (scurvy) was rife in the days of the sailing ships. It was stamped out in the Royal Navy¹ by a compulsory ration of lime juice. Leading features of the stomatitis due to this cause are bleeding gums and loosening of the teeth. (For scurvy-rickets, p. 245.)

Iron-deficiency anæmia of long standing, especially in women about the menopause, may be accompanied by a smooth tongue and, if the pharyngeal mucosa is also affected, by dysphagia (Paterson-Kelly, or Plummer-Vinson syndrome, Chap. 31). The condition may be held in check and slowly relieved by the prolonged administration of iron. If left untreated, carcinoma of the hypopharynx or of the mouth is liable to develop.

Pernicious anæmia also produces a smooth tongue, but less often dysphagia. Vitamin B₁₂ sometimes partially restores the buccal mucosa but not the lack of gastric secretion.



FIG. 598. — 'Smooth sore tongue' due to vitamin P₂ (riboflavine) deficiency; note angular stomatitis. (Professor Harold Rodgers, Belfast.)

¹ Lind urged the use of lime juice, and it was due to him that scurvy was eventually eradicated from the Royal Navy. British sailors are known abroad as 'limeys'.

James Lind, 1716–1794. Surgeon's Mate, Royal Navy; later Physician (civilian), Royal Naval Hospital, Haslar.
Donald Ross Paterson, 1803–1939. Oto-rhino-laryngologist, Cardiff.
Adam Brown Kelly, 1865–1941. Surgeon, Ear, Nose and Throat Department, Victoria Infirmary, Glasgow.
Henry Stanley Plummer, 1874–1936. Physician, Mayo Clinic, Rochester, Minn., U.S.A.
Porter Paisley Vinson, Contemporary. Physician, Mayo Clinic, Rochester, Minn., U.S.A.

Tropical sprue, the malabsorption syndromes and post-gastrectomy malnutrition all give rise to a similar condition. If adequately treated, some recovery is possible.

Angular Stomatitis (*syn.* Cheilosis) (fig. 599).—Although cracks at the corner of the mouth were a recognized manifestation of congenital or tertiary syphilis, they are now much more likely to be due to other causes.

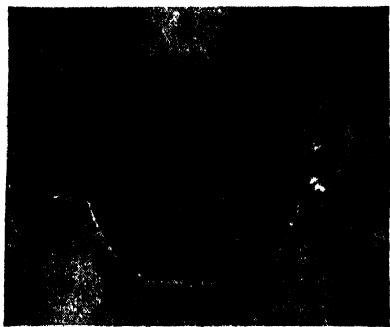


FIG. 599.—Angular stomatitis
(Professor Charles Wells, Liverpool.)

(a) *Perlèche*¹ is a superficial ulceration limited to the angles of the mouth. It appears in children of school age and is of interest chiefly because it has to be distinguished from syphilis. The corners of the mouth become somewhat brown in colour, and often moist fissures make their appearance. There is a burning sensation, and the patient licks the patches frequently, hence the name. Some observers

have recovered anaerobic streptococci from these lesions, and others *Candida albicans*. The lesions usually heal without scarring. Treatment consists in improving general hygiene and in making sure that the diet is adequate.

(b) *Overclosure* is the commonest cause of angular stomatitis in older patients. It can be seen in its most marked form in the 'dentureless edentulous' people. The condition is aggravated if, for any reason, the oral mucosa is atrophied or if there is a tendency to seborrhœic dermatitis. These patients should be referred to a dental surgeon for correction of the overclosure.

Stomatitis due to Drugs.—Excessive doses of bismuth, lead, iodides, and especially mercury give rise to stomatitis, and gingivitis, accompanied by profuse salivation and loosening of the teeth.

Foot and Mouth Disease.—Occasionally this well known disease of cattle is transmitted to man by direct contact or through infected milk. The causative organism is a virus present in the fluid of the vesicles. The incubation period is from two to five days. In man, the constitutional symptoms are usually of moderate severity. The buccal mucosa becomes congested and swollen, and two or three days later vesicles appear on the lips, tongue, cheek, and pharyngeal wall. Later the vesicles rupture, leaving tender, reddish, shallow ulcers, which soon heal. Similar lesions on the hands and feet, particularly round the nails, are usual. The attack confers no lasting immunity, and the same patient may have several visitations of the condition.

Treatment consists of maintaining satisfactory oral cleanliness with bland mouth washes.

Cancrum Oris (fig. 600).—This condition is now seldom seen except as a complication of serious diseases such as the blood dyscrasias. It may also occasionally occur in neglected cases of severe infection, particularly in the undernourished. Various bacteria have been isolated, but there is no conclusive proof that cancrum oris is due to any one organism, although the *Borrelia Vincenti* and *F. fusiforme* are usually present in large numbers. The lesion



FIG. 600.—Cancrum oris.
(R.C.S. Museum)

¹Perlèche—French, *se pourlécher* = to lick one's lips.

appears as an area of acute inflammation in any part of the mouth. There is rapid swelling of the affected area, which is rendered ischæmic and eventually gangrenous. Large areas of lips, cheeks, or jaw may be destroyed. The origin of the process is usually in the gums, lips, or cheek, but occasionally the infection may start in the maxillary antrum. Toxæmia is always severe, and unless treatment with antibiotics is begun early the patient usually dies.

CYSTS

Retention cyst of a buccal mucous gland (fig. 601) occurs in any part of the mucous surface of the mouth. It forms a translucent globular swelling which should be excised under local anaesthesia.



FIG. 601.—Retention cyst of a buccal mucous gland.

A **ranula**¹ implies a *transparent* cystic swelling in the floor of the mouth, mainly, if not entirely, unilateral.

Simple Ranula.—

The patient may state that the swelling has come up before and burst, perhaps several times. When the swelling (fig. 602) is observed closely, tortuous veins can be seen coursing over it, and at one point towards the apex the buccal mucosa seems deficient, as though the cyst was bursting through its covering. An opaque strand can often be made out traversing the anterior wall of the cyst; this is Wharton's duct, which, although displaced by the cyst, takes no active part in the pathological process. Before concluding the examination the possibility of a deep prolongation of the cyst must be excluded by palpating beneath the mandible. The diagnosis of simple ranula is, as Butlin remarked, "plainly written on the face of the tumour".

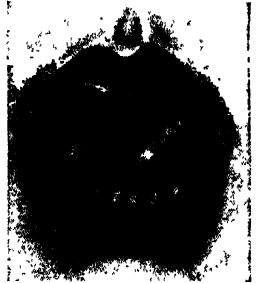


FIG. 602.—A large ranula.



FIG. 603. — 'Retention cyst' of the gland of Blandin and Nuhn.

Pathologically both the mucous cysts and the larger ranulae are in the first place retention cysts, but as the result of trauma the cyst has ruptured and the continued secretion leads to an extravasation of mucous into the tissues. The cyst removed at operation seldom contains an epithelial lining. There is only a mild connective tissue reaction. Any of the salivary glands may be involved. The lower lip is the commonest place. They occasionally involve the glands of Blandin and Nuhn (fig. 603).

Treatment.—*Complete Excision.*—A difficulty hindering ideal treatment is that the cyst bursts before dissection can be completed. If some of the fluid within the cyst can be aspirated before commencing enucleation, complete dissection is usually

¹ So named because of the likeness of the swelling to the belly of a little frog (Latin, *ranula*, diminutive of *rana* = a frog).

possible. Often, however, the contents are of the consistency of jelly, and will not flow through a hollow needle.

Partial Excision with Marsupialisation.—A considerable portion of the cyst wall, together with its superimposed mucous membrane, is excised. The cut edge of the cyst wall is then united by sutures to the cut edge of the mucous membrane; thus the cavity becomes part of the floor of the mouth.

Whichever method is practised, it is necessary to preserve the integrity of Wharton's duct.

Deep or plunging ranula, from the mouth, appears to be a typical ranula, but when the neck is examined a cervical prolongation is found continuous with the intrabuccal one. Possibly these cysts are derived from the cervical sinus—an embryological structure. At any rate, this hypothesis furnishes a logical basis for adequate surgery; this type of ranula must be approached through the neck. Complete destruction of the wall of the sac is essential.

Sublingual Dermoid.—Although congenital, sublingual dermoids are seldom noticed below the age of ten years; usually the patient seeks advice between the ages of thirteen and twenty-five. These swellings are divided into two varieties, median and lateral. Each is again subdivided into those situated above and those situated below the diaphragm of the mouth (the mylohyoid muscles). All are thin-walled cysts filled with sebaceous material, and, unlike other dermoid cysts, never contain hair.



FIG. 604.—Large median sublingual dermoid.

Median variety, which is probably derived from inclusion of ectoderm between the halves of the developing mandible. *When situated above the mylohyoid*, the cyst often attains considerable dimensions (fig. 604) with, apparently, but little inconvenience. *When situated below the mylohyoid*, the patient, if a female, often seeks advice because of a double chin. In this situation considerable care must be exercised in making a diagnosis, for it is not possible to rule out with certainty a suprahyoid thyroglossal cyst. An ectopic thyroid must also be taken into consideration.

Lateral variety, which is somewhat less common than the median variety, is derived from the second branchial cleft. *When situated above the mylohyoid* (fig. 605), there is an opaque swelling in the floor of the mouth to one side of the middle line as opposed to a transparent one (a ranula). *When situated below the mylohyoid*, a cystic swelling in the region of the submandibular salivary gland is present.

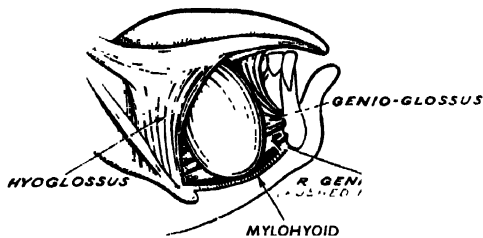


FIG. 605.—Relationships of a lateral sublingual dermoid situated above the mylohyoid.

Treatment of both varieties is removal by dissection through an external incision beneath the mandible.

TUMOURS OF THE CHEEK AND THE FLOOR OF THE MOUTH

Papilloma.—A true papilloma is uncommon. It is soft and fleshy. It should be removed with an adequate margin of tissue. There is a remote chance of malignancy.

Fibro-epithelial polypi are firmer and usually arise after repeated trauma to the inner side of the cheek. They have a dense core of fibrous tissue (fig. 606).

Lipomas can arise in any part of the fatty supporting structures of the mouth—including the sucking pad (fig. 607).

Hæmangioma sometimes occurs under the mucous membrane of the cheek and the floor of the mouth.

Mixed Salivary Tumours may arise from any of the salivary glands in the mouth and occasionally in ectopic tissue (fig. 608).

Adenitis of the Facial Lymph Node.—A few individuals possess a facial lymph node (which lies on the mandible in relation to the facial artery); consequently infection coursing along the lymphatics of the cheek usually passes direct to the submandibular nodes. When a facial lymph node is present its enlargement will perplex the diagnostician unaware of its existence.



FIG. 608.—An ectopic salivary tumour of the cheek. It lies superficial to the buccinator muscle. (After the late L. R. Field.)



FIG. 606.—Fibro-epithelial polypus of the mucosa of the cheek.



FIG. 607.—Well-developed sucking pad.

Carcinoma can arise in any part of the mucous lining of the mouth. In Western races, although carcinoma of the mucous membrane of the cheek is not a rarity, it is less common than carcinoma of the tongue or that of the floor of the mouth. In those

Eastern races who indulge in chewing the betel-nut¹ and store the quid thereof in their cheek, carcinoma of the mucous aspect of the cheek is a common occurrence.

Treatment of Carcinoma of the Cheek.—The indications for irradiation do not differ from those of carcinoma of the tongue (p. 501).

THE TONGUE

Tongue-tie.—Although tongue-tie is really a very rare condition, nearly every mother fears that her first-born is tongue-tied (fig. 609). A former generation of surgeons must have agreed with the mothers; for witness, the grooved director (fig. 610) is even today fitted with a guard for use when dividing the frænum linguæ. This shield is held against the uplifted tongue and the frænum is snipped near the floor of the mouth, the better to avoid the frænal artery and veins. The operation is rarely indicated, as a short frænum has little or no effect on speech or on eating. Its division, especially if done too drastically and followed by sepsis, may lead to fibrosis which might then, in fact, anchor the tongue.



FIG. 609.—Short frænum linguæ.

FIG. 610.—Director with guard and frænum slit.



SCALE 2

¹ This East Indian masticatory is composed of betel-nut rolled up in betel-leaf with lime and catechu.

MACROGLOSSIA

While acute parenchymatous inflammation results in very great enlargement of the tongue (p. 495) the term *macroglossia* should be reserved for cases of chronic painless enlargement of the organ.

Lymphangioma.—When noticed soon after birth usually there is only a small circumscribed patch of dilated lymph vesicles. Occasionally the lesion remains stationary in size for long periods; more often it increases rapidly. Attacks of inflammation occur at irregular intervals. At last the swollen tongue protrudes permanently from the mouth—*lymphangiomatous macroglossia*. Treatment by radium was said to give satisfactory results but excision of the tumour is the treatment of choice.

Hæmangioma, when widespread, can result in *macroglossia*. Occasionally congenital fistula between the lingual artery and vein gives rise to an enormous protruding tongue that sometimes pulsates. Partial excision or, in the case of an arterio-venous fistula, ligation of both lingual arteries, followed by partial glossectomy, is the treatment, but it often does not control the progressive enlargement.

Neurofibroma is on rare occasions the cause of *macroglossia*, which is sometimes confined to one half of the tongue. Usually other manifestations of von Recklinghausen's disease of nerves (p. 56) are present.

Muscular macroglossia is practically confined to idiots and cretins (p. 542). The large tongue protrudes from the mouth and is liable to become dry and cracked. It is being bitten constantly, and if the patient has attained the age of three years, the protruding portion should be excised.

Primary Mesodermal Amyloidosis.—Attention is sometimes drawn to this disease by the *macroglossia* which it produces.



FIG. 611. — Congenital fissured tongue. Note the transverse direction of the fissures.

CONGENITAL FISSURED TONGUE
(*syn.* CONGENITAL FURROWING)

When a patient presents a fissured tongue, it used to be assumed that he or she is suffering from hereditary or acquired syphilis. Fissures, even deep fissures, are usually due to congenital furrowing. John Thomson, after a study of a large number of cases, showed that the furrowing of the tongue was not present at birth, but was acquired in early childhood. What is very important is that in congenital fissured tongue the fissures are mainly *transverse* (fig. 611), whereas in syphilitic fissured tongue they are inclined to be longitudinal.

TRAUMATIC LESIONS

Injuries.—Anæsthetists are familiar with the possibility of the unconscious patient biting his tongue, and so commonly does this accident occur in epileptics that attendants are provided with rubber gags to put between the patient's teeth when a seizure is imminent. The most common deep wound of the tongue follows a blow or a fall while the patient is smoking a pipe, which breaks and is driven into the musculature of the tongue.

As a means of checking severe hæmorrhage from the posterior part of the tongue, Heath recommended passing the finger as far back as possible and hooking the tongue forward on to the jaw, and so applying pressure on the wound.

Sutured wounds of the tongue heal readily with simple mouth-washes, and an almost completely divided segment, if sutured into position, will often remain viable and unite.

Extravasation of blood into the tongue, floor of the mouth, and the adjacent fascial spaces, is a relatively common manifestation of hæmophilia (p. 76). As a rule

the hæmorrhage occurs spontaneously. If facilities for endotracheal intubation are not available, immediate tracheostomy is required.

INFLAMMATIONS OF THE TONGUE

Acute superficial glossitis may follow scalds or other injuries. Under this heading is included *herpes of the tongue*, which is unilateral and painful. Healing occurs rapidly under treatment by bland mouth-washes (see stomatitis, p. 486).

Acute Parenchymatous Glossitis.—In well-marked cases the tongue swells enormously, protrudes from the mouth, and threatens life by asphyxiation. The patient often becomes extremely toxic. The condition, which is rare, arises in a number of different ways:

1. Classically, a wasp inserts its sting into the tongue of a holiday-maker who is drinking beer from a bottle which has been left open. Adrenalin (1 ml. of 1:1000 soln) should be given and ice to suck. In severe allergic cases tracheostomy may be necessary in order to relieve œdema of the glottis.

2. From pyogenic infection of a deep lingual wound.

3. As a part of Ludwig's angina (p. 527).

It is necessary to be prepared for tracheostomy, which, however, in cases of bacterial infection, is seldom required if the patient is treated as follows: Ice is applied to the tongue, oxygen inhaled as required through a Hewitt's air way if necessary. An antibiotic, usually penicillin, is administered, and the patient is reassured.

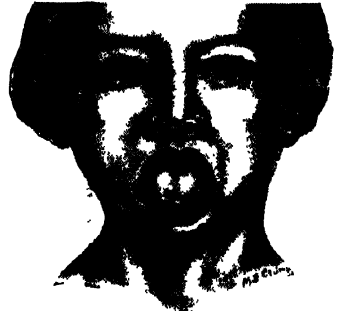


FIG. 612. — Acute glossitis. *Borrelia vincenti* was isolated from a specimen of serum removed from one of the vesicles.

Furring of the tongue.—Furring of the tongue is produced by:

- (1) Accumulation of food debris between the filiform papillæ of the tongue;
- (2) Lack of desquamation of epithelial cells;
- (3) Overgrowth of the normal bacterial flora of the mouth.

In an acutely ill person with malaise and anorexia, such as a patient with peritonitis, the normal movements of the tongue are reduced and the products mentioned above accumulate and produce a fur.

In people who are up and about and eating normally, no fur forms unless the epithelium is constantly irritated by tobacco smoke. In a fit person a furred tongue usually means an excessive smoker (fig. 613). In such individuals, if the fur is thick it is often black or brown in colour. In the atrophic tongue with no filiform papillæ, a fur cannot form.



FIG. 613.—Tongue of an inveterate smoker, showing the typical dark fur.

Black Hairy Tongue.—In some people who smoke excessively, the fur becomes so thick and so long that it gives the appearance of being hairy. A large number of organisms can be recovered from these tongues, but in some cases an unusual fungus, *Aspergillus niger*, is found.

The fur is rubbed off by the teeth and so is usually found only on the dorsum of the tongue (fig. 613). In some cases only a rhomboid area is left in the middle of the tongue at the apex of the sulcus terminalis (the V-shaped dividing line between the

anterior two-thirds and posterior one-third). This area rubs against the teeth less than any other part of the surface of the tongue.

Although smoking and a sedentary way of life are usually associated with this condition, it occasionally occurs in active non-smokers and even in young children. In these cases, among numerous factors which have been blamed, are: (1) Alteration of the oral flora, by the giving of antibiotics, particularly oral penicillin. (2) Vitamin deficiencies, particularly Vitamin A and the Vitamin B complex.

Treatment, if indicated, consists in reducing the smoking, and in cleansing the mouth and teeth after each meal. Dequadin paint usually cures the condition.

Median Rhomboid Glossitis (Glossitis Rhombica Mediana). This is a developmental abnormality of the tongue. In it a reddish patch, found in the midline immediately in front of the circumvallate papillæ, is probably due to inadequate covering of the tuberculum impar in the formation of the anterior part of the tongue. The condition requires no treatment, but it is occasionally mistaken for squamous carcinoma of the dorsum of the tongue.

Chronic Superficial Glossitis and Leukoplakia.—Tradition has it that the six leading ætiological factors are Smoking, Syphilis, Sepsis, Sharp edge of a tooth, Spirits, and Spices, to which one might add Susceptibility to explain why the condition sometimes occurs without any obvious cause.

The essential process in leukoplakia is a slowly progressive hyperkeratosis. The milder changes are widespread, but as the condition gets worse the more severe stages are found in smaller and smaller patches within the widespread areas.

Clinical Features.—Five stages are recognisable. These correspond approximately with the stages shown in fig. 614 which is a diagram of the

histological stages of leukoplakia leading to carcinoma.

Stage 1.—There is a mild thickening of the surface producing a *thin milky film* usually over a widespread area of the mouth; within this area where the hyperkeratosis has progressed we see:

Stage 2.—Here the mucosa looks as if it is covered with areas of smooth paint.

Stage 3.—As the surface thickens still further it becomes irregular like *wrinkled paint*.

Stage 4.—Little *warty projections* appear—this is the stage of ‘cancer in situ’.

The instability of the abnormal mucosa allows another phenomenon to be superimposed on this pattern—desquamation. The surface is shed and leaves *red glazed patches* of smooth mucosa. These raw looking patches are

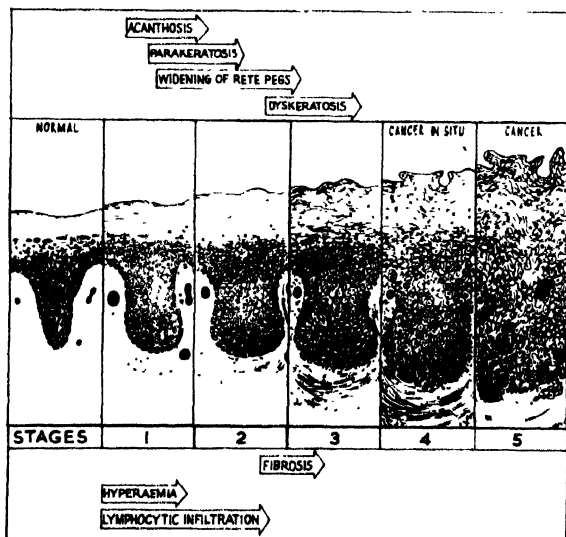


FIG. 614.—Diagram of the histological stages of leukoplakia leading to carcinoma.

more likely to occur in women especially if they suffer from iron deficiency and already have a thin mucosa.

Microscopically a thick layer of keratin may be present on the surface of the epithelium (keratosis or hyperkeratosis). Often the process of keratinisation is incomplete and flattened nuclei remain visible in this layer (parakeratosis). There is overgrowth of the prickly cell layer. This produces elongation and widening of the rete pegs. The subepithelial tissues show mild to moderate chronic inflammation. An important feature, with regard to prognosis, is the finding of abnormal cells in the deeper layers of the epithelium. Thus occasional cells with large deeply-staining nuclei, cells with two nuclei, cells showing abnormal



FIG. 616.—Leukoplakia on the palate and checks. This patient had had three carcinomas 'in situ' removed from his tongue.



FIG. 615.—'Chronic superficial glossitis' showing stages 2 and 3.

mitoses, and cells prematurely forming keratin may be found. The presence of such cells indicates epithelial dysplasia and is described as a dyskeratotic change. Epithelium showing dyskeratosis may eventually become frankly malignant. An intermediate phase where dyskeratosis is very marked with loss of the normal cellular arrangement within the epidermis, but without actual infiltration (i.e. tumour growth into the underlying connective tissue), is termed carcinoma 'in situ'. These changes are diagrammatically shown in fig. 614.

Distribution.—If there is a local cause such as a jagged tooth or the habit of holding a pipe always in the same place, the leukoplakia may be localized to that one area, but in some cases the disease is diffuse and affects the whole lining of the mouth.

Symptoms are few, and apart from some soreness in the bare red patches of desquamation, the main complaints are of the peculiar look of the tongue and of difficulty with dentures.

Treatment.—Leukoplakia may be pre-malignant, and although the severity of the condition is often suggested by the clinical appearance, early biopsy to confirm the diagnosis and to indicate any epithelial dysplasia is necessary. In particular, an indurated white patch appearing in the adult mouth should be biopsied at once, since squamous carcinoma may present in this way.

Small patches of leukoplakia may be removed. When the affected area is larger than can be excised and the diagnosis has been confirmed histologically, the patient should be examined at intervals of not longer than three months. Any areas of 'warty excrescence' or any doubtful area which appears, must be removed and examined histologically.

Radiotherapy will improve the appearance of the condition but probably increase the likelihood of malignant disease, and this, if it occurs, will then probably be resistant to further radiotherapy.

Primary Syphilis.—An extra-genital chancre can occur on the tongue (fig. 28, p. 28). The submaxillary and submental lymph nodes become greatly enlarged, as in the case of a similar lesion on the lip.

Secondary Syphilis.—1. *Multiple shallow ('snail track') ulcers* may be present on the sides and under-surface of the tongue. 2. *Mucous patches* occur on the tongue as well as on the fauces. 3. *Hutchinson's wart*, really a condyloma, is a strictly median 'wart' (fig. 617) and has to be distinguished from a papilloma which happens to occupy the middle line.



FIG. 617.—Hutchinson's wart. Wassermann reaction strongly positive.

Tertiary Syphilis.—1. *Gumma of the tongue* which is extremely rare in England nearly always occupies the middle line. It possesses the characteristics of gummata elsewhere and does not tether the tongue (fig. 38). 2. The frequency of chronic superficial glossitis in syphilitic patients has been noted already.

Lingual Tuberculosis.—In the pre-antibiotic era *secondary lingual tuberculous ulcers* were fairly common: they were seen particularly in persons under treatment for advanced pulmonary or laryngeal tuberculosis. When a patient is under treatment for pulmonary tuberculosis by the modern powerful chemotherapeutic agents, tuberculous ulceration of the tongue and the mouth does not occur. Thus at the present time secondary lingual tuberculous ulcers are encountered only in persons with established but *undiagnosed* pulmonary tuberculosis. Not a few of such cases are discovered by the dental surgeon before the pulmonary disease has become evident. Typical tuberculous ulcers of the tongue are shallow, often multiple (fig. 618), ovoid and greyish-yellow, with a slightly reddened margin. When the ulcer or ulcers are situated on a mobile portion of the tongue, they are so painful as to require amethocaine lozenges for their relief.



FIG. 618.—Typical tuberculous ulcers in a patient with pulmonary tuberculosis. (Maynard K. Hine, D.D.S., Indianapolis, U.S.A.)

Geographical tongue (*syn. glossitis areata exfoliata, glossitis migrans*).

This condition is seen most commonly in patients who have a severe abdominal condition such as peritonitis and who are being treated by broad spectrum antibiotics, gastric suction and continuous intravenous feeding. It possibly indicates that there is a severe strain on the patient's metabolism in which his power to regenerate his alimentary epithelium as a whole is deranged.

The tongue is at first heavily furred. Then, on about the third or fourth day of the illness, when the patient is still very ill and feverish, the tongue becomes a little sore,



FIG. 619.—March 5th. FIG. 620.—March 7th. FIG. 621.—March 9th. FIG. 622.—March 12th, patient making satisfactory recovery.

FIGS. 619-622.—Glossitis migrans—this patient had a partial gastrectomy on March 1st, followed by paralytic ileus and pulmonary collapse which was treated by tetracycline.

and large areas rapidly desquamate in an irregular circinate pattern. The areas change from day to day (figs. 619 to 622). No causative organism is known to produce the condition, but there is always a change in the flora of the mouth and *Candida albicans* is almost always present in abundance.

No local treatment is indicated. As the patient's general condition improves, the tongue returns to normal.

ULCERS OF THE TONGUE

It is advisable at this stage to review briefly the more common types of ulcers of the tongue, as their differential diagnosis is so important in clinical surgery.

Dental ulcers always occur at the side of the tongue and are due to the laceration of the mucosa by a sharp tooth or sharp clasp of a denture. They are therefore longitudinal scratches that have become infected. The cause should be removed (fig. 623).



FIG. 623.—Dental ulcer caused by the sharp edge of the clasp of a denture.

Post-pertussis ulcer is seen at the frænum linguæ. Of necessity, this occurs only in children with whooping-cough.

Chronic non-specific ulcer is a clinical entity that gives difficulty in diagnosis. The ulcer is not very painful, moderately indurated, and is usually



FIG. 624.—Chronic non-specific ulcer of the tongue.

situated on the forepart of the tongue (fig. 624). There is no history or obvious cause to attribute the lesion to trauma. The Wassermann reaction is negative. There is no evidence of tuberculosis in the lungs or on histological examination of the lesion.

Local excision should be carried out in order to confirm the diagnosis, as well as to cure the condition.

Aphthous ulcer (p. 487).

Specific.—Syphilitic Ulcer (p. 38).

Tuberculous ulcers (p. 498).

Malignant.—Carcinomatous ulcer has typically the clinical features of a squamous-celled carcinoma—viz. an everted edge and an indurated base.

If doubt exists as to the nature of an ulcer—and it is sometimes difficult to be certain when dealing with cases of early carcinoma—the ulcer should be excised under local anæsthesia and submitted to histological examination. In this instance a Wassermann reaction is a hindrance rather than a help in establishing the diagnosis, for even at the present time it is not unusual for a patient with carcinoma of the tongue to have had syphilis; in the pre-antibiotic era it was almost the rule.

NERVE LESIONS OF THE TONGUE

Lingual neuralgia can occur idiopathically, or be due to the lingual nerve being implicated in scar tissue or neoplastic infiltration. A not infrequent cause is as an aftermath of lingual herpes. In severe and persistent cases section of the chorda tympani brings relief. The chorda tympani contains not only gustatory and secretory

August von Wassermann, 1866–1925. Director, Institute for Experimental Therapy, Berlin.

fibres, but also sensory fibres. The nerve can be severed in its canal close to the posterior border of the tympanic membrane. The operation can be conducted under local anæsthesia.

Glossodynia, or burning tongue, is a troublesome condition: it is a symptom, and not a disease. When it occurs in connection with an observable lesion, the treatment consists in removing the cause. However, in many cases, especially in women over forty years of age, nothing amiss with the tongue can be discovered, and it is really part of an anxiety state. The symptom is often associated with cancerophobia and reassurance is all that is necessary. If it occurs in a dry mouth, the sucking of sweets or magnesia tablets may give some benefit.

Hemiatrophy of the Tongue.—In so far as the tongue, the floor of the mouth, and the submaxillary region are concerned, hemiatrophy of the tongue is the result of damage to the hypoglossal nerve. This causes paralysis of the musculature of the tongue, so that on protrusion the tongue deviates to the affected side. There is some thickening of speech and interference with deglutition. Usually, however, after a few months the patient manages to accommodate his speech and eating habits to his glossal weakness.

BENIGN NEOPLASMS

Benign neoplasms of the tongue are comparatively rare: they are over-shadowed by the frequency of carcinoma of this organ. The following, in order of frequency, occur from time to time:

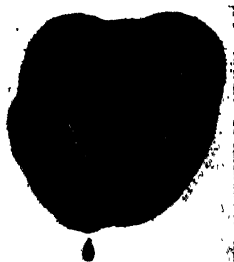


FIG. 625. — Venous hæmangioma of the tongue in a woman of twenty-three.

Papilloma is the most common benign tumour of the tongue. It can be sessile or pedunculated, and must be distinguished from a Hutchinson's wart (fig. 617). It should be excised, together with a small wedge of normal tissue about its base and subjected to histological examination.

Angioma in this situation is usually venous (fig. 625). The veins which form the tumour are liable to become wounded and bleed. Such hæmorrhage may be so persistent as to render the patient severely anæmic.

The treatment is excision of the tumour.

Although not neoplasms this is a suitable place to draw attention to:

The 'Caviare' Lesion of the Under-surface of the Tongue.—On asking the patient to put the tip of the tongue on the palate, so as to display the under-surface of the tongue, it is not uncommon in ageing persons to see a cluster of small varicose veins beneath the transparent, shiny mucous membrane. When pronounced, these blue-black clusters resemble sturgeon's eggs—hence the name.

Lingual thyroid (p. 565).

Lymphangioma (Macroglossia, p. 494).

Plexiform neuroma, Neurofibroma (Macroglossia, p. 494).

A **lipoma** of the tongue is never very large, because the patient is continually conscious of its presence, and seeks advice early; as a rule it is about the size of a coffee bean.

Myoblastoma (Granular cell myoblastoma) presents as an irregular indurated area beneath the epithelium of the tongue. The epithelium shows a pseudo-epitheliomatous thickening, usually without ulceration. Biopsy is required to confirm the diagnosis. Very slowly growing, this tumour is composed of large granular cells whose origin is uncertain. Symptoms are few and recurrence after excision is rare.

Osteoma of the tongue is a clinical curiosity, only 9 cases having been reported in the past forty years. In all, a hard swelling was discovered in the posterior third

of the tongue, and removed. Most of these osteomas have been centred beneath the foramen cæcum. The bone arises from a remnant of a branchial arch.

MALIGNANT NEOPLASMS

Carcinoma of the Tongue.—Since 1910 there has been a decreasing incidence of carcinoma of the tongue in *males*. The more efficient treatment of syphilis, the passing of the clay pipe, the price of tobacco, the welcome activity of our dental colleagues, and possibly the decrease in the consumption of spirituous liquors are the chief reasons for this decline.

While the reduction in the incidence of carcinoma of the tongue in males is gratifying, it is perhaps all the more disconcerting to learn that there has been an increase in the number of female sufferers (Russell). The sex incidence is now equal.

Clinical Features.—As in carcinoma in other situations, in its early stages carcinoma of the tongue is virtually symptomless. The most frequent locations are the lateral margins of the anterior two-thirds of the tongue (fig. 626). If the patient is observant and fastidious, he or she seeks advice because of the actual lesion on the tongue, such as:

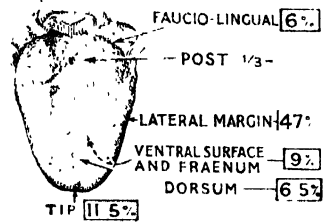


FIG. 626.—Relative frequency of the seat of carcinoma of the tongue. (Birmingham United Hospitals' Statistics.)

1. A small warty outgrowth, 2. An ulcer (fig. 627), 3. A fissure, or 4. An indurated mass.

It is a sad fact that a large number of patients fail to notice or else disregard the lesion in its early stages; consequently the average lapse of time between the onset of symptoms and seeking relief is five months, the patient reporting only because of one or more of the later symptoms, which are:

(a) *Pain* is experienced either in the tongue, or it is referred to the ear. The latter is not unusual, and many a patient with carcinoma of the tongue presents with a wad of cotton-wool in his ear, complaining solely of earache. The explanation of this phenomenon is that the lingual nerve is involved and the pain is referred to another branch of the third division of the fifth cranial nerve, to wit, the auriculo-temporal.

(b) *Salivation*.—Profuse salivary secretion is common in lingual carcinoma. If an elderly man, sitting in the surgical out-patient department, is seen to spit repeatedly into his handkerchief, it is highly probable that he has a carcinoma of the tongue. In late stages the saliva is blood-stained.

(c) *Ankyloglossia*.—The tongue cannot protrude fully and deviates to the affected side. This bespeaks extensive carcinomatous infiltration of the lingual musculature or the floor of the mouth.

(d) *Dysphagia*.—The patient experiences difficulty in swallowing. This symptom is more pronounced when the growth is in the posterior third of the tongue.



FIG. 627.—Carcinoma-tous ulcer of the tongue. (Dent. Bodenham, F.R.C.S., Bristol.)

(e) *Inability to Articulate Clearly*.—Factors (a), (b) and (c) may all play a part (fig. 628).

(f) *Fætor*.—The patient becomes offensive to his associates because of the secondary bacterial stomatitis.



FIG. 628.—The patient sought advice because of difficulty in speaking and swallowing. Referred from a radio-therapist as unsuitable for radiotherapy.

(g) *A lump in the neck*, due to secondary deposits in the cervical lymph nodes.

A growth situated right at the back of the tongue often escapes the notice of an intelligent patient and even of his medical adviser. *Early alteration of the voice* is often a feature of these cases. Palpation of the posterior part of the tongue and laryngoscopic examination are cardinal methods in detecting a neoplasm in this situation. Because the diagnosis of a growth in this secluded area is late, its average diameter is greater and the incidence of palpable cervical lymph nodes is higher than when a carcinoma is situated in other parts of the tongue.

Spread of the Disease.—(1) **Local**.—*Carcinoma of the anterior two-thirds of the tongue*, which usually starts on the lateral margin, invades the floor of the mouth early but seldom extends across the fibrous septum in the midline. *Carcinoma of the posterior third of the tongue* tends to spread to the corresponding tonsil, the epiglottis, and the soft palate.

(2) **Lymphatic**.—It is important to realise that in 50 per cent. of individuals the lymphatic vessels draining the anterior two-thirds of the tongue and the floor of the mouth traverse the periosteum of the mandible on the way to the submental and submandibular lymph nodes. Long before carcinoma-bearing cervical lymph nodes are palpable, careful microscopical scrutiny reveals that some of them are implicated. Because of their secluded position and consequent late diagnosis, growths of the posterior part of the tongue show the highest incidence of cervical metastases.

(3) **By the Blood-stream**.—Metastasis by this route is exceptional; indeed it occurs in only 2 per cent. of patients, and in these the neoplasm is situated in the extreme posterior part of the tongue in almost every instance.

Terminal Events.—Unsuccessfully treated, the disease runs a variable, but inevitably fatal, course. Death occurs usually in one of the following ways:

1. *Inhalation bronchopneumonia*, from the superadded oral sepsis.
2. *Combined cancerous cachexia and starvation*.
3. *Hæmorrhage*, from the primary growth, or on account of involved lymph nodes ulcerating and eroding an artery.
4. *Asphyxia*, which is due either to secondary carcinomatous cervical lymph nodes pressing upon the air passages or to œdema of the glottis. The latter is rare, and is due to an extension of lymphatic œdema around a growth at the back of the tongue.

Treatment

1. Preliminary measures must first be taken to make the mouth as clean as possible. (a) A culture of the ulcer is taken to determine the predominant organism and the most suitable antibiotic to use. (b) The mouth is cleaned by frequent antibiotic mouth washes. (c) Dental treatment for carious teeth or gingival sepsis is carried out. (d) If the Wassermann reaction is positive, anti-syphilitic treatment is begun.

2. Surgery.—(a) *If the lesion is small*, under 1 cm. in diameter, or if it is one of the doubtful excrescences like a 'fragment of cauliflower', excisional biopsy is required—a wide margin of mucosa not less than $\frac{1}{2}$ cm. being removed around the lesion. If this shows either a cancer 'in situ' or that an early invasive carcinoma has been adequately excised, no further treatment is required but *monthly follow-up is obligatory*. (b) *Larger lesions* should be treated by radiotherapy (see below) which is successful in a majority of cases (40–80 per cent., depending on the site and type of the tumour). In those cases that fail to respond within two months of the completion of treatment, surgery, and usually major surgery, is indicated. If the lesion is still clinically localized to the anterior two-thirds of the tongue, wide local excision (fig. 629), or partial (fig. 630), or sub-total glossectomy (removal of the anterior two-

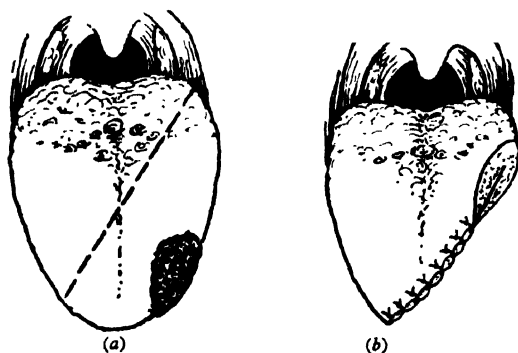


FIG. 629(a) and (b).—Partial glossectomy suitable for neoplasms on the lateral margin or tip of the tongue.

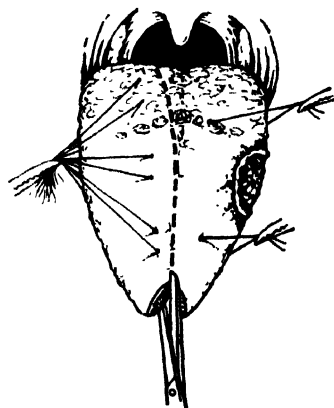


FIG. 630. — Hemiglossectomy with scissors. A diathermy knife should not be used.

thirds) is carried out. To do this it may be necessary to split the lower lip in the midline and carry the incision through the mandible, bisecting it with a Gigli saw. Good access is obtained and the tongue can be removed under good vision. The mucosa of the floor of the mouth is sutured with catgut.

Diathermy in the mouth, although often used and recommended, is undesirable because it produces a deep burn which always becomes infected, healing is delayed until the slough separates and secondary hæmorrhage is thus encouraged. A scalpel or scissors should be used and bleeding arrested in the usual way.

(c) *Where the growth involves or reaches to within 2 cm. of the jaw*, removal of that half of the mandible is required.

If the division of the mandible includes the attachments of the genio-glossus and genio-hyoid muscles to the genial tubercles, tracheostomy may be re-

quired, especially if the patient has chronic bronchitis with copious sputum. It will be difficult for him to expectorate immediately after operation, and fatal broncho-pneumonia may otherwise occur. Eddey uses a piece of steel wire to hold the remaining half of the jaw in position (fig. 631). This can also be achieved by the use of dental prostheses. Even if no fixation is applied, the results are usually quite satisfactory.



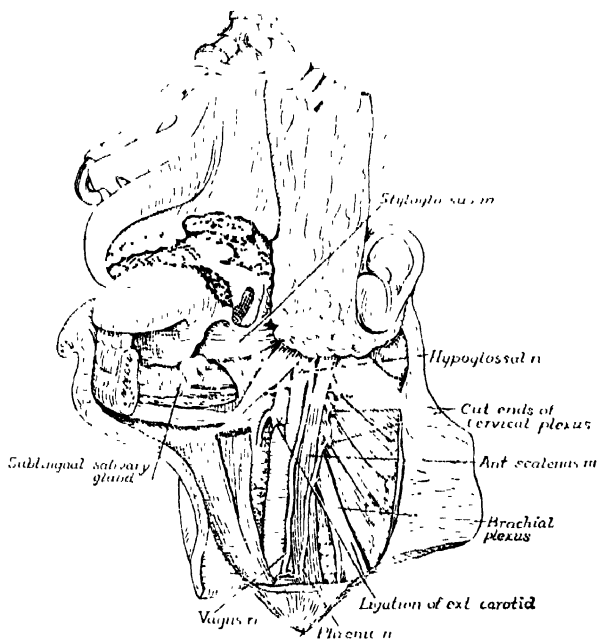
FIG. 631.—Kirschner's wire interposed between the cut surfaces of the mandible to hold the chin in a central position. (H. H. Eddey, F.R.C.S., Melbourne.)

(d) If the lymph nodes are palpable and firm, and they are suspected of being involved, procedures (b) and (c) (see above) are combined with a block dissection (fig. 632).

(e) 'Block' Dissection.—Most surgeons do 'block' dissection (p. 535) only if lymphatic involvement is diagnosed clinically. A careful monthly follow-up is maintained, by the same person if possible, and the operation performed only if the lymph nodes enlarge. The best results have been achieved by this policy although it is somewhat anomalous to do an operation which deliberately delays removing possible malignant tissue and also which leaves behind

some of the tissue lying between the primary and the lymphatic deposits—namely, the floor of the mouth and the peri-mandibular tissues. The

FIG. 632.—The so-called 'commando' operation. Partial mandibulectomy having been performed, wide access is afforded to the posterior part of the tongue. Note that the external carotid artery has been ligated. (After Hayes Martin.)



term 'block' dissection is thus a misnomer. If palpable lymph nodes appear on the other side, 'block' dissection on that side will be necessary. This carries a risk of disturbing the cerebral circulation caused by ligating both internal jugular veins. Therefore only the vein on one side may be removed.

3. **Radiotherapy.**—(a) *Interstitial irradiation* is used for lesions larger than 1 cm. in diameter in the anterior two-thirds. Radium needles, radon seeds or radio-active tantalum in the form of wire are placed in the tissues in one plane at 1 cm. apart. These methods are unsuitable if the growth cannot be treated by a simple single plane implant. Therefore if the growth is larger than 2 cm. in diameter or extends more than 1 cm. into the tongue—and this is difficult to determine—then the radiotherapy of choice is some form of:

(b) *Teletherapy.*—The form usually used is the Cobalt 60 unit. When the growth is situated in the posterior one-third of the tongue, the anatomical difficulty of interstitial treatment is so great that teletherapy is employed for all such growths.

There is widespread agreement that radiotherapy is of little or no value in the treatment of lymphatic metastases whether as a primary treatment or when the disease is recurrent after 'block' dissection.

Radiotherapy in any form applied to the jaw renders it liable to necrosis, especially if it is already infected or involved in growth or if the radiotherapy is excessive, as it may be if a radium needle lies in contact with bone. It is also worse with the use of 250 Kv. than with the higher voltages, i.e. 2,000 Kv. Necrosis of the jaw may lead to severe sepsis which will not clear up until the jaw has been removed. Thus, some consider that a growth involving the jaw should be excised and not irradiated.

Irradiation can cause a *delayed pathological fracture*, even years after treatment. This, although troublesome, is not a dangerous condition and should not deter the radiotherapist.

4. **Chemotherapy.**—Regional intra-arterial administration of Amethopterin (50 mg. per day for about five days) can be used as a preliminary to excision. In 5–10 per cent. of the cases the growth disappears; in about 75 per cent. of cases the growth gets a little smaller and the patient improves for a few months. No long-term survival is likely (Johnston).

Prognosis.—The five-year survival rate after treatment (all varieties) is about 25 per cent.

Palliation

1. Radiotherapy to a large primary growth, even when untreatable secondaries are present, is well worth while.

2. Surgical resection of a recurrence after radiotherapy in the mouth, if feasible, often increases the patient's comfort even if irremovable metastases already exist.

3. For the pain of the advanced growth, blocking the Vth nerve and the cervical plexus with 10 per cent. phenol may help.

4. Antibiotics are useful for a limited time by reducing the pain of secondary infection.



FIG. 635.—Stones in the parotid gland.

conclusively, for a small calculus is often difficult to visualise by radiography (fig. 635).

Sialography.—A parotid calculus too small or too radio-transparent to cast a shadow on a plain radiograph can be demonstrated by contrast.

Treatment.—If the stone can be palpated from within the mouth, it can be removed by slitting up Stensen's duct, but we are seldom so favoured. More often it is deeply placed within the parotid tree. The best method of treatment is to expose the gland and remove the calculus through a transverse incision in the gland substance.

If multiple stones are present superficial lobectomy or complete parotidectomy (p. 513) should be carried out.

INFLAMMATIONS

Acute Parotitis.—As a rule bacteria reach the gland by retrograde infection from the mouth; in a few instances the infection is blood-borne. In fulminating cases the causative organism is nearly always a hæmolytic *Staphylococcus aureus*. In some of the less severe examples the pneumococcus is responsible.

In contradistinction to epidemic parotitis (mumps), the infection is often confined to one parotid gland, although in a small percentage of cases the other becomes implicated later.

Ætiology.—Acute parotitis is encountered in several dissimilar circumstances.

1. *Idiopathic.*—In not a few instances there is no predisposing cause. The patient presents for the first time with signs of acute parotitis.

2. *Post-operative parotitis* has become relatively infrequent; the reason for the decline is the frequent use of antibiotics after major operations, combined with better oral hygiene, better control of fluid and electrolytic balance, and blood replacement. At the present time, when post-operative parotitis does occur, it is uræmic subjects who are more likely to be attacked than others, and the organism is frequently a penicillin-resistant staphylococcus.

3. *As a complication of debilitating medical disease*, especially typhoid and cholera. As in post-operative parotitis, a dry infected mouth is the probable reason for the supervention.

4. *As a Complication of Acute Pancreatitis.*—The structural and functional similarity of the pancreas and the parotid glands is seemingly a good reason for both structures being attacked via the blood-stream by organisms having an affinity for secretory glands of this type.

5. *Secondary to Obstruction of Stensen's Duct.*—This may be due to a parotid calculus but foreign bodies sometimes find their way into Stensen's duct.

6. *As a Complication of Septicæmia.*—Embolic parotitis may occur.

Clinical Features.—There is a brawny swelling on the side of the face, (fig. 636). Signs of toxæmia are variable; usually the temperature is well over 100° F. (37·8° C.). On many occasions pus or purulent fluid can be expressed from Stensen's duct, enabling the sensitivity of infecting organisms to be tested against antibiotics.

Treatment.—If acute parotitis threatens, no effort should be spared to cleanse the mouth. Boroglycerol is a useful adjunct in this respect. A

ialogogue, in the form of chewing-gum or pineapple, is also valuable.

Early unilateral cases often respond to anti-biotic therapy.

Fulminating Cases.—When the response to the above measures is not obvious within forty-eight hours, and particularly in bilateral cases, early decompression of the gland or glands is strongly recommended.

Decompression of the Parotid Salivary Gland.—Local anæsthesia is entirely satisfactory. A vertical incision is made down to the capsule of the gland. With suitable undercutting of the skin nearly

the whole of the parotid gland can be exposed. In order so spare the branches of the facial nerve, the capsule is incised transversely, if necessary in several places. The skin is closed with a few interrupted sutures and drainage is provided at the lower end of the wound.

If decompression is not performed, provided the patient is not overwhelmed by toxæmia, suppurative parotitis goes on to abscess formation. If incision is withheld, the abscess may burst externally, usually between the bony and cartilaginous parts of the external auditory meatus.

Recurrent subacute and chronic parotitis are rather more common than acute cases.

Pneumococcus

or *Streptococcus viridans* are the usual causative organisms. The condition can be unilateral but is frequently bilateral (fig. 637).

Children and young women are the usual sufferers. Inspection of the orifice of Stensen's duct while gentle pressure is exerted over the gland often reveals a gush of purulent saliva in process of ejection (fig. 638), and the diagnosis, which up to that time is often in doubt, becomes indisputable. Parotid calculus (p. 507) must be eliminated.

Sialography.—In chronic parotitis the main duct, the ductules, and the acini are all dilated (fig. 639) (cf. sialectasis where the main duct is normal).

Treatment.—Oral hygiene and antibiotic therapy can be tried, but these measures are frequently disappointing. Patients with chronic parotitis are benefited, often dramatically, by catheterising Stensen's duct with a fine ureteric catheter and injecting a bland antiseptic fluid, such as 1 per cent.



FIG. 636.—Acute parotitis.



FIG. 637.—Bilateral chronic bacterial parotitis. Present for nine months. Slight variations in the size of the swellings noticed.



FIG. 638.—Purulent saliva being ejected from Stensen's duct.



FIG. 639.—Sialograph in a case of chronic parotitis (lateral view).

mercurochrome. This measure can be repeated as necessary. A few long-standing cases eventually require parotidectomy.

SALIVARY FISTULA

A salivary fistula may be internal or external. As an internal fistula does not give rise to symptoms, and as an external fistula of the submaxillary gland is both rare and cured readily by removal of that gland, the subject resolves itself into a consideration of the troublesome condition *external fistula of the parotid*.

Parotid Fistula.—The amount of discharge varies with the site of the fistula. Apart from some moisture on the face when eating, an external fistula connected with the gland itself produces but little inconvenience. On the other hand, a fistula of a large duct is associated with extreme discomfort. Every time the patient has a meal, smells food, or even thinks of it, there is an outpouring of parotid secretion on to the cheek. In addition to the annoyance of such a phenomenon, the skin in the neighbourhood tends to become excoriated. These fistulæ usually follow a badly placed incision for the opening of a parotid abscess, but may be an aftermath of a penetrating wound, especially by glass splinters. Although some leakage of saliva occurs for several days, a persistent fistula following partial parotidectomy is of rare occurrence. A salivary fistula that has continued to discharge for several months seldom closes spontaneously. Usually the external opening is pin-point.

Sialography is invaluable in these cases. It will indicate whether it is the main duct or a ductule that communicates with the surface. From the information thus gained the proper course to adopt for the cure of the fistula can be formulated.

Treatment.—When a fistula has been proved to be connected with a minor branch of the parotid tree, the atropine-like effect of probanthine (propantheline bromide) 50 mg. every six hours for a week is likely to succeed. In other circumstances operative measures must be invoked. Of many plans suggested, Newman and Seabrook's is the most satisfactory:—

Operation.—Prophylactic systemic antibiotic therapy is commenced twenty-four hours before the operation. One probe is introduced through the orifice of Stensen's duct and another through the external fistula. With the probes in position, a suitable incision is made in the cheek, so as to display Stensen's duct and the area of gland containing the probes. After a tedious dissection to free the severed conducting mechanism of the gland, No. 10 tantalum wire, which is twisted on itself, is passed as shown in fig. 640. Any portion of the twisted wire that lies exposed is

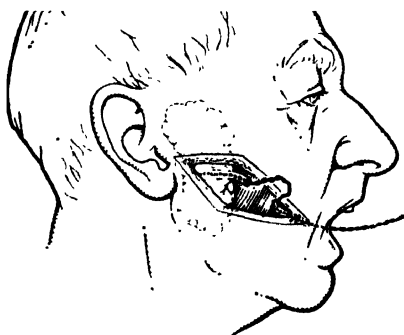


FIG. 640.—Newman and Seabrook's operation for parotid fistula.

buried with fine catgut sutures, and the skin is closed. The distal end of the wire is suitably bent around the corner of the mouth, and anchored there by adhesive plaster.

Post-operative Treatment.—Antibiotic therapy is continued for several days. Oral hygiene is most important, and each day the wire splint is moved very gently; it should remain in place for about three and a half weeks.

Should a reconstructive operation such as the above prove a failure, complete parotidectomy (p. 513) should be performed.

CYSTS OF THE PAROTID GLAND

Most cysts of the parotid gland occur in the region overlying the angle of the jaw, and as they are often found to be lined with epithelium, it seems probable that they are derived from the first branchial cleft. The condition is much less common than adenolymphoma which, however, is seldom so fluctuant when as small as the cyst (which was lined by squamous epithelium) shown in fig. 641. Occlusion of a main branch of the parotid tree by a calculus occasionally gives rise to a cyst.

The treatment is extracapsular excision (p. 512). If the cyst is secondary to a calculus, removal of the stone will cure the condition.



FIG. 641. — Cyst of the parotid.

NEOPLASMS

Parotid Tumours	{	<i>Benign</i> = Adenolymphoma.	}	
		<i>Potentially malignant</i> = Mixed parotid tumour.		
		<i>Malignant from the onset though for some time not obviously so</i>		
				= Carcinoma

Adenolymphoma (Warthin's tumour).—Although classified as an innocent *parotid* tumour, the parotid gland, were it able to do so, might, with some justification, disclaim paternity. Embryological studies of this region have shown that frequently ductal structures from the primary parotid bud grow into the developing juxta-parotid lymph nodes. As a result the encompassed ductules lose all connection with the parotid tree. At least one juxta-parotid lymph node containing normal salivary tissue is found in all infants. Here lies the explanation of the birth of an adenolymphoma: it is an epithelial tumour arising within a periparotid lymph node.

Pathology.—Most frequently the neoplasm is situated outside the parotid capsule or just beneath the capsule, embedded in the superficial lobe. On macroscopical section multiple cysts are revealed, varying in size from a pin's head to a hazel nut, and greyish-pink in colour. Microscopically the essential components are papillary epithelium embedded in a lymphoid stroma—an extremely characteristic picture. The tumour grows slowly, and is benign.

Clinical Features.—Warthin's tumour is not uncommon. In 90 per cent. of cases it does not appear until the patient is over forty years of age. Males are more often affected than females (5:1). The tumour appears to be confined to white races. While its commonest location is similar to that of a mixed parotid tumour (*vide infra*), not infrequently at least some part of the swelling is situated in the neck below the angle of the mandible. This neoplasm is never stony hard, and in 30 per cent. of cases it is sufficiently cystic for fluctuation to be elicited, which suggests that the swelling in question is a Warthin's tumour.

Treatment.—Extracapsular excision of the tumour is curative. The

tumour is not sufficiently radio-sensitive to warrant recommending X-ray therapy which, if unsuccessful, makes excision more difficult.

'Mixed parotid tumour', the commonest parotid tumour, is a well-known clinical entity that occurs with equal frequency in either sex, and usually first appears in early adult life. For some obscure reason parotid

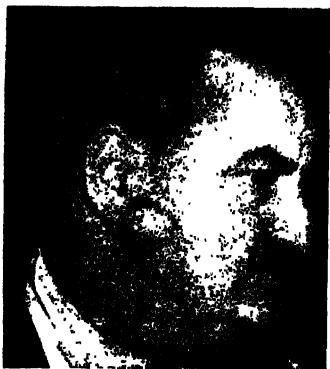


FIG. 642.—Mixed parotid tumour, typical location.

tumours are seldom, if ever, confined to the upper part of the gland. A firm, somewhat rounded, slowly growing neoplasm, nearly always commencing in that part of the parotid gland overlying the angle of the jaw (fig. 642), renders the diagnosis tolerably simple. Usually benign for a varying period from several months up to ten or twenty years, it sooner or later breaks its confines and exhibits characteristics of malignancy. It now tends to invade the pterygoid fossa and the upper part of the neck, and sometimes causes facial paresis from involvement of the seventh nerve. A benign parotid neoplasm rarely, if ever, causes

facial palsy. When first seen in a comparatively advanced state it is difficult to diagnose from other malignant tumours of the region.

Pathology.—A 'mixed parotid tumour' commences its existence as a *pleomorphic adenoma* containing fibrous, myxomatous, pseudo-cartilaginous, and epithelial elements in varying proportions. It grows very slowly, and may remain almost stationary for many months or years and then, comparatively suddenly, the variegated epithelial elements burst into mitotic activity and a pleomorphic carcinoma develops (some 2 to 3 per cent. of cases). On rare occasions one epithelial component alone runs riot, and a highly malignant anaplastic carcinoma results.

Carcinoma.—Many of these tumours are of peculiar histological pattern, in which alternating layers of cylinders of cells and hyaline material are arranged around a central cavity that sometimes contains mucus (cylindroma). The tumour is more quickly and more highly invasive and in all respects more malignant than a mixed parotid tumour. If it is not removed *in toto* early, relentless recurrence is inevitable. The tumour is radio-resistant.

Other varieties of parotid carcinoma may show the histological pattern of adenocarcinoma, anaplastic carcinoma or, rarely, squamous carcinoma.

Treatment of Tumours of the Parotid Gland.—Nearly all of these tumours are radio-resistant, consequently the patient should be urged to have the tumour extirpated while it is comparatively small. The aim must be to excise the tumour, together with its capsule, without opening the latter. In order to accomplish this, generous exposure (fig. 643, inset) is essential. It is a very great help to have a 'nerve stimulator' which provides an intermittent galvanic current for this operation. If a strand of doubtful tissue is exposed, a touch with the nerve stimulator will immediately demonstrate whether it is a twig of the facial nerve or not.

1. **Extracapsular excision** is suitable for small superficial tumours. Unless *extracapsular* excision (fig. 643) is carried out, recurrence usually takes place within two years.

2. **Superficial lobectomy** is advised for larger tumours. The dissection

is commenced at the postero-inferior border of the parotid and the main trunk of the seventh nerve is found. As explained on p. 506, by meticulous dissection it is possible to follow and preserve the two divisions of the facial nerve and their branches between the two lobes. The superficial lobe having been mobilised on all sides, it is amputated. Most tumours lie in the superficial lobe (fig. 644).



FIG. 643.—Extracapsular excision of a moderate-sized parotid tumour. The tumour must be removed with a layer of healthy parotid tissue without encroaching upon the capsule. Inset shows the skin incision.

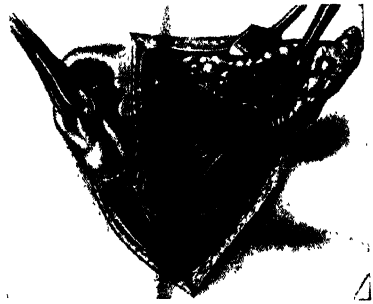


FIG. 644.—Superficial lobectomy nearing completion.

3. **Complete parotidectomy** is indicated (1) where the tumour has broken its confines and has commenced to enlarge comparatively rapidly, and (2) when the tumour has recurred after local excision.

While the patient must be warned that it may be impossible to preserve the facial nerve, the results of superficial lobectomy and total parotidectomy (fig. 645) show a low incidence of permanent facial palsy—considerably

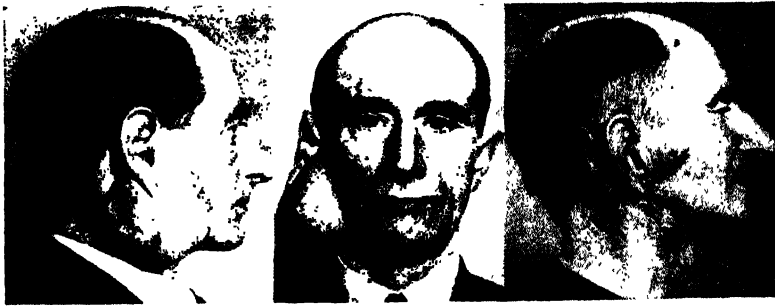


FIG. 645.—Mixed parotid tumour of over twenty years' duration, latterly increasing rapidly in size. Complete parotidectomy with preliminary ligation of the external carotid artery. The facial nerve was preserved. The patient was free from recurrence twelve years later.

lower than when extracapsular excision is performed for comparatively deep tumours. On the other hand, when the tumour is frankly malignant or is recurrent, it is often impossible, and indeed inadvisable, to spare the seventh nerve at the expense of incomplete extirpation of the growth.

Patients with malignant parotid tumours should always have radiotherapy after surgical extirpation. They are often inoperable when first seen and in this instance radiotherapy is the only feasible treatment; in these circumstances the prognosis is extremely poor. Block dissection (p. 535) usually on the side of the tumour is occasionally of value when operable metastatic lymph nodes are found after successful treatment of the primary tumour.



FIG. 646.—A strip of fascia lata inserted subcutaneously as shown helps to overcome the deformity of facial palsy. (After W. O. Lodge.)

the cheek becomes red, hot, and painful; this is followed by beads of perspiration appearing upon it. There is also cutaneous hyperæsthesia in front of and above the ear, especially noticed during shaving.

The explanation of the phenomenon is open to dispute. A satisfying hypothesis is that, presuming the nerve has been severed, axis cylinders conveying secretory impulses grow down the sheaths of the cutaneous element of the nerve. In this way a stimulus intended for saliva production evokes cutaneous hyperæsthesia and sweating.

The only effective treatment is avulsion of the nerve, but cases severe enough to merit this undertaking are few and far between. In most instances the patient, having tried many remedies, becomes resigned to endure the inconvenience; in a few, years of patience bring some amelioration.

MIKULICZ DISEASE

Mikulicz decreed the following triad as constituting the disease: (1) Symmetrical enlargement of salivary glands;

(2) Narrowing of the palpebral fissures due to involvement of the lachrymal glands;

(3) Parchment-like dryness of the mouth.

In its fully developed form (fig. 647) it can hardly be mistaken, although the disease can be limited to one parotid gland.

When associated with generalised arthritis the condition is known as Sjögren's syndrome.

Differential Diagnosis.—When limited to the parotid glands the condition usually is asymptomatic. The whole parotid is enlarged, which helps to distinguish the condition from a parotid tumour. Moreover, the incidence of bilateral occurrence is greater than that of any parotid neoplasm. Like parotid sialectasis, the swelling is stated to vary in size, but unlike sialectasis, the swelling is always present. A plain radiograph sometimes reveals small areas of calcification throughout the involved gland, a finding that is believed to substantiate chronic inflammation.

Sialography shows displacement of the duct system around a central mass, and always clubbing of the terminal ducts, which again favours chronic inflammation.

Pathology.—The microscopical findings reveal replacement of salivary tissue by lymphoid tissue containing islets of epithelial tissue, the amount of lymphoid replacement being proportional to the duration of the disease. While most pathologists

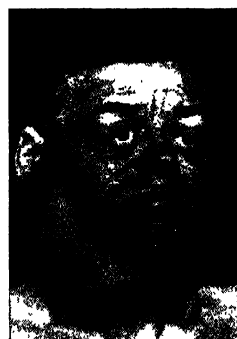


FIG. 647.—Mikulicz disease. (C. Fischer.)

Lucja Frey, 1889–1944. Physician, Neurological Clinic, Warsaw. She was killed during the German occupation of Poland.

Henrik Samuel Conrad Sjögren, Contemporary. Professor of Ophthalmology, University of Göteborg, and Head of the Eye Clinic, Jönköping, Sweden.

col of that the condition is due to an obscure chronic inflammation, few would ca state dogmatically that it is not a benign neoplasm.

Course.—A number of cases of spontaneous cure after several years have been r orted.

Treatment.—If a confident diagnosis can be made, X-ray therapy should be employed. When, at operation, the whole parotid gland is found to be enlarged, if possible frozen section biopsy should be performed, and if the diagnosis is Mikulicz disease, it may be considered unnecessary to proceed. On the other hand, massive deformity such as in the case illustrated (fig. 647) calls for removal of the submandibular salivary glands, and possibly bilateral superficial lobectomy of the parotids. The lachrymal glands are better left alone.

BOECK'S SARCOIDOSIS (p. 158)

This is a granuloma of unknown origin and can attack any tissue. In some parts of the world it has an ill-understood relationship with tuberculosis. In other parts (notably the U.S.A.) it is associated with inhalation of pine-pollen. Some cases are indubitably due to beryllium poisoning. It is usually, but not necessarily, associated with cutaneous lesions. When both parotid glands are attacked, Boeck's sarcoidosis simulates Mikulicz disease in several respects. If one parotid alone is involved, the swelling is usually diagnosed as a mixed parotid tumour. Sarcoid of the parotid, which is perhaps the most characteristic manifestation of Boeck's disease, is frequently associated with inflammation of the uveal tract (Iridocyclitis) (p. 159).

THE SUBMANDIBULAR SALIVARY GLANDS

Calculus.—The most common sites for a salivary calculus are within the submandibular salivary gland (fig. 648) or its duct (Wharton's duct). Indeed,



FIG. 648.—Salivary calculus within the submandibular salivary gland (actual size).

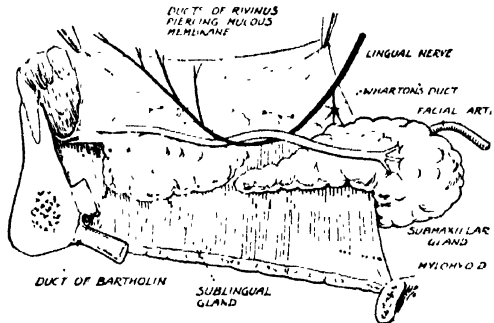


FIG. 649.—Dissection to show Wharton's duct and the structures in relation to it. (After Allen Thompson.)

they are more than fifty times more frequent here than in the parotid gland and its duct. These stones vary in size. One no larger than a millet seed may give rise to troublesome symptoms. At the other end of the scale specimens the size of a date-stone have been recorded.

Chemical analysis has shown that the composition of salivary calculi is mainly phosphates of calcium and magnesium, closely resembling that of tartar that collects upon the teeth.

Clinical Features.—Swelling of the gland (fig. 650) before or during meals is pathognomonic of the condition. In order to reproduce the swelling at the time of the clinical examination the patient should be given some lemon juice, or substitute, to sip. The orifices of Wharton's ducts are then examined, and compared. Saliva will be seen pouring forth on the non-



FIG. 650.—Enlargement of the submandibular salivary gland due to a calculus in Wharton's duct.

in this instance the radiograph is superfluous, for the stone can be felt. When a calculus or calculi is displayed in the gland itself the information gained is extremely valuable, but a negative X-ray does not eliminate a stone of comparatively low mineral content.

Treatment.—A stone in Wharton's duct should be removed under surface anaesthesia; injection into lax tissues is not advisable, as suffusion obscures the surgical field. Cotton wool, moistened with 5 per cent. cocaine, placed under the tongue for a few minutes, is entirely satisfactory. The tissues immediately behind the stone are grasped with tenaculum forceps, which steady the stone and elevate it. An incision is then made on to the stone in the long axis of the duct, and the stone slips out. The wound is left unsutured.



FIG. 652.—Mixed tumour of the submandibular salivary gland. Duration forty years.

affected side, whereas little if any is ejected on the side of the swelling. If a stone is in Wharton's duct, not infrequently it can be seen, and always it can be detected by bidigital palpation.

Salivary colic sometimes occurs, typically at the commencement of a meal. The pain is described by the patient as like toothache; on this account he is liable to be referred to a dental surgeon. The pain is also sometimes referred to the tongue due to irritation of the lingual nerve as it hooks around Wharton's duct.

Radiology.—A calculus (or calculi) in Wharton's duct casts a particularly clear shadow (fig. 651), but



FIG. 651.—Radiograph of stone in Wharton's duct.

When the stone lies within the submandibular salivary gland, extirpation of the gland is necessary (fig. 649).

An incision is made over the gland, the lower edge is dissected from the platysma, which is incised at a lower level and retracted upwards. In this way the cervical branch of the seventh nerve is protected from injury. The facial artery must of necessity be ligated as it traverses the gland.

Neoplasms.—Mixed tumours of the submandibular salivary gland (fig. 652) are comparatively rare. If diagnosed reasonably early, the treatment is eminently satisfactory, for the submandibular salivary gland can be excised *in toto* so readily. More rapidly growing neoplasms of the submandibular salivary gland, some of which are cylindromas, also occur.

ECTOPIC SALIVARY TUMOURS

Tiny, unnamed salivary glands are dotted here and there in the bucco-pharyngeal cavity; therefore it is not surprising that salivary tumours occur in locations other than those of the large salivary glands.

Ectopic salivary tumours are found in the palate, the cheek (fig. 608), the tongue (more often near its base), the floor of the mouth, the maxillary antrum, and the post-nasal space. The commonest site is the hard palate, and more than half of all cases are situated on the hard or in the soft palate. They make up some 7 per cent.

of all salivary gland tumours and roughly equal those of the submandibular gland in frequency.

Pathology.—Considerably under half of these neoplasms are mixed tumours. The remainder are cylindromas (Harrison).

Clinical Features.—The great majority of these tumours are quite symptomless and are first noticed accidentally. Occasionally they are reported by the patient within a few months of being discovered; usually they are concealed for much longer periods, but rarely for more than five years. By the time the patient reports, the surface of the tumour is often ulcerated (fig. 653 and fig. 527). When the tumour is situated in the base of the tongue the patient has difficulty in speaking; if it is in the maxillary antrum there is a painful swelling of the maxilla. In the most usual situation on the hard palate or in the cheek a clinical diagnosis is by no means impossible; in other situations, a biopsy report of a salivary tumour may cause consternation to those who have not encountered this condition. Unless the tumour is excised early and widely, local recurrence is relentless, and eventually metastases occur in the regional lymph nodes and sometimes in the viscera and skeleton.

Treatment.—In accessible situations such as the palate, the tongue, and the cheek, the tumour should be excised widely. Radiotherapy is advisable if excision is incomplete, but cylindromas are relatively radio-resistant.



FIG. 653.—Ulcerating palatal salivary tumour: commonest site.

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CHAPTER 23

THE NECK

THE BRANCHIAL APPARATUS AND ITS ABNORMALITIES

In a foetus approximately thirty-five days old, four grooves can be seen on each side of the neck. These are the branchial clefts, which resemble the gills of a fish; the intervening bars are the branchial arches. Each arch contains a central cartilage. The clefts in human embryos are composed of grooves on the outside (fig. 654) and pouches on the inside (pharynx). The first cleft persists as the external auditory meatus; the second, third, and fourth clefts normally disappear. The whole, or a portion, of one of the clefts that normally disappear may persist. Alternatively, a portion can become sequestered.



FIG. 654. — Foetus, showing branchial grooves and arches.

Branchial Cyst.—A cyst arising from the second branchial cleft is the most common of these vestigial remnants, and if there are tuberculous glands also present it may be mistaken for a tuberculous abscess. Usually the cyst makes its first appearance

between the twentieth and twenty-fifth years, but its advent may be postponed until the patient is over fifty. Its position is constant; it protrudes beneath the anterior border of the upper third of the sternomastoid (fig. 655). The cyst is nearly always lined by squamous epithelium, and its contents bear a striking resemblance to tuberculous pus.



FIG. 655. — Typical branchial cyst.

There is a rare variety of branchial cyst that is found lying closely related to the pharynx. It is lined by columnar epithelium, and filled with mucus. Occasionally small symptomless cysts of this type are discovered at necropsy.

If doubt exists, it is wise to aspirate the cyst, introducing the needle from above downwards (in case it is a tuberculous collar-stud abscess (p. 529) and may form a sinus). A drop is examined

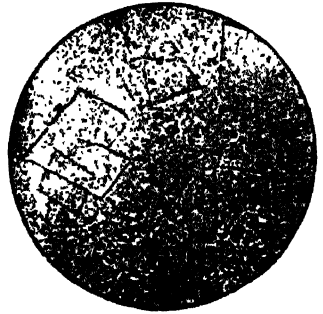


FIG. 656. — Branchial fluid. Note the abundance of cholesterol crystals (rectangular, with a notch at one corner).

under the microscope when cholesterol crystals will be seen (fig. 656). This confirms the diagnosis.

Treatment.—Excision of a branchial cyst through an incision following one of Langer's lines (fig. 681), gives uniformly satisfactory results. After the anterior wall of the cyst is exposed, some of the content is aspirated. This procedure permits the wall of the cyst to be grasped in suitable forceps, and assists materially in a dissection which, in some instances, entails following a

track that passes through the fork of the common carotid artery as far as the pharyngeal wall. The hypoglossal and the spinal accessory nerve may be in danger and should be recognised and protected. If the specimen is examined microscopically, the wall of the cyst is often found to be surrounded by a layer of lymphadenoid tissue. This suggests that the cyst arose as a result of branchial epithelium becoming entrapped within a lymph node during development. It also explains why branchial cysts become inflamed: the lymphadenoid shell participates in regional lymphadenitis.

Branchiogenic carcinoma undoubtedly does occur, but *very rarely*; such a diagnosis is unjustifiable until every possible source of a primary growth in the mouth, pharynx, and external auditory meatus has been scrutinised with a negative result (p. 534).

Branchial fistula may be unilateral or bilateral (fig. 657) and it is highly probable that the fistula represents a persistent second branchial cleft, the occluding membrane of which has broken down. Nearly always the external orifice of the fistula is situated in the lower third of the neck near the anterior border of the sternomastoid muscle. Branchial fistulæ which are clothed with muscle and lined by columnar ciliated epithelium discharge mucus, and are often the seat of recurrent attacks of inflammation. In cases where the lining epithelium has been exposed to chronic infection for a long time, its characteristics are lost.

When complete, commonly the internal orifice of the fistula is situated in the anterior aspect of the posterior pillar of the fauces, just behind the tonsil (Wilson). As a rule the track is incomplete and ends blindly in the region of the lateral pharyngeal wall; it is, therefore, strictly speaking, not a fistula, but a sinus. A branchial fistula is frequently a congenital condition, but it can be acquired. If a branchial cyst in an inflamed state is incised, the resulting sinus, which usually is situated in the upper or middle thirds of the neck, continues to discharge, either continuously or intermittently. The extent of the fistula can be determined by radiography following the injection of a radio-opaque medium (fig. 658).



FIG. 658.—Complete branchial fistula. External opening in the lower third of the neck; internal opening in the tonsillar region. Injected with uropac.

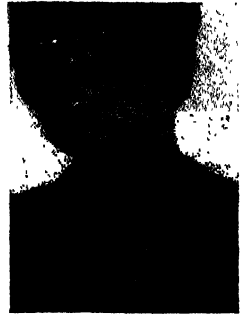


FIG. 657.—Bilateral branchial fistulæ; left side inflamed.

Treatment.—When causing troublesome symptoms, e.g. by a discharge of mucus, branchial fistulæ should be excised.

Special Pre-operative Measures.—In the case of a fistula without an internal opening, dissection can be facilitated in the following way: two or three days before

the operation a purse-string suture is inserted subcutaneously around the external orifice. After radio-opaque medium has been injected, the suture is tied, and subsequently radiographs are taken. Pent-up secretion and the medium distend the tract, which subsequently can be followed more easily in the depths of the wound.

When the fistula is complete, the insertion of a ureteric catheter along the tract immediately prior to the operation facilitates dissection.



FIG. 659.—The step-ladder method of removing a branchial fistula.

The operation should be conducted as follows : through an incision conforming with Langer's lines (fig. 681), and encompassing the orifice, dissection of the fistula proceeds in an upward direction as far as the limits of the wound permit. A second incision, parallel to the first, is then made at a higher level, and the mobilised portion of the fistula is brought out of it. The dissection of the fistula is then continued (fig. 659). The fistula is followed to its termination ; usually it passes through the fork of the common carotid artery and extends to the lateral pharyngeal wall.

Branchial Cartilage.—A small piece of cartilage, connected to the deep surface of a cutaneous dimple in the position of an external orifice of a branchial fistula, is occasionally encountered. Usually the patient finds it accidentally, and often thinks that it is a foreign body. Histologically, it is composed of typical cartilage cells.

Cervical Auricle.—So named because of its morphological significance, this cutaneous projection is found almost invariably in the position of the external orifice of a branchial fistula (fig. 660). Cervical auricles were recognised in the days of the Roman Empire, and are represented in some of the statuary of that period.



Fig. 660.—Cervical auricle.

Pharyngeal Pouch (p. 622) } are possibly derived from the branchial apparatus.
Laryngeal Pouch (p. 639) }

Cystic Hygroma

Ætiology.—About the sixth week of embryonic life the primitive lymph sacs develop in mesoblast, the principal pair being situated in the neck between the jugular and subclavian veins ; these, which correspond to the lymph hearts of lower animals, are known as the jugular lymph sacs. Sequestration of a portion of a jugular lymph sac and consequent failure of the isolated endothelial-lined space thus formed to link up with the tributary of the anlage of the lymphatic system, accounts for the appearance of these swellings.

Of all the swellings of the neck, cystic hygroma rivals, and at times surpasses, sternomastoid 'tumour' as the earliest to appear : usually it manifests itself during early infancy, occasionally it is present at birth, and exceptionally it is so large as to obstruct labour. Typically, the swelling occupies the lower third of the neck, and as it enlarges it passes upwards towards the ear (fig. 661); often it is the posterior triangle of the neck that is mainly involved. Due to intercommunication of its many compartments, the swelling is softly cystic and is partially compressible; it visibly increases in size when the child coughs or cries, but *the* characteristic that distinguishes it from all other cervical swellings is that it is brilliantly translucent.

The cheek and the axilla are other, though less frequent, sites for a cystic hygroma. Another infrequent, though striking dual lesion is that of a cystic hygroma and a lymphangiogenetic macroglossia. Exceptionally a cystic hygroma occurs in the groin or in the mediastinum. When situated wholly within the thorax (p. 685), it cannot be differentiated, prior to operation, from other benign neoplasms.

Pathology.—The swelling consists of an aggregation of cysts like a mass of soap bubbles. The larger cysts are near the surface, viz: → while the smaller ones lie deeply and tend to infiltrate muscle planes. Each cyst is filled with clear lymph and is lined by a single layer of endothelium having the appearance of mosaic.



FIG. 661.—Cystic hygroma. The swelling is brilliantly translucent. (The late Professor Sir John Fraser, Edinburgh.)

Clinical Course.—The behaviour of cystic hygromas during infancy is so uncertain that it is impossible at that age to prognosticate as to what will happen. Sometimes growth is extremely rapid and occasionally respiratory difficulty ensues, a contingency that demands immediate aspiration of much of the contents of the cyst and possibly tracheostomy. At other times, as a result of nasopharyngeal infection, the swelling becomes the seat of inflammation and spontaneous regression of the cyst may then occur.

Treatment.—Excision of the entire cyst at an early age is the treatment of election. It is often helpful to give preliminary injections, at weekly intervals, of boiling water into the cyst. The tumour will then slowly reduce in size and the cyst wall become more fibrous. Dissection is thus facilitated.

Operation.—Admittedly tedious, because it is necessary to follow to their terminations numerous finger-like projections, the walls of which are of tissue-paper-like thinness, the operation is not particularly difficult, provided anæsthesia is adequate. On no account must the cyst wall be picked up with dissecting forceps, because it will tear, and partial collapse of the cyst will result; the tissues must be dissected from the cyst. If the cyst is removed incompletely, there is a danger that during the early post-operative period there will be so much leakage of lymph that unless fluid balance can be maintained the child will become dehydrated. In all cases the wound must be drained for forty-eight hours.



FIG. 662.—Solitary lymph cyst of the neck.

Solitary lymph cyst is a condition akin to the foregoing, but differs in that it is nearly always first seen in adult life. As its name implies, it is a single cyst filled with lymph, and usually found in the supraclavicular triangle (fig. 662).

Treatment by excision is eminently satisfactory.

WEBBING OF THE NECK

This rare condition is usually associated with Turner's syndrome, which comprises webbing of the neck (fig. 663), an increased carrying angle of the elbow joints, and sexual infantilism. Turner's syndrome is one of several syndromes associated with chromosomal abnormalities (in this instance one sex chromosome is missing) but no satisfactory embryological explanation of the webbing has been put forward. It may well be an example of atavism, for it occurs in the chimpanzee.



FIG. 663.—Webbing of the neck.
(Geoffrey Flavell, F.R.C.S., London.)

In a little more than half of these the cervical rib is unilateral, and somewhat more frequent on the right side. It is paradoxical that a cervical rib or ribs found in the course of routine X-ray examination hardly ever gives rise to symptoms, whereas more often than not, when a radiograph of the cervical region is requested on account of nerve-pressure symptoms, no such rib is demonstrable.

Usually extra ribs spring from the seventh cervical vertebra (figs. 664 and 668) associated with spinal anomalies elsewhere.

Four main varieties of cervical rib are recognised:

(a) A complete rib, often containing a false joint in its length, articulates anteriorly with the manubrium or the first rib.—

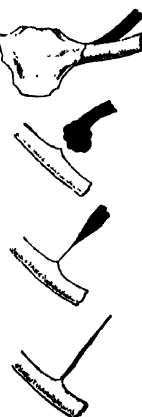
(b) The free end of rib expands into a large bony mass.—

(c) A rib ending in a tapering point, which is connected by a fibrous band to the scalene tubercle of the first rib.

(d) A fibrous band closely applied to, or incorporated in, the scalenus medius alone is present. This not infrequent variety, of course, cannot be demonstrated radiologically.



FIG. 664.—Cervical ribs that recurred after (subperiosteal) removal. Male aged forty-three years.



At their exit from the neck, the brachial plexus and the subclavian artery pass through a narrow triangle (fig. 665). It is to the *base* of the triangle that attention must be focused.

Pathology.—Should the base of the triangle be raised the height of one vertebra by the interposition of a cervical rib, the subclavian artery and the first dorsal nerve are bound to be angulated, if not compressed, as they pass over the new floor, to wit, that formed by the cervical rib, instead of the first thoracic rib.

Pathology of the Vascular Symptoms.—Owing to the angulation over the cervical rib, the lumen of the artery at this site becomes constricted. *Pari passu* with the formation of the constriction, fusiform dilatation of the first 2 to 4 cm. of the artery occurs distal

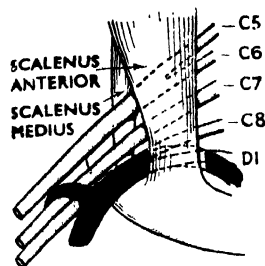


FIG. 665.—The anatomy of the parts concerned. (After the late Professor Lambert Rogers, F.R.C.S., Cardiff.)

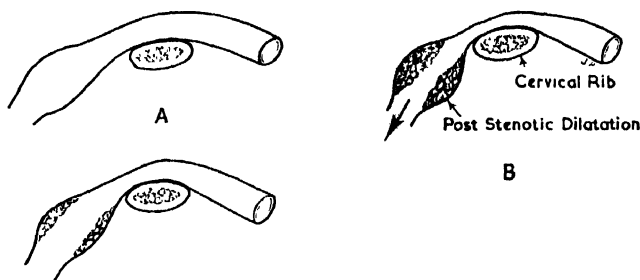


FIG. 666.—A, showing the narrowing and post-stenotic dilatation of the vessel, with early thrombus formation; B, an embolus has been thrown off.

to the constriction (fig. 666A.) Within the post-stenotic dilatation (or possibly at the site of the constriction) clotting occurs on the intima. Portions of this mural thrombus may become detached, and give rise to an embolus (fig. 666B) or emboli.

Pathology of the nerve-pressure symptoms is far less factual than the foregoing. It is surmised that the first dorsal nerve becomes similarly angulated and fibrosed.

Three clinical types are encountered:

1. **Cervical Rib with Local Symptoms.**—From time to time a patient presents on account of a lump in the lower part of the neck which may be visible, or, more commonly, because of tenderness in the supraclavicular fossa. On palpation the lump is found to be bony hard and totally fixed. Type (b), (p. 522), is most likely to give rise to these signs.

2. **Cervical Rib with Vascular Symptoms.**—*Vascular symptoms* occur only when a cervical rib is complete.

Pain is the prevailing symptom. It is located in the forearm, but in some instances it radiates to the upper arm. What is so characteristic is that the pain is brought on by use of the arm: should the arm be in a raised position at the time of exercising it, the onset of the pain is accelerated. The pain is relieved by rest. Without doubt, this pain is ischæmic muscle pain, comparable in all respects to intermittent claudication in the leg.

Temperature and Colour Changes.—Some observant patients notice that the hand on the affected side (a) is colder than its fellow, (b) becomes unduly pale when held aloft and (c) becomes unduly blue when it is dependent for any length of time. In any event the arm should be suitably positioned to ascertain if colour changes can be produced. At the same time the radial pulse should be sought. Sometimes it is as full as that of the other side; sometimes it is absent, and at others it is feeble, depending upon whether the collateral circulation is good, bad or indifferent. The distal part of the subclavian artery should be auscultated; a systolic bruit is significant.

Numbness of the fingers may be complained of, in which event trophic changes with ulceration or, more rarely, gangrene (fig. 667) are liable to ensue. In these patients it is not unusual for the brachial, as well as the radial, pulse to be imperceptible, and arteriography may show that the greater part of the brachial artery is occluded.

Treatment is timely extraperiosteal excision of the cervical rib, together with any bony prominence of the first rib. At the same time it is advisable to perform sympathetic denervation of the upper limb.



FIG. 667.—Cervical rib with vaso-motor symptoms, culminating in gangrene of the index finger. (Telford and Stopford.)

3. Cervical Rib with Nerve-Pressure Symptoms.—Nerve-pressure symptoms, due to angulation of the first dorsal nerve is of doubtful occurrence and most, if not all, cases previously described were due to cervical spondylosis (p. 425) or the carpal tunnel syndrome (p. 312). Only if these and other localised nerve lesions are excluded, should the scalene syndrome be considered as the cause of pain and tingling in the hand and forearm, whether wasting of the thenar and hypothenar muscles is present or not.

Differential Diagnosis.—When nerve-pressure symptoms are present the differential diagnosis must be made from progressive muscular atrophy, syringomyelia, and peripheral nerve injury. Above all, newer pathological concepts must be taken into consideration. First, many of the nerve-pressure symptoms formerly attributed to a cervical rib can be, and are, produced by pressure on the cervical roots in the region of the intervertebral foramina by lateral protrusion of intervertebral discs. Secondly, acroparæsthesia and wasting of the thenar eminence are often due to pressure on the median nerve at the wrist, in which case it can be cured easily by division of the flexor retinaculum (p. 312). Thirdly, hypothenar wasting can also arise from angulation of the ulnar nerve behind the elbow (Griffiths).

Treatment.—In mild cases the use of a sling and exercises aimed at strengthening the muscles of the shoulder girdle may alleviate the symptoms, at least temporarily. In about 70 per cent. of cases even if a cervical rib cannot be recognised, the symptoms are relieved by dividing the scalenus anterior (scalenotomy) (fig. 668). Other surgeons remove the cervical rib or the corresponding band in addition, and in this way reduce the number of unsatisfactory results. When a cervical rib is excised, it is essential to remove it with its periosteum or it will probably regenerate (fig. 664).

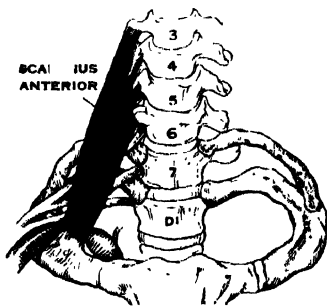


FIG. 668.—Showing the rationale of scalenotomy.

INJURIES

Cut Throat.—In more than half the cases of cut throat that reach surgical aid the wound does not involve any vital structure—only the skin, platysma, and perhaps the sternomastoid or other muscles are severed. Even the external jugular vein does not necessarily come under the category of a vital structure in this respect. The treatment of these superficial injuries follows elementary surgical principles.

Serious Cases.—Self-inflicted wounds of the neck are usually perpetrated with the head extended, the wound being more or less transverse. In this extended position the great vessels of the neck are protected by the sternomastoids and the larynx. Thus the great vessels of the neck are comparatively rarely injured, while the air passages bear the brunt.

Treatment.—Attention is directed, firstly, to arresting hæmorrhage; secondly, to dealing with the wounded air passage; and, thirdly, to the repair of other structures.

Various moderate-sized arteries and veins will require ligation. Hæmorrhage from an inaccessible branch of the external carotid is best dealt with by ligating that trunk near its origin. Injury to the main vessels is comparatively rare, and when it occurs, death usually supervenes before surgical aid is forthcoming.

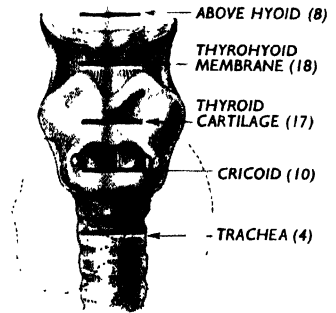


FIG. 669.—Position of the wound into the air passages of fifty-seven cases of suicidal cut throat with a deep wound.

The principal sites of wounds of the air passages are indicated in fig. 669.

Wounds above the Hyoid Bone.—After cleansing the area the wound is explored with a finger. Quite often it will be found that the cavity of the mouth has been entered. The epiglottis is often partially divided near its base. This should be repaired with catgut sutures. The mucosa of the pharynx is trimmed and united. Skin closure can then be undertaken.

Wounds of the Thyrohyoid Membrane.—Again the epiglottis is often damaged. The severed thyrohyoid membrane can usually be sutured. If there is respiratory distress, it is advisable to perform tracheostomy.

Division of the Thyroid Cartilage.—The thyroid cartilage can be repaired with sutures, provided these are not tied tightly, for a stitch through cartilage tends to cut out. Tracheostomy is usually indicated.

Wounds about the Cricoid Cartilage.—Tracheostomy should be performed. The larynx is repaired with interrupted sutures, and after debridement the wound is closed with drainage.

Division of the Trachea.—Wounds of the trachea are comparatively rare. In order to obtain adequate exposure it is usually necessary to divide the thyroid isthmus between hæmostats. In most instances it is advisable to perform tracheostomy below the wound, and then to proceed to repair the latter with sutures.

Injury to Nerves.—It is remarkable how rarely important nerves are injured in self-inflicted wounds. In stab wounds any nerve may be involved. In one of our patients, a sailor, the most inaccessible nerve in the neck, the cervical sympathetic, was divided in this way, the assailant's weapon being a small penknife.

COMPLICATIONS OF CUT THROAT

1. **Loss of Blood.**—If the hæmorrhage has been severe, dextran, plasma, or blood transfusion is indicated.

2. **Air Embolus.**—In cases encountered in medico-legal practice the cause of death is frequently venous air embolism (Keith Simpson).

3. **Infection of the wound** is not very frequent, but these wounds should always be drained as they have been inflicted with a potentially infected instrument. Cellulitis sometimes supervenes, and this may spread to the mediastinum.

4. **Pneumonia.**—In spite of antibiotic therapy, pneumonia is a relatively common complication, especially in those cases where the air passages have been opened.

5. **Aerial Fistula.**—A persistent communication between the air passages and the exterior is likely to occur when there has been actual loss of substance of the larynx or trachea. In suitable cases a plastic operation may be undertaken.

6. **Stenosis of the Larynx or Trachea.**—Due to cicatrization; rarely it necessitates permanent tracheostomy.

7. **Oesophageal Fistula.**—Oesophageal or pharyngeal fistula is a very rare occurrence, and it tends to heal spontaneously.

8. **Surgical emphysema** is another rare complication, and it usually occurs when a tracheostomy tube has been omitted in the treatment of the case.

9. **Aphonia or dysphonia** may follow injury to the vocal cords or division of a recurrent laryngeal nerve.

WOUNDS OF THE CERVICAL PORTION OF THE THORACIC DUCT

Wounds of the thoracic duct are rare, and usually occur during dissection of lymph nodes in the left supraclavicular fossa. When the accident is not recognised at the time, chyle pours from the wound—as much as 2 or 3 pints (1 to 1.5 L.) in twenty-four hours—and, as a result, the patient wastes rapidly.

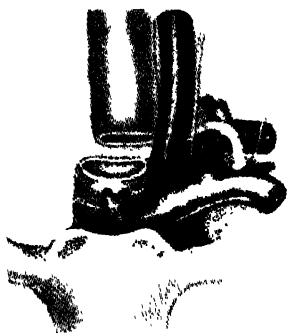


FIG. 670.—The termination of the thoracic duct.

Treatment.—Should the accident be recognised during an operation, the proximal end of the duct must be ligated with fine silk. Ligation of the duct is not harmful, for there are a number of anastomotic channels between the lymphatic and the venous systems in the neighbourhood. Usually the first intimation of a severed thoracic duct is a copious chylous discharge from the wound on the day following the operation. That the fluid is chyle is substantiated if it has a specific gravity of over 1.012 and if fat can be extracted from it with ether. Firm pressure by a pad and bandage should be applied, but this simple expedient is seldom successful. More often the wound must be reopened. If the patient is given cream to drink an hour before the operation, more especially

if the cream is coloured with confectioners' green dye (D-C 6), there is seldom any difficulty in locating a cut thoracic duct, which is about the size of a straw and an immediate external relation of the last 1½ inches (3.75 cm.) of the left internal jugular vein (fig. 670). If the duct is found, it should be ligated, but in any case the wound should be packed firmly and allowed to heal by granulation. Thanks to subsidiary anastomotic channels, these measures are regularly satisfactory.

INFLAMMATORY CONDITIONS

Acute cellulitis is either superficial or deep to the deep cervical fascia.

Superficial cellulitis is common, and methods of treating it follow that of cellulitis elsewhere. When, however, it occurs above the level of the hyoid bone it is especially dangerous, because sudden asphyxia from oedema of the glottis is an ever-present possibility.

Deep cellulitis in the lower third of the neck, on the other hand, is free from this danger. Consequently, it can be treated by antibiotic therapy with every confidence, and should an abscess develop, it is opened.

There are three closed fascial spaces in the upper third of the neck, all of

which require early decompression if symptoms and signs persist after a short trial of antibiotic treatment. The three suprahoid infections of the neck are Ludwig's angina, infection of the pharyngomaxillary space, and infection of the masticator space.

Ludwig's Angina.—Ludwig described a clinical entity characterised by a brawny swelling of the submandibular region combined with inflammatory oedema of the mouth. It is the *combined* cervical and intrabuccal signs that constitute the characteristic feature of the lesion (fig. 671). The cause is a virulent (usually streptococcal) infection of the cellular tissues surrounding the submandibular salivary gland.

Clinical Course.—Unless the infection is controlled, these cases may rapidly assume a grave aspect. The swollen tongue is pushed towards the palate and forwards through the open mouth, while the cellulitis extends down the neck in that most dangerous plane—deep to the deep fascia.

Ludwig's angina is an infection of a closed fascial space, and, untreated, the inflammatory exudate often passes via the tunnel occupied by the stylohyoid to the submucosa of the glottis, in which event the patient is in imminent danger of death from oedema of the glottis.

Treatment.—When the condition is diagnosed early, the results of antibiotic therapy are sometimes dramatic. In cases where the swelling, both cervical and intrabuccal, does not subside rapidly with such treatment, a curved incision beneath the jaw, as is shown in fig. 672, is made. The incision is deepened, and after displacing the superficial lobe of the submandibular salivary gland, the mylohyoid muscles are divided. This decompresses the closed fascial space referred to. The wound is lightly sutured and drained. The operation can be conducted with the greatest safety under local anaesthesia.



FIG. 672.—Incision for decompressing thoroughly the space beneath the mylohyoid muscle.



FIG. 671.—Ludwig's angina. The brawny swelling beneath the mandible and the oedema of the floor of the mouth are characteristic.

Infection of the Pharyngo-maxillary Space (Parapharyngeal Abscess).—The pharyngo-maxillary space is a potential cone-shaped space, base uppermost. The base is formed by the base of the skull; the apex abuts the great cornu of the hyoid bone; the medial wall consists of the superior constrictor muscle; the lateral wall, from above downwards, is composed of the fascia covering the internal pterygoid muscle, the mandible about its angle, and the submandibular salivary gland, below which the apex of the space becomes relatively superficial. Usually infection of this space originates in the tonsil, and may occur after tonsillectomy, especially when the operation has been performed under local anaesthesia.

Clinical Features.—Every posterior, peritonsillar abscess is a potential pharyngomaxillary space infection, the general reaction of which is greater than that accompanying peritonsillar abscess. There is often *slight* trismus, and swelling over the lower part of the parotid gland: this is never present in quinsy. Tenderness, and later swelling below the angle of the mandible, appears when the apex of the space is involved. As the carotid sheath runs through the space, the dreaded complications of thrombophlebitis of the internal jugular vein and/or erosion of an artery, usually the internal carotid, may occur if the space is not adequately drained. Sometimes the abscess bursts spontaneously between the cartilaginous plates of the external auditory canal, but obviously such an eventuality is a fortunate escape from death, for which the patient's medical advisers can take no credit.

Treatment.—As soon as the diagnosis is strongly suspected an incision should

be made below and behind the angle of the mandible, on a line towards the hyoid bone. A finger is passed upwards, medial to the mandible and the distended space is entered by rupturing its wall. The space is drained with a large soft wick drain.

CERVICAL LYMPHADENITIS

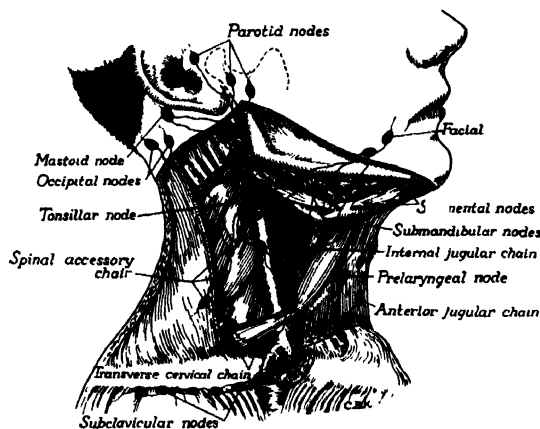


FIG. 673.—The lymphatics of the facio-cervical region.
(After Murry M. Copeland.)

is a varying degree of pyrexia. The treatment, in the first instance, is directed to the general condition and to the focus of infection, the neck itself being simply protected by a bandage over wool. If, in spite of antibiotic therapy, pain continues or certain lymph nodes appear to be getting larger, fomentations are applied locally. Abscess formation calls for adequate drainage.

Chronic Lymphadenitis.—In the early stages it is extremely difficult to distinguish tuberculous adenitis from subacute (fig. 674) and chronic non-tuberculous



FIG. 674.—Subacute non-tuberculous cervical lymphadenitis.

adenitis, but clinical experience shows that chronically inflamed lymph nodes which do not resolve in the space of three or four weeks are nearly always tuberculous.

Tuberculous cervical adenitis is now less common in the British Isles though it still occurs. The majority of patients affected are children or young adults, but the condition can occur for the first time at any age. Usually one group of cervical nodes is first infected (fig. 675), most frequently those of the upper jugular chain. More rarely there is widespread cervical lymphadenitis, and in these cases especially, periadenitis or matting of the lymph nodes is evident.

Source of Infection.—In the majority of instances tubercle bacilli gain entrance through the tonsil of the corresponding side. Sometimes tubercle bacilli can be

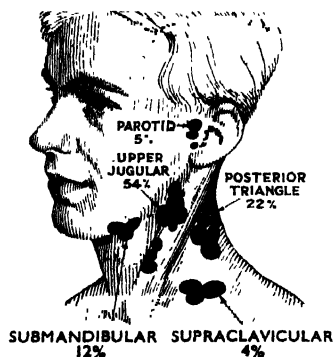


FIG. 675.—Groups of cervical lymph nodes infected by tuberculous, founded on 372 consecutive cases. The 3 per cent. not labelled are divided between the mid-jugular group (shown) and the submental group (not shown).

demonstrated in carious teeth, and this is a portal that should be suspected when the submandibular or submental groups of lymph nodes are principally affected. The frequency (22 per cent.) with which the nodes of the posterior triangle are infected is more difficult to explain. It is unlikely that tubercle bacilli often gain entrance through the scalp, which is rightly looked upon as the main portal of these nodes for other infections. Seeing that some of the lymphatic vessels from the adenoid area pass directly to the lymph nodes of the posterior triangle, it is probable that air-borne infection reaches them through this often-diseased filter.

Contrary to what is believed generally, it is the human,¹ and not the bovine, bacillus that is responsible for tuberculous cervical adenitis in about 70 per cent. of cases. In fully 80 per cent. of cases the tuberculous process is virtually limited to the clinically affected group of lymph nodes; nevertheless, especially in widespread adenitis and in that occurring in the supraclavicular fossæ, a primary focus in the lungs must be suspected. Renal tuberculosis may co-exist and the urine should be tested for this organism.

In the event of the patient developing a natural resistance to the infection or (more often) as a result of appropriate general treatment, fibrosis or calcification may occur. In other circumstances the caseating material (fig. 676) liquefies, breaks through the capsules of the lymph nodes, and a



FIG. 676.—Caseating tuberculous lymph nodes.

'cold abscess' forms. The pus is at first confined by the *deep* cervical fascia. In a few weeks this dense sheet becomes eroded at one point, and the pus flows through the small opening into that more commodious space beneath the *superficial* fascia. The process has now reached the well-known stage of collar-stud abscess (fig. 677 and p. 154). The superficial abscess enlarges steadily, and unless suit-

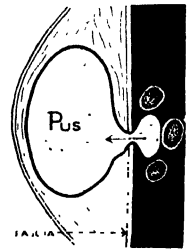


FIG. 677.—Showing the abscess and the source of the pus beneath the deep fascia.

able treatment is adopted, the skin will soon become reddened over the centre of the fluctuating swelling, and before long a discharging sinus, with its attendant evils, is at hand.

The various clinical stages are illustrated in figs. 678, 679, and 680.



FIG. 678.—Tuberculous cervical adenitis. Tonsillar lymph node chiefly involved.



FIG. 679.—Tuberculous collar-stud abscess. (Iodine has been applied to the skin.)



FIG. 680.—If the skin becomes involved and a discharging sinus results, sometimes, as in this case, the external opening of the sinus is at a distance from the original lesion (ringed).

¹ The human bacillus is responsible for 100 per cent. of cases among the Bantu-speaking urban population of South Africa (Keen).

Paul Keen, Contemporary. Formerly Senior Surgeon, Non-European Hospital, Johannesburg, South Africa.

DIFFERENTIAL DIAGNOSIS

When the swelling is solid, from {

 Chronic non-tuberculous lymphadenitis.
 Hodgkin's disease (p. 156).
 Reticulo-sarcoma.
 Secondary malignant disease.

When the swelling is cystic, from {

 Branchial cyst.
 Extension of an abscess connected with a tuberculous cervical vertebra.

When a sinus or sinuses have formed, from {

 Actinomycosis.
 An acquired branchial fistula.

TREATMENT OF TUBERCULOUS LYMPHADENITIS¹

General Treatment.—The patient should be placed under the best dietetic and hygienic conditions, the most important being open air, together with natural or artificial sunlight.

No matter what line of treatment is adopted, hypertrophied adenoids, infected tonsils, and carious teeth must receive appropriate attention.

Conservative Measures.—For more than half a century opinion has vacillated between conservative treatment—best carried out in a sanatorium—and operative treatment. The introduction of antibiotic therapy added zest to non-operative treatment, but early hopes that it might supplant operative treatment have not been fulfilled.

Chemotherapy (p. 23).

Contraindications to Operative Treatment.—When there is active tuberculosis of another system, e.g. pulmonary tuberculosis, removal of tuberculous lymph nodes of the neck is, of course, illogical. In cases where enlarged lymph nodes are not confined to one region in the neck, where there is much periadenitis, or any discharging sinuses, operative treatment is best avoided, at any rate for several months, during which time sanatorium treatment is advised. Repeated aspiration of a collar-stud abscess cannot be recommended unless the patient is under constant supervision in a sanatorium, because so frequently this predisposes to sinus formation and secondary infection.

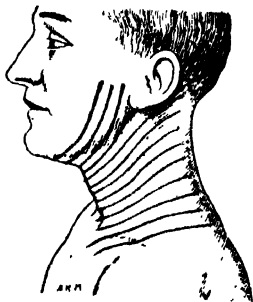


FIG. 681.—Langer's lines: cervical region.

Operative Treatment.—In the majority of cases the tuberculous process is limited to one group of lymph nodes. For this type, extirpation of the affected nodes through an oblique incision (fig. 659), following one of Langer's lines (fig. 681), gives rapid and eminently satisfactory results.

¹ For 600 years the King's touch was believed to cure this prevalent disease. Charles II touched on an average 10,000 sufferers a year. In addition, he presented each with half a sovereign.

During the dissection of cervical lymph nodes no effort should be spared to preserve : the spinal accessory nerve ; the mandibular branch of the facial nerve ; the hypoglossal nerve ; which are the nerves most likely to be injured.

To minimise unnecessary injury to large veins, no tissue should be divided when stretched taut. Should the internal jugular vein prove to be involved to such an extent that freeing it is difficult or impossible, this great vessel can be ligated, or a portion of it can be resected, without any untoward effect at any age (cf. ligation of the common carotid artery, p. 129).

There is no contraindication to dividing the sternomastoid muscle if, as is frequently the case, such a step facilitates access to the diseased nodes and enables the operator to visualise the spinal accessory nerve more easily. The divided muscle is subsequently reunited.

Even the presence of a collar-stud abscess does not jeopardise healing by first intention if the diseased lymph nodes lying beneath the deep cervical fascia are displayed by enlarging the small communicating opening (fig. 682), and excised completely, provided the overlying skin is healthy and hæmatoma formation is guarded against by careful hæmostasis and a pressure dressing.

When the overlying skin is involved and/or sinuses are present, the unhealthy skin should be excised, and after the diseased lymph nodes have been removed, the wound is packed with petroleum-jelly gauze and a viscopaste bandage is applied. Large gaping wounds often heal with a linear scar.



FIG. 682.—Seeking the opening in the deep fascia. The abscess cavity is lined by soft granulation tissue which tends to obscure the small hole in the deep fascia.

ACTINOMYCOSIS OF THE NECK

Two-thirds of all human cases of actinomycosis occur in the neck and face.

Ætiology (p. 22).

Clinical Features.—A sinus or sinuses about the upper part of the neck (fig. 683), particularly indurated sinuses, should arouse suspicion immediately. The skin about the sinus often appears somewhat bluish or violescent in colour. On palpation each burrow feels hard, like a strand of whipcord. Lymph nodes are seldom enlarged except as the result of secondary infection. There is no pain unless the disease is advanced and nerves have become implicated in fibrous tissue. As the disease progresses the patient becomes increasingly anæmic. The discharge is extremely characteristic. It is thin and watery, and typically it contains 'sulphur granules'.



FIG. 683.—Actinomycosis. Indurated sinuses extend from the neck to the orbit.

Early in the course of the disease the granules are small and difficult to see macroscopically. A swab of the discharge is insufficient for the pathologist to make a definite diagnosis; the clinician should collect a few ml. of the purulent exudate in a sterile test-tube, and first examine it himself by holding it to the light. The presence of sulphur granules is extremely suspicious, but fragments of necrotic or caseous material can

James Israel, 1848-1926, first found 'sulphur granules' in pus from a discharging sinus of the neck in man. He afterwards became a famous Berlin Urologist.

simulate them. The finding of sulphur granules containing Gram-positive mycelia, from which are isolated anaerobic actinomyces, clinches the diagnosis.

Modes of Spread.—The disease spreads by burrowing in the connective tissues, upwards towards the scalp and downwards into the supraclavicular region, whence the mediastinum and pleural cavities become involved. Spread by the lymphatic stream is practically unknown, and it is truly remarkable that this favourite channel for the dissemination of all other infective processes should enjoy such a degree of immunity in the case of the ray fungus. The usual explanation of this is that actinomyces are too large to pass along a lymphatic vessel. Late in the course of the disease blood-borne metastases occur, notably in the liver and the brain. Untreated, the disease runs a chronic but surely fatal course.

Treatment.—The dental surgeon attends to carious teeth, erring on the side of extraction rather than repair.

Antibiotic Therapy.—Five to ten million units of penicillin daily for six weeks is curative in a number of cases, but it is necessary to be guided by the sensitivity tests as to which antibiotic to prescribe. Not a few strains of actinomyces are now, or always have been, resistant to penicillin but may be sensitive to tetracycline or other antibiotics. What is extremely important to realise is that penicillin-resistant associated bacteria are capable of maintaining activity of the lesion, and the appropriate antibiotic to which the bacteria are sensitive should therefore be prescribed.

In cases where the discharge persists, a bottleneck sinus should be suspected, in which case the sinus or sinuses should be slit up and packed with gauze soaked in 2·5 per cent. tincture of iodine. In former days many cures resulted from giving 5 minims (0·3 ml.) of tinct. iodine in milk, and increasing the dose to 10 minims (0·6 ml.) t.d.s. In refractory cases this simple treatment may still be tried.

BENIGN NEOPLASMS OF THE NECK

Deep Cavernous Hæmangioma.—Like a lymph cyst (p. 520), a hæmangioma can be emptied by pressure, but it is non-translucent. In some instances it commences in the retropharyngeal space and extends into the neck.

Treatment.—The effect of injecting a sclerosing agent should be tried first. Excision may prove a difficult and dangerous undertaking; in cases which prove at operation to be less circumscribed than anticipated, multiple ligation is the safer course.

Neurilemmoma (*syn.* **Schwannoma**) is a tumour of the neurilemma, or nerve sheath of Schwann. It is characteristically benign, smooth, encapsulated, soft, and gelatinous. It arises from the vagus, cervical sympathetic chain, or the glossopharyngeal nerves or, indeed, from any of the nerves. The tumour therefore is inconstant in position. Although innocent, it gives rise to compression of adjacent vital structures, and is often mistaken for a malignant tumour.

A neurilemmoma of the vagus should be strongly suspected if there is medial displacement of the internal carotid artery as well as dysphagia and hoarseness; the coughing and choking attacks that are often present are due to involvement of the superior laryngeal nerve. The tumour must be removed in its entirety which, except when situated in the supraclavicular fossa, does not present particular difficulties. These tumours are completely radio-resistant.

Lipoma.—The ubiquitous lipoma, usually subcutaneous, can appear anywhere in the neck and a very common situation is in the nape of the neck.

PRIMARY MALIGNANT TUMOURS OF THE NECK

Carotid Body Tumour ('Potato' Tumour).—The carotid body, which is situated at the bifurcation of the carotid artery, is the most important

Hans Christian Joachim Gram, 1853–1938. Professor of Medicine, Copenhagen, Denmark.

Otto von Bollinger, 1843–1909, Professor of Pathology, Munich, first detected the branching mycelium in pus from the diseased jaw of a cow.

Theodor Schwann, 1810–1882. Professor of Anatomy, Liège, Belgium.

moiety of the chemoreceptor system (fig. 684). The cells of this system are sensitive to changes in the hydrogen ion concentration and temperature of the blood.

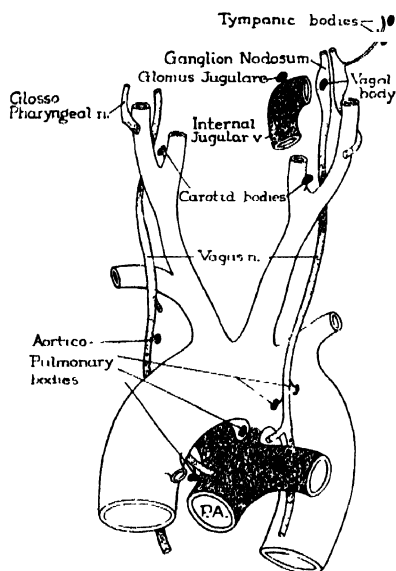


FIG. 684.—Sites where chemodectomas are known to occur. In addition, they have been found in the orbit, along the course of the inferior alveolar artery, and in proximity to the femoral artery. (After S. O. Burman.)

Pathology.—Histologically a non-chrom-affin paraganglioma, a carotid body tumour is now classified as a chemodectoma. The former belief that this tumour is benign has been dissipated. Although, as a rule, the tumour remains localised for years, eventually regional metastases occur in about 20 per cent. of cases, and distant metastases somewhat less frequently.

Clinical Features.—Nearly always unilateral, a carotid body tumour is considered difficult to diagnose because, contrary to expectation, it often exhibits less transmitted pulsation than the solid lumps from which it has to be differentiated. Usually the tumour first becomes apparent in middle life, and increases in size



FIG. 685.—Carotid body tumour of twelve years' duration.

very slowly; indeed, it behaves like a mixed parotid tumour (p. 512). Unfortunately, as a rule the lump is at least the size of a plum before the patient seeks advice (fig. 685). The diagnosis is suggested by a long history, a mass at the carotid bifurcation which moves from side to side but not vertically and, usually, a pulsating vessel (external carotid) over its outer surface (Westbury).

The Peculiar Danger of Excision of a Carotid Body Tumour.—While recurrence after complete excision is most unusual it is important to realise that if the tumour is large it is probably so blended with the carotid tree that removal is impossible without resecting the fork of the carotid artery (fig. 686), and unless special precautions are taken, ligation of the internal carotid artery is followed by death or hemiplegia in 33 per cent. of cases (Lahey).



FIG. 686.—Carotid body tumour removed successfully from a female. Glass rods are in the carotid tree.

Arteriography is valuable and usually shows the carotid fork to be splayed and a mass of abnormal tumour vessels are outlined. Confirmation by *open biopsy* may be necessary as the operation carries the risk of carotid artery damage.

Treatment.—Hardly any of these tumours are radio-sensitive. Extirpation should therefore be carried out in all cases except in the old and enfeebled, in whom it may be decided that it is best to let Nature take her course. The tumour is explored through an ample incision:

1. In some cases it can be separated from the fork of the carotid artery by blunt and sharp dissection, and so removed.

2. In comparatively early cases excision can sometimes be effected in the manner shown in fig. 687. Should a main artery be wounded in the process it can, on occasions, be repaired.

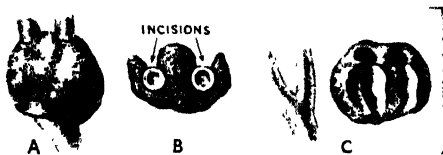


FIG. 687.—The tumour is split in the manner shown, so that it can be dissected from the arteries it envelops. (After T. Farrar *et al.*)

3. When the tumour is large and inseparable from the fork of the carotid artery, resection with the arteries, as is shown in fig. 686, must be carried out. It is essential to maintain the blood supply to the brain and a graft—autogenous vein, homologous artery, or synthetic prosthesis—between the common and internal carotid will be required. Hypothermia of 29 to 30° C. is the method of choice to enable the

graft to be inserted while the oxygen requirements of the brain are reduced to a minimum.

Hodgkin's Disease (syn. Lymphadenoma in neck), see p. 156.

Reticulosarcoma (including Lymphosarcoma in neck) see p. 155.

SECONDARY CARCINOMA OF THE NECK

Secondary carcinomatous infiltration of the cervical lymph nodes is only too common. When a patient presents with enlargement of cervical lymph nodes that are suspiciously indurated, search for a primary growth is imperative. Often the primary growth lies within the buccal cavity; when this is not the case, the search must continue. Among the sites that are prone to be overlooked are the pharynx and cervical œsophagus, the external auditory meatus, the bronchi, the breast, the stomach, and the testes.

Management.—In so far as cervical lymph nodes are concerned, patients with oral, pharyngeal, or facial carcinoma can be categorised as follows:

Stage 1.—Those without clinical evidence of metastases. These patients are kept under careful (bi-monthly) observation, dissection of the neck being withheld unless the nodes become palpable. Exceptions to this rule are (1) melanoma, (2) papilliferous carcinoma of the thyroid, and (3) possibly cases of carcinoma of the tongue (see remarks under (e), p. 504).

Stage 2.—Those with palpable cervical lymph nodes where the primary growth is operable, or can be destroyed with radium. It should be noted carefully that lymph node enlargement, especially when the primary growth is foul, is not necessarily carcinomatous. After removal of the primary neoplasm a period of non-intervention and antibiotic therapy will determine whether or not the enlargement subsides. In the latter event block dissection of the neck is indicated, provided, of course, there is no clinical or X-ray evidence of distant metastases, and the involved cervical nodes have not progressed to a stage where it seems unlikely that they can be encompassed. Adherence to the mandible, or to the larynx, or to the skin, should not be regarded as constituting inoperability, for each of these structures can be sacrificed, provided the patient's general condition is such as to be able to withstand either of the first two procedures, which are extremely formidable.

Stage 3.—Those in whom the primary growth is inoperable or otherwise

incurable, and/or the cervical metastases are fixed to deeper structures, and/or distant metastases are present. Palliative deep X-ray treatment is sometimes indicated, but there is always the risk that high dosage of irradiation will cause necrosis of the mandible or the laryngeal cartilages. In some patients irradiation is ill-advised, as it may cause unnecessary suffering, without conferring commensurate benefit.

Block dissections of the neck are of two varieties:

1. **Crile's block dissection** is conducted through the wide display afforded by skin incisions, such as are shown in fig. 688.

The skin flaps having been dissected up, the sternomastoid is divided about 1 inch (2.5 cm.) above the clavicle. The muscle is freed and retracted upwards. Next, the internal jugular vein is divided between ligatures low down in the neck. The dissection proceeds upwards methodically and the muscle, fascia, fat, lymph nodes, the internal jugular vein, together with the submaxillary salivary gland, are dissected and removed *en bloc*. Attention must be directed to clearing the space between the parotid and the great vessels, and also the submental triangle between the hyoglossi, for



FIG. 688.—An incision for block dissection of the neck.

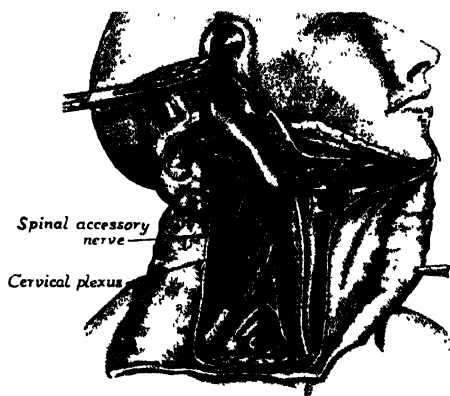


FIG. 689.—Crile's block dissection of the neck nearing completion. (After W. W. Carroll.)

it is in these areas that a lymph node can easily be overlooked. Bleeding vessels are ligated as they occur; finally the upper end of the internal jugular vein is ligated by transfixion, and divided. When the dissection has been completed, the carotid artery is laid bare, and lying with it is the vagus nerve, which has been carefully preserved. The operation aims at removing the whole of the lymphatic-bearing tissues on the affected side of the neck (fig. 689). The skin flaps are approximated and the wound drained. Surprisingly little deformity follows this extensive dissection, but the neck is stiff and there is drooping of the corner of the mouth. (The cervical branch of the facial nerve is severed.) When *bilateral* block dissection is required it must be undertaken, not simultaneously, but consecutively with an interval of about three weeks. Experience has shown that removal of both internal jugular veins is not attended by as much obstructed cerebral circulation as was previously conjectured.

2. **Suprahyoid block dissection** of the neck is indicated in cases of carcinoma of the lower lip, early cases of carcinoma of the tip of the tongue, and carcinoma of the floor of the mouth. Its advantage is that both sides of the neck can be attended to at one operation (fig. 690). Sometimes a unilateral suprahyoid block dissection is indicated combined with a Crile's block dissection of the opposite side.



FIG. 690.—Suprahyoid block dissection of the neck.

During these operations it is advisable to transfuse an amount of blood proportional to the estimated loss. Usually tracheostomy is required only when a monobloc (commando, p. 504) operation is to be performed; in other circumstances tracheostomy should be avoided when possible, because it heightens the incidence of wound infection. Should it be necessary to keep the bronchial tree free from accumulating secretions during the post-operative period, this contra-

indication must be over-ridden.

Post-operative Treatment.—Suction-drainage of the wound for two or three days is best, for it reduces to a minimum the possibility of accumulated blood and blood-clot pressing on the larynx and trachea. Blood transfusion is continued for as long as is deemed necessary, and antibiotic therapy is given as a routine prophylactic measure.

The mortality of cervical-block dissection is, with modern anæsthesia which has done so much to facilitate it, about 3 per cent. The over-all five-year survival rate is 35 per cent.

CHAPTER 24

THE THYROID GLAND AND THE
THYROGLOSSAL TRACT

SURGICAL ANATOMY AND PHYSIOLOGY

Embryology.—The thyroid gland is developed mainly from the median bud of the pharynx (the thyroglossal duct) which passes from the foramen cæcum at the base of the tongue to the isthmus of the thyroid. The isthmus and the major part of the lateral lobes arise from this structure; a lateral bud from the fourth pharyngeal pouch¹ of each side amalgamates with and completes the corresponding lateral lobe.

Anatomy.—The secretory units of the gland are the follicles (alveoli), lined by cubical epithelium and filled with colloid containing stored thyroid hormone. Twenty to forty follicles are bound together with connective tissue to form a lobule, and aggregations of lobules within the capsule form the lobes of the thyroid gland.

Blood Supply.—The thyroid possesses an abundant blood supply and its glandular epithelium is brought into intimate relation with the vascular endothelium owing to the absence of a basement membrane. For the blood-vessels of the thyroid and the nerves in relation to the gland, see fig. 691.

Lymphatics.—From the sub-capsular plexus the lymph passes to the prelaryngeal and pretracheal (Delphic)² lymph nodes, and thence to paratracheal nodes and the inferior deep cervical group.

Physiology.—The thyroid is an iodine trap which accomplishes iodine storage in three distinct steps, each apparently controlled by a specific enzyme: (1) Clearance of inorganic iodine from the blood and trapping within the gland, (2) Oxidation of iodide to free iodine, (3) Iodination of tyrosine within the protein thyroglobulin, and storage within the colloid.

Under the influence of the thyroid-stimulating hormone (TSH) secreted by the

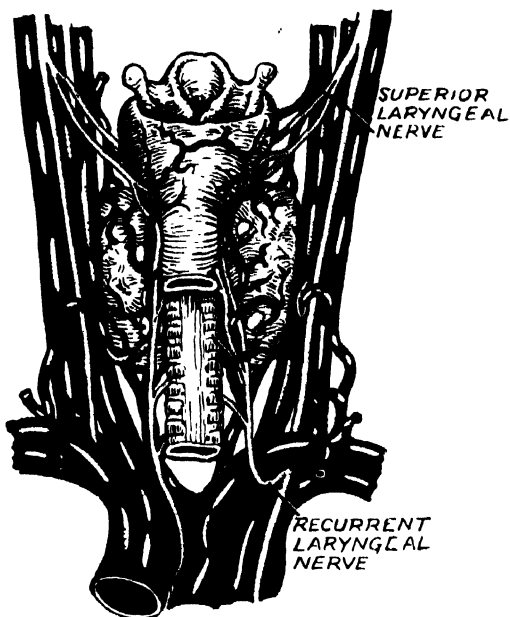


FIG. 691.—The thyroid gland from behind, showing the laryngeal nerves and the four parathyroids. (After Martin Norland.)

¹ Though frequently referred to as a branchial pouch, true branchial arches do not occur in the human foetus.

² Pythia, the snake-woman oracle of Delphi, sat on her tripod, clutching the ribbons of the monolithic 'omphalos' of the world. She inhaled sulphurous fumes and laurel, and uttered a meaningless jargon which was interpreted equivocally by the attendant priests for those who came to consult her. Formerly it was believed that these lymph nodes could have any meaning and were therefore called 'Delphic'.

anterior lobe of the pituitary, thyroglobulin releases thyroxine (T_4)¹ and tri-iodo-thyronine (T_3). The T_4 and the T_3 in the serum are bound in greater and lesser degree to a carrier globulin (thyroid binding globulin TBG), but a little remains free and presumably is the biological active fraction affecting the metabolic activity of the cells of all tissues. T_3 is a quick-acting substance (within a few hours), and T_4 acts slowly (4–14 days); for this reason T_3 used therapeutically is given in extremely small doses.²

The normal amount of iodine secreted by the thyroid as hormone is approximately 70 μ g. per day and within the normal gland there is stored a reserve supply sufficient for approximately two months.

THE PITUITARY-THYROID AXIS

In succeeding pages several references will be made to the inter-relation between the internal secretion of the thyroid gland (thyroxine) and that of the anterior lobe of the pituitary, the thyroid-stimulating hormone (TSH). Production of the latter hormone is inhibited by thyroxine circulating in the blood (fig. 692).

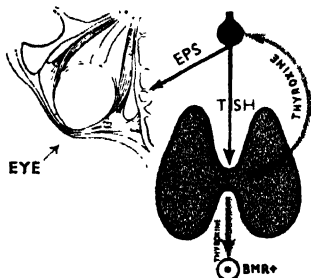


FIG. 692.—Reciprocal arrangement between the anterior lobe of the pituitary and the thyroid in the production of their respective hormones; the so-called pituitary-thyroid axis. TSH = Thyroid-stimulating hormone. EPS = Exophthalmos-producing substance.

Thyrotoxicosis Factitia.³—Sometimes taking too much desiccated thyroid for too long causes an excess of circulating (ingested) thyroxine, and thyrotoxicosis factitia results. In time, complete suppression of TSH ensues. The thyroid gland, bereft of its stimulus, atrophies, and when the ingestion of thyroid extract is stopped, temporary hypothyroidism appears.

Exophthalmos-producing substance (EPS).—Animal experiments in fish and guinea-pigs have shown that the injection of TSH produces exophthalmos, but now a specific hormone is thought to be responsible (fig. 692).

Long-acting thyroid stimulator (LATS) is a recently discovered factor in the aetiology of thyrotoxicosis. It is known to be a globulin, is not demonstrable in normal serum and has a much longer duration of action (8–24 hours) than TSH.

THYROID FUNCTION TESTS

The clinical application of the wide choice of tests available, each one measuring a particular physiological parameter,⁴ requires a knowledge of thyroid physiology. The conclusions drawn from the results do not provide a final diagnosis but can only indicate hyper- or hypofunction in that particular parameter. Every test has false positives and false negatives.

The tests involving direct measurement on patients are the B.M.R. and radioiodine studies. *In vitro* tests on the patients serum are the chemical protein-bound iodine estimation and the resin-uptake (T_3) test.

1. **Basal Metabolic Rate (B.M.R.)** if performed correctly is of value in diagnosing hypothyroidism, or for following the effects of treatment of hyperthyroidism. The apparatus requires frequent servicing and replacement of soda-lime.

2. **Radio-iodine Studies.**—When any radio-isotope is used diagnostically, it is necessary to follow its path—the presence of radio-activity must be demonstrated by a Geiger counter. In other words, negative results are of little value. Secondly, *in vivo* isotope studies measure a rate, so that the unit time is recorded in the result.

¹ Extracted by E. C. Kendall in 1916.

² **Therapeutic Notes:** *L-thyroxine* (T_4) is the official name; trade name Eltroxin; tablet size 0.1 mg. and 0.05 mg. *Tri-iodothyronine* (T_3), official name liothyronine; trade names Cynomel, Tertroxin. Tablet size 20 microgrammes and 5 microgrammes (initial therapeutic dose 5 microgrammes daily, increasing to four times daily).

³ **Factitia**—Latin, *factitius* = to make by art, as opposed to what is natural.

⁴ A parameter is any yard-stick.

When a tracer dose of ^{131}I -iodine is drunk (about $25\mu\text{c}$.,¹ a standard always being kept to allow for natural decay), it is rapidly absorbed from the small bowel into the circulation. At this point the thyroid and kidneys compete for it; if there is thyroid hyperfunction less is excreted in the urine and the counting rate over the thyroid gland will be high. Ultimately the radio-activity will appear in the plasma, being known as the 48-hour protein-bound ^{131}I (this is different from the chemical PBI—see below).

The high uptake of ^{131}I seen in thyrotoxicosis is not suppressed by the administration of liothyronine (given for seven days, $25\mu\text{c}$. q.d.s.), thus differentiating it from chronic thyroiditis in the intermediate phase (p. 564) (Werner's test).

After partial destruction of the thyroid gland, whether due to surgery, excision, previous ^{131}I therapy or thyroiditis, the 48-hour ^{131}I is raised because the remnant is hyperfunctioning to maintain euthyroidism.²

Scanning.—Apart from these timed measurements, scanning of the gland is of value, particularly in cases of solitary nodule. The scan (fig. 693) will show whether

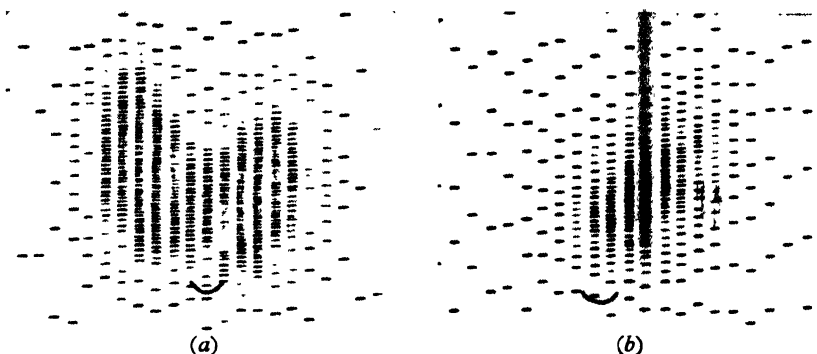


FIG. 693.—(a) Thyroid scan of a normal gland. The position of the sternal notch is indicated. (b) Thyroid scan showing autonomous 'hot' nodule in the left lobe. (Dr. A. W. G. Goolden, London.)

the nodule is functioning, i.e., is 'hot' or is 'cold', the latter being more likely to be malignant. If there is any clinical evidence of metastases, such as pathological fracture, it is of great importance to perform whole-body scanning, because of the possibility of using ^{131}I for treatment if the deposit takes up radio-iodine. In this case it would be justifiable to use a larger tracer dose of ^{131}I of the order of $100\mu\text{c}$.

^{132}I may also be used as a tracer for diagnosis. It is prepared from Tellurium 132. It has a half-life of only 2.3 hours (as opposed to the 8 days of ^{131}I) and can be used on children, for serial tests, and in pregnancy. The radiation dose is one-thirtieth of ^{131}I for the same dose of microcuries.

3. Chemical Protein-Bound Iodine (PBI).—Over 85 per cent. of organic iodine is carried by the serum proteins (the thyroid-binding globulin TBG). The normal range is 3.5–8 microgrammes per cent. Low values are seen in hypothyroidism, in healthy pregnancy, and in acute/subacute thyroiditis. The test is extremely sensitive and is particularly vulnerable to contamination by extraneous iodine.

False Low Results.—Organic mercurial diuretics (e.g. mersalyl) block the catalytic reaction of the estimation. **False High Results.**—Iodine contamination (inorganic and organic) e.g. enterovioform, X-ray contrast media, application of tincture of iodine to the skin, and expectorants containing potassium iodide. The high values obtained in pregnancy and during the taking of oral contraceptives are due to the enhanced protein-binding of T₄.

4. ^{131}I tri-iodothyronine Resin Uptake Test (The T₃ Test).— ^{131}I -T₃ is incubated with the patients serum for forty-five minutes in the cold. An aliquot is

* ^{131}I . Oxygen has an atomic weight of 16. This is the foundation of the atomic scale. The atomic weight of the radio-active isotope of iodine is 131, that of ordinary iodine 127.

¹ Microcurie—a millionth part of a curie (abbreviation μc). Millicurie—a thousandth part of a curie (abbreviation mc).

² Euthyroidism is the state of normal thyroid function.

Pierre Curie, 1869–1906, and Marie Skłodowska Curie, 1867–1934. Co-discoverers of radium. Madame Curie succeeded her husband as Professor of Physics at the Sorbonne, Paris.

Sidney C. Werner, Contemporary. Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons, New York.

Alan Wilfrid Gough Goolden, Contemporary. Consultant Radiotherapist, Royal Postgraduate Hospital, London.

then shaken with a resin having the specific property of absorbing any free $^{131}\text{I}-\text{T}_3$ which has not become bound to the serum protein. After washing, the resin is counted and the result compared with a normal control. The normal range varies from lab to lab and must be established by the use of reference sera. The greater the binding property of a patient's serum proteins, the lower will be the radioactivity (as T_3) absorbed in the resin. Consequently the percentage of $^{131}\text{I}-\text{T}_3$ bound to the resin is high in hyperthyroidism because the binding globulin is already occupied by thyroxine. A similar result occurs in severe liver disease, nephrotic syndrome, advanced malignancy and during treatment with thyroxine, anticoagulants, epanutin, phenylbutazone and anabolic steroids. The resin uptake is low in hypothyroidism, during treatment with oestrogens, and with tri-iodothyronine for hypothyroidism.

The T_3 test does not replace the chemical PBI estimation, but should be used in conjunction with it; for example, in pregnancy, and during the use of oral contraceptives, the chemical PBI is raised the T_3 test is low.

4. **Serum Cholesterol.**—This is a poor parameter of thyroid function, since it does not correlate well with the severity of the disease. It is of some use in following changes during the treatment of hypothyroidism. The serum cholesterol is raised in hypothyroidism, nephrotic syndrome and familial hypercholesterolaemia.

HYPOTHYROIDISM

Clinical Types of Thyroid Failure (Myxœdema and Cretinism)

1. Myxœdema due to thyroid atrophy (fig. 694).
2. Iatrogenic, which may appear up to twenty years after subtotal thyroidectomy or radio-iodine therapy.
3. Late chronic thyroiditis (struma lymphomatosa) (p. 565).
4. Dyshomogenetic goitre.
5. Cretinism, juvenile hypothyroidism.

Presenting Features (fig. 695).—The first step towards making a diagnosis of hypothyroidism is to consider its possibility, but because of the

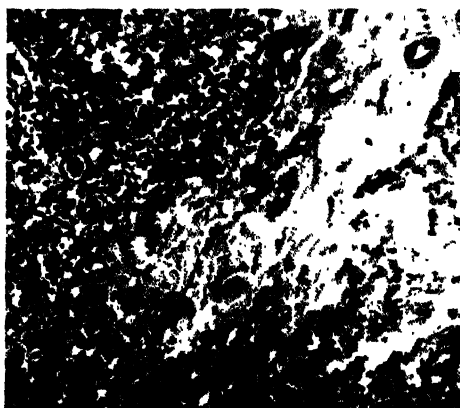


FIG. 694.—Myxœdema. Histology $\times 100$. Gross fibrosis, lymphocytic infiltration and atrophy of acini. Compare with normal histology fig. 700. (Dr. Bernard Fox, London.)

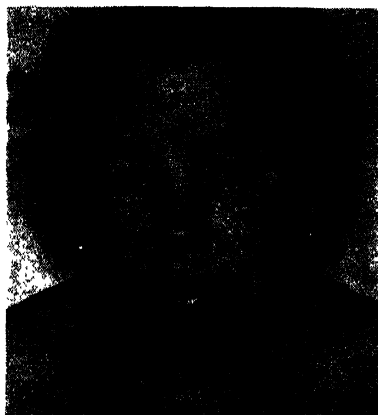


FIG. 695.—Myxœdema: note the bloated look and the dull expression. (Dr. V. K. Summers, Liverpool.)

large number of symptoms associated with various stages of the disease even florid cases are overlooked. (a) The patient with advanced myxœdema is seen to be agreeable, slow-witted, apparently anæmic, and is usually unable to give a history. (b) Hoarseness of the voice, due to infiltration of the vocal

cords; also deafness (of mixed types) and nasal obstruction. (c) Paros-
thesiæ of the fingers caused by carpal tunnel compression (p. 312) associated
with the water retention which is a constant feature of the disease (Hubble).
(d) Cold intolerance as shown by the wearing of extra clothes. (e) Menstrual
disturbances, and infertility, due possibly to secondary hypopituitarism, since
all the tissues are infiltrated with myxœdema. (f) Constipation. (g) Un-
explained trauma with delayed recovery, e.g. a minor head injury without
loss of consciousness, but with prolonged confusion. (h) Cerebellar ataxia,
contributing to accident proneness. (i) In hypothermic coma. (Though
clinically indistinguishable from pituitary coma, the absence of pubic hair is
an important sign). (j) In childhood, a failure to thrive and grow, puberty
being delayed.

Physical Signs.—Amongst the many physical signs are coarsening of the
skin, puffiness round the eyes, slow movements, seen for example when
dressing, and slow relaxation of the ankle jerks are the more important. The
scalp hair and the eye-brows are very thin, and because of dryness the patient
tends to wash her hair infrequently.

Tests of Thyroid Function.—Estimation of the serum PBI (p. 539) is
the simplest confirmatory test. The radio-iodine studies give a low 'T'
index calculated from the urine excretion of the radio-isotope, indicating that
the tracer dose is preferentially excreted by the kidney.

Treatment.—To bring the average myxœdema patient back to health
costs less than twopence a week, and on maintenance, with thyroid extract,
six shillings a year. The initial dose must not be more than 0.05 mg. of
l-thyroxine, and the ultimate dose can only be ascertained by trial. The
treatment must be continued for life and the dose should be reviewed at six-
monthly intervals. If the patient has had a past history of cardiac failure or
ischæmic cardiac disease, it will be advisable to commence treatment with
tri-iodothyronine (liothyronine) 5 microgrammes because its effect is short-
lived. In mild or doubtful cases of hypothyroidism, a therapeutic trial of
l-thyroxine can be a helpful diagnostic procedure.

Myxœdema coma is an emergency and it carries a high mortality. The patient
is hypothermic and the skin is reminiscent of the coldness of the skin of a toad.
The rectal temperature should be monitored with a low reading thermometer. It is
important that the patient is not warmed too quickly, and intravenous liothyronine,¹
intravenous hydrocortisone and antibiotic cover are given.

Pre-tibial myxœdema: Is not a feature of hypothyroidism and is dealt with in
the section on thyrotoxicosis (p. 549).

Dyshomogenetic Goitre.—This is a rare familial condition due to thyroidal
enzyme defects of which there are several types. The patient has usually had at least
one partial thyroidectomy for recurrent goitre whereas the only treatment necessary
is administration of thyroxine, adequate doses of which will melt the goitre. Treat-
ment must be continued throughout life. Mono- and di-iodotyrosines may be found
in considerable quantities in plasma and urine.

Cretinism.—*In the fœtus.*—Evidence of intrauterine athyroidism is found in the
absence of epiphyses (lower end of femur, upper end of tibia), and the ossific centre
of the cuboid, all of which should be apparent radiologically in a nine months' fœtus.

In early infancy.—There are two varieties, endemic (common) and sporadic (rare).

¹ Liothyronine for intravenous use is supplied by Glaxo laboratories 20 Mcg/ampoule.

In endemic areas (p. 554) cretinism with goitre¹ is well known. Elsewhere a cretin usually, but not invariably,^{2,3} has an atrophic gland. The condition may appear in normal families, the other children and the parents presenting no thyroid disorder. Fig. 696 depicts the infant cretin-potbellied, with an umbilical hernia, protruding



FIG. 696.—An infant cretin. (*The late Professor de Quervain, Berne.*)



FIG. 697.—Showing dystrophy of the lumbar spine in a cretin aged ten months. (*Prof. Douglas Hubble, Birmingham.*)



FIG. 698.—A cretin girl aged twenty-two. (*The late J. Thomson, F.R.C.P.*)

tongue and pale puffy face. Radiography may show dystrophy of the twelfth dorsal and the first and second lumbar vertebræ (fig. 697), i.e. where spinal stress is greatest when the child sits up.

In adolescent and adult life (fig. 698), the patient is a dwarf; the skin is dry, redundant and wrinkled, and supraclavicular pads of fat are present. The mentality is below normal.

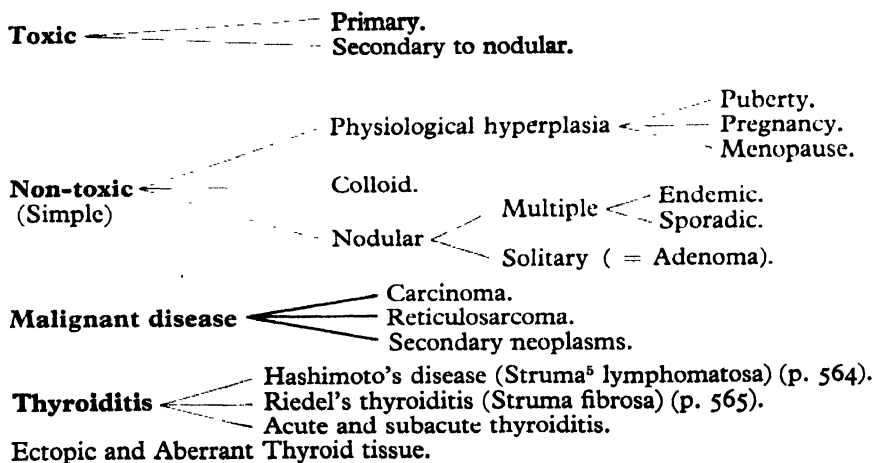
Early treatment on the lines mentioned above, but with the

commencing and maintenance dosage adjusted to the child's age, is essential if fair results are to be obtained, especially with regard to the mental development.

ENLARGEMENTS OF THE THYROID GLAND

A goitre⁴ is an enlarged thyroid gland.

CLASSIFICATION OF GOITRES



¹ In the volcanic regions of Java cretins can be counted by the thousand; animals—domestic and wild—are affected also.

² Cretinism with goitre has been reported in infants of mothers receiving thiouracil compounds during the pregnancy.

³ A number of non-endemic goitrous cretins have been born to a group of itinerant tinkers living in Scotland who intermarry (Hutchison).

⁴ Goitre—Latin, *guttur* = the throat.

⁵ Struma. In the mountains of Bulgaria arises the River Struma that flows into the Ægean Sea. Along its banks, and those of its tributaries, dwell persons of several nationalities among whom endemic goitre has been long prevalent.

HYPERTHYROIDISM (Thyrotoxicosis)

Clinical Types

1. Graves' disease (Primary toxic goitre, Exophthalmic goitre) (fig. 699).
2. Toxic nodular goitre (p. 549).
3. Solitary toxic adenoma—'hot' nodule (p. 555).
4. Acute and subacute thyroiditis (p. 565).
5. Factitia (p. 538).
6. Jod-Basedow phenomenon (p. 550).

PRIMARY THYROTOXICOSIS

Presenting Features.—The diagnosis should spring to mind in any of the following circumstances: (a) excessive sweating, (b) increased appetite and weight loss, (c) preference for cold, (d) arrhythmia or tachycardia especially over the age of fifty, (e) in old people with features of anxiety-depression, (f) in juveniles with a behaviour disorder or growth spurt.

The condition occurs most often in the 3rd and 4th decade of life, being commoner in females and in immigrants. The patient frequently complains of nervousness, excessive sweating, a preference for a cold environment, and an increased appetite in spite of which she is losing weight. Diarrhoea is a late symptom.

On examination, the hands are warm and moist. There may be eye-lid retraction and a lid lag. When the patient is dressing, movements are seen to be clumsy and jerky. The presence of a palpable thyroid gland or cardiac arrhythmia lends support to the diagnosis. The exophthalmos or goitre may be absent but seldom both. The exophthalmos may be unilateral. The thyroid is enlarged uniformly, is smooth and firm, and because of its vascularity emits a thrill and bruit. Myopathy affecting the deltoids and quadriceps are not uncommon when looked for.

Differential Diagnosis—Anxiety State.—Although features of anxiety frequently co-exist, in a classical anxiety state the hands are cold and the radial pulse is likely to be of diminished volume, whereas in thyrotoxicosis the hands are warm and the pulse is bounding. In an anxious patient the pulse rate settles during sleep (if necessary with sedation) but in thyrotoxicosis a sleeping tachycardia persists.

Cardiac arrhythmias are superimposed on the sinus tachycardia as the disease progresses, and they are commoner in older patients with thyrotoxicosis because of the prevalence of coincidental ischaemic heart disease.

Stages in Development of Thyrotoxic Arrhythmias.—1. multiple extra-systoles; 2. paroxysmal atrial tachycardia; 3. paroxysmal atrial fibrillation; 4. persistent atrial fibrillation, non-responsive to digoxin.

The myocardium is exceptionally susceptible to thyrotoxicosis; yet even in fatal cases no definite histological change is discernible in the cardiac muscle. Even slight thyrotoxicosis acting upon a heart the seat of mitral stenosis or other organic lesion, not uncommonly culminates in acute cardiac failure. There is no such thing as 'masked hyperthyroidism', but often so much attention is focused on the heart that a seemingly minor lesion of the thyroid is overlooked. The hyperthyroidism is not masked, but missed (Rundle).

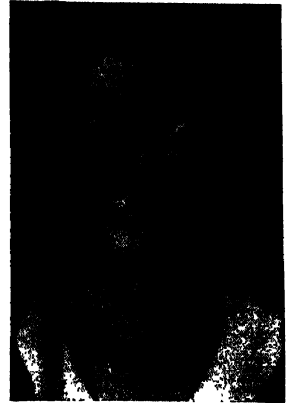


FIG. 699.—Primary toxic goitre. (Professor E. J. Wayne, Glasgow.)

Histology.—The normal thyroid gland (fig. 700) consists of acini lined by flattened cuboidal epithelium and filled with homogenous colloid. In hyperthyroidism (fig. 701) there is hyperplasia of acini which are lined by high columnar epithelium. Many of them are empty, and others contain vacuolated colloid.

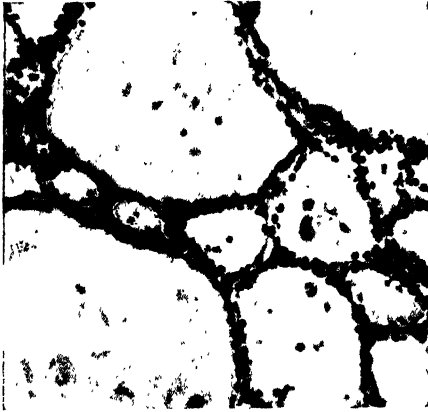


FIG. 700.—Normal thyroid (see text).
× 100.

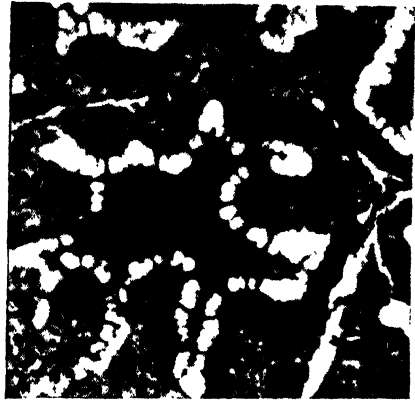


FIG. 701.—Thyrotoxicosis (see text).
× 100. (Dr. Bernard Fox, London.)

TREATMENT OF THYROTOXICOSIS

Three alternative forms of treatment are available—(1) medical, (2) surgical resection after medical preparation, and (3) radio-iodine in therapeutic dose. The ultimate choice depends on a balance of factors not the least of which may be the patient's prejudices, but it is good practice to defer the decision regarding definitive treatment until the patient has been rendered euthyroid by thyroid-blocking drugs.

1. Medical Treatment

A patient with severe thyrotoxicosis should be confined to bed, effectively sedated, and encouraged to eat and drink as liberally as possible—the higher the calorie intake the more successful the treatment (high-calorie foods include ice-cream and dairy produce). At the same time treatment should be commenced with either of the two thyroid-blocking drugs in current use, propylthiouracil^{1,2} or carbimazole.³ It is important that either should be prescribed on a 6–8 hourly basis. A latent period of several days passes before there is clinical evidence of improvement, and any previous treatment with inorganic iodine further delays the response.

During treatment with thyroid-blocking drugs an initial enlargement of the gland is to be expected. Treatment should be maintained for at least

¹ In 1943 Astwood first treated thyrotoxicosis successfully with thiourea.

² *Therapeutic Notes: Propylthiouracil*—50 mg. tablets, usual dose 100 mg. 6-hourly—maintenance dose 50 mg. 8-hourly. Side effects uncommon (less than 3 per cent.), the commonest being a papular rash which may be purpuric, and which usually subsides spontaneously without the drug being discontinued; pain and stiffness in joints; drug fever is very rare.

³ *Carbimazole*—proprietary name Neomercazole, tablets of 5 and 10 mg. Initial dose 10 mg. 6-hourly. The patient should be instructed to discontinue either treatment if she develops a sore throat until the white cell count has been checked (see agranulocytosis).

two years after which time it is hoped a remission has occurred. Nevertheless, about 50 per cent. of cases relapse after stopping treatment, but it has recently been claimed that an iodine-free diet throughout treatment and afterwards reduce the relapse rate considerably (Crooks). This, in practice, means avoiding iodised table salt and fish.

A large goitre will enlarge further on drug treatment and will obviously weigh the decision in favour of operation.

Complications of Treatment with thiouracils.

(a) **Thiouracil Goitre.**—High dosage, if prolonged, will produce a hyperplastic gland—a thiouracil goitre. Ten to twenty-one days of pre-operative preparation with potassium iodide (p. 550), without thiotherapy, reduces considerably this vascularity which, however, is sometimes still formidable.

(b) **Transmitted Thiouracil Goitre.**—Thiotherapy may only be given during pregnancy provided the mother remains euthyroid and the treatment is stopped one month before term. Thiouracil medication causes a thyroxine deficiency that stimulates the pituitary to excessive thyrotropic hormone secretion. The transference of this hormone by the placenta to the fetal thyroid sometimes causes formation of a thiouracil goitre with accompanying hypothyroidism in the foetus and the infant (fig. 702).

(c) **Compression of the Trachea.**—Thiotherapy causes the thyroid gland to become larger and softer, therefore this form of treatment should be avoided in cases of toxic goitre that can be demonstrated radiologically to be retrosternal or otherwise causing compression of the trachea. Its use in such cases can bring about a dangerous degree of respiratory obstruction.

(d) **Agranulocytosis** is a condition in which there is an absence of, or a great diminution in, the granular leucocytes. It may occur with catastrophic rapidity at any time during the treatment, irrespective of the dosage. The incidence of agranulocytosis with either propylthiouracil or carbimazole is less than 1 in 500. Any patient undergoing drug treatment who develops a sore throat, a pyrexia, a maculopapular skin rash, or one of the rarer manifestations (conjunctivitis, enlargement of the salivary glands or the lymph nodes, arthralgia or haematuria) should be investigated without delay. In severe cases blood transfusion is necessary. The penicillins are given as a prophylaxis against infective complications, but sulpha drugs and chloramphenicol are banned, as they induce agranulocytosis. In spite of treatment the condition proves fatal in about a quarter of the patients affected.



FIG. 702.—Transmitted thiouracil goitre.

2. Surgical Treatment

The indications for thyroid surgery are reviewed on p. 550, followed by an account of the necessary preoperative preparation, and the operation of subtotal thyroidectomy.

3. Radio-active Iodine (^{131}I) Treatment¹

The time during which 50 per cent. of the radio-activity will be emitted is known as the half-life of the isotope. ^{131}I has a half-life of eight days. Whereas its gamma-rays, which are highly penetrating, are used for detection of thyroid tissue, its beta-rays are highly destructive to tissues, and particularly to thyroid tissue, because the gland entraps the circulating radio-iodine.

Indications

- (i) Patients with primary thyrotoxicosis over forty-five years of age.
- (ii) Those in whom intercurrent disease reduces the expectancy of life.

¹ Radio-active iodine was first used in the treatment of thyrotoxicosis by Hertz and Roberts in 1942. Isotope—Greek, *isos* = equal + *topos* = place. E.g. iodine and radio-active iodine are chemically identical.

James Crooks, *Contemporary*. Reader in Materia Medica and Therapeutics, University of Aberdeen.
Saul Hertz, *Contemporary*. Director of Radioactive Isotope Research Institute, Boston, Mass., U.S.A.
Arthur Roberts, *Contemporary*. Associate Professor of Physics, University of Iowa, U.S.A.

(iii) Those refractory to antithyroid drugs, and those who refuse operation.
 (iv) Recurrence after operation, particularly after a second operation, when distorted anatomy increases the risk of damage to the recurrent laryngeal nerves.

(v) Primary thyrotoxicosis associated with uncompensated cardiac failure.

The reasons for not treating patients under forty-five years of age by radio-iodine are (a) the possibility of detrimental genetic defects in future generations; (b) the possibility that radio-iodine may induce carcinoma. Experts agree that were carcinoma to develop as a result of radio-iodine therapy, it would do so after twenty years. Consequently reports that it has not done so already are not conclusive. (c) In a patient who is pregnant, radio-iodine passes through the placental barrier to reach the foetus, whose thyroid is especially vulnerable to this isotope. Radio-iodine is also secreted in the milk. Therefore radio-iodine is contraindicated throughout pregnancy and during lactation, if the infant is to be breast-fed.

When radio-iodine treatment is used, the maximum effect is delayed for up to 8 weeks after the dose. The disease may initially be controlled with Carbimazole which is stopped for four days prior to the therapy dose and then recommenced after it.

Administration.—It is now agreed that special precautions against irradiation exposure are unnecessary with the doses used in treating primary thyrotoxicosis. Most patients can remain completely ambulatory and carry on their usual activities: patients who live within reasonable distance of the hospital can be treated as out-patients. The prescription of an entirely accurate radiation dose being impracticable, there are many variations in the manner of administration in various centres. That used at the Royal Infirmary, Edinburgh, will be described. The patient drinks a small amount of water containing the dose requested from the central laboratory. This is sent in a screw-capped bottle containing between 1 and 2 ml., the dose requested being:

4 millicuries of radio-iodine if the thyroid is impalpable.

5 or 6 millicuries of radio-iodine if the thyroid enlargement is insignificant.

This dose is increased in accordance with the estimated size¹ of the gland, but 12 millicuries is seldom exceeded. The most toxic patients are given a course of an antithyroid drug before the ¹³¹I. It should be noted that if an antithyroid drug is given, a relatively larger dose of ¹³¹I is necessary. It may be justifiable to continue antithyroid drugs after ¹³¹I therapy, except in very toxic patients as a preliminary to further isotope treatment. Should there be no clinical improvement within twelve weeks after the administration, a further dose is given (Macgregor).

When compared with operation, the results of ¹³¹I treatment are slow: it is fully three months before substantial improvement is registered.

Untoward reactions are surprisingly few. Thyroid crises following the treatment have been reported, but they are rare. However, a mild flare-up of the disease is frequent. Muscular and joint rheumatic pain lasting months, or longer, is the chief complaint (Wayne). Hair sometimes falls out, but it grows again. The incidence of aggravation of exophthalmos is possibly lower than that following operation.

¹ The weight of the thyroid gland during the prime of life is between 20 and 60 gm.

Alastair G. Macgregor, *Contemporary*. Regius Professor of Materia Medica and Therapeutics, University of Aberdeen.
 Edward Johnson Wayne, *Contemporary*. Regius Professor of Practice of Medicine, University of Glasgow.

The chief serious sequel of ^{131}I therapy is hypothyroidism. Even in specialised centres this may occur in 30 per cent. or more of patients. The symptoms can of course be adequately controlled with thyroxine, but hypothyroidism must be suspected in any patient who has had radio-iodine therapy.

Hyperthyroidism in Children.—This is rare and results in accelerated growth and a behaviour disorder to the point of hypomania; in most cases the child is restless and is unable to concentrate at school. Medical treatment with careful supervision of dosage is needed.

THE EXOPHTHALMOS OF PRIMARY THYROTOXICOSIS

For purposes of treatment it is convenient to divide the exophthalmos of primary thyrotoxicosis into three categories—mild, severe, and progressive. The production of exophthalmos by EPS is mentioned on p. 538.

Mild is the most frequent of the varying degrees of exophthalmos that accompany primary thyrotoxicosis. There is hardly any true proptosis, minor degrees of which are best observed from above, viz : —————→

The staring look is due mainly to exposure of the whites of the eyes by retraction of the upper lid. Lid-lag (von Graefe's sign), as tested by asking the patient to follow the examiner's finger moved up and down, is present.



Severe.—There are three components : (a) congestive features are the earliest and most easily recognised. Watering of the eyes is in evidence, especially in the morning, and is often mistaken for conjunctivitis.



(a)



(b)

FIG. 703.—(a) Prior to sub-total thyroidectomy following preparation with carbimazole and potassium iodide; note lid retraction, which is more marked on the right, no exophthalmos. (b) Relapse of thyrotoxicosis. Weight loss; a bilateral partial tarsorrhaphy has been performed because of early exposure keratitis. The thyroidectomy scar is just visible. (Dr. Bruce Fowler, London.)

(b) Protrusion of an eyeball and its lid is frequently more obvious on one side than on the other (fig. 703).

(c) Paresis of one or more of the extrinsic muscles of the eyeball due to oedema and round-celled infiltration of the muscles is at times revealed by

diplopia. The superior rectus muscle is most commonly affected. When more than one muscle is involved the condition is known as *ophthalmoplegia*.

Progressive occurs comparatively infrequently, and may follow ablation of thyroid tissue for primary thyrotoxicosis, whether accomplished by operation, antithyroid drugs, or ^{131}I . Visual acuity deteriorates progressively and the acuity should be assessed daily; visual failure is due to corneal ulceration (exposure keratitis), excessive protrusion of the eyeballs, or pressure on the optic nerve. Early corneal ulceration may be demonstrated by the use of fluorescein as an intra-vital stain.



FIG. 704.—Progressive exophthalmos, with chemosis and exophthalmic ophthalmoplegia. (Clinical Collection, the Lahey Clinic, Boston, Mass. U.S.A.)

Often chemosis is pronounced (fig. 704). Unless arrested, the condition leads to corneal ulceration, papilloedema, dislocation of the eyeballs, and ultimately to panophthalmitis.

Treatment.—Hypothyroidism, if present, must be corrected, care being taken to avoid over-treatment; tablets of thyroid, 3 to 6 grains

(180–360 mg.) daily, or, preferably, large doses of triiodothyronine, should be prescribed and continued for three months after the danger of progressive exophthalmos appears to have passed. Other simple measures can also help—sleeping with the head elevated by several pillows to aid drainage, and a dehydrating regimen, e.g. a low salt diet and a diuretic. When diplopia is the main disability, spectacles with one lens frosted are often helpful, provided the earpieces have a universal joint, so that the lens can be transposed (Fraser).

The intravenous administration of corticotrophin (ACTH) or prednisone (20 mg. six-hourly) cures some patients, especially if congestive features predominate. Intraorbital injections also may be effective. However, in at least 50 per cent. of cases these measures are ineffective.

Operative Treatment :

Lateral Tarsorrhaphy (suturing together the eyelids) is of great value. It protects the cornea, restrains the exophthalmos, and gains time in which to try to bring the exophthalmos under control by conservative measures without invoking the risk of irreparable damage (fig. 703).

Orbital decompression is a method of preserving the eyes when the above measures have failed to arrest progressive exophthalmos.

Rowbotham's Operation.—The incision commences half an inch (1.3 cm.) posterior to the outer margin of the bony orbit, and extends upwards to the lateral end of the eyebrow, and then along the margin of the origin of the temporal muscle for 3 inches (7.5 cm.). After the scalp flap has been reflected, the temporal muscle and its fascia are dissected downwards. With a perforator, and then with a burr, an opening is made through the orbital plates of the sphenoid and the zygomatic bone, thus exposing the peri-orbital fascia (fig. 705). The opening in the



FIG. 705.—Orbital decompression completed. (After G. F. Rowbotham.)

bone is enlarged with nibbling forceps so that the lateral wall and the roof of the orbit are removed, the dura of both the anterior and the middle cranial fossæ being exposed. In severe cases the outer margin of the orbit also is sacrificed. The peri-orbital fascia is incised, and the orbital contents bulge forth. The wound is closed with drainage for twenty-four hours. The original operation for decompression of the orbit was devised by Naffziger, who employed the frontal route to remove the roof of the orbit.

Section of the pituitary stalk is the most recent method of treating progressive exophthalmos which does not respond to conservative measures, and in the few cases in which it has been carried out, it has proved remarkably successful (McCullagh).

Pretibial myxœdema, affects both sexes and takes the form of œdema of the legs, occurring at any stage in the course of *hyperthyroidism*, usually after thyroidectomy, ^{131}I therapy, or prolonged thiotherapy. It is associated particularly with pronounced and progressive exophthalmos. The earliest manifestation is pitting œdema with orange-coloured pigmentation. Symmetrical in distribution, it involves the skin of the legs above the area constricted by the shoes to within a few inches of the knees. As time passes the colour changes to red, and then deep purple, closely resembling venous thrombosis. By this time the œdema is the solid type.

Treatment.—It is not relieved by thyroid administration but given time it usually disappears spontaneously. If the swelling in the legs is considerable, pressure bandaging is indicated.

Thyrotoxic Crisis (Storm)

Since the advent of effective pre-operative preparation (p. 550), a thyrotoxic crisis occurring post-operatively is now extremely rare, but it may occur in a patient who has been unable to tolerate thyroid-blocking drugs because of granulocytopenia (p. 545). Very rarely the patient may present in a thyrotoxic crisis when first seen. The clinical features are dramatic, and include sustained pyrexia, a tachycardia of over 120, sweating and diarrhœa leading to severe dehydration, and a toxic confusional state.

Treatment.—The patient must be nursed in a cooled quiet single room and effectively sedated. Blood should be taken for full blood count, packed cell volume (p. 68) and serum electrolytes, and if the patient is unable to drink copiously, an intravenous dextrose infusion is set up. Iodine therapy may be given intravenously using sodium iodide solution, together with antibiotic cover and intravenous hydrocortisone. There is good evidence that Propanalol, a beta-adrenergic blocking agent is valuable in the early stages (dose 20 mg. 6-hourly orally). Ultimately, the patient will escape from the temporary control by iodide, and carbimazole should be added in full doses.

SECONDARY THYROTOXICOSIS

Thyrotoxicosis may be secondary to some form of pre-existing simple goitre. It is encountered most frequently in women over forty years of age who may have had a solitary adenoma of the thyroid (p. 555)—often since their 'teens'. Secondary thyrotoxicosis is also prone to occur in retrosternal goitres. Although the initial symptoms are less severe than those of primary toxic goitre, the condition is steadily progressive, and remissions are absent. Eventually cardiovascular symptoms (p. 543) become very much in evidence, and many of these patients have ischæmic heart disease. Exophthalmos is nearly always absent. That an adenoma of the thyroid is hyperfunctioning often can be proved by a tracer dose of radio-iodine ('hot nodule') (fig. 693).

Solitary Toxic Adenoma.—Long over-production of thyroxine by an adenoma causes the anterior pituitary virtually to cease production of TSH (p. 538): the normal thyroid tissue ceases to function and eventually it will atrophy (fig. 706). Consequently, while it is essential to remove the adenoma intact within its capsule, without



FIG. 706.—Hyperfunctioning solitary adenoma with atrophic uninvolved gland. (After O. Cope et al.)

squeezing it, it is contraindicated to excise the contralateral lobe. Atraumatic removal of the adenoma is best achieved by unilateral subtotal lobectomy.

Toxic Multinodular Goitre.—Thyrotoxicosis is an infrequent, late complication. Radio-active iodine studies indicate that it is the internodular parenchyma rather than the nodes (which are almost bereft of function) that gives rise to thyrotoxicosis. Subtotal thyroidectomy is the only regularly curative treatment.

Unless the patient is an exceptionally poor surgical risk, a toxic nodular goitre (solitary or multinodular) is not treated with radio-iodine because :

1. The recurrence rate is higher than in cases of diffuse toxic goitre.
2. Larger doses are required for a remission—often twice that necessary for a diffuse toxic goitre.
3. A small percentage (1 per cent.) are or will become malignant.

Jod-Basedow Thyrotoxicosis.—The prophylactic use of iodine is said to increase the incidence of secondary thyrotoxicosis occurring in cases of endemic goitre (p. 553). Secondary thyrotoxicosis arising in this way is known as Jod-Basedow.¹

INDICATIONS FOR THYROID SURGERY

These may be absolute or relative, and tend to shade from one to the other:

- | | |
|-----------------|--|
| Absolute | <ul style="list-style-type: none"> — ‘cold’ solitary nodule (p. 555) (15 per cent. are malignant). — large goitre with compression symptoms (p. 555). — large toxic nodular goitre. — ‘hot’ nodule (p. 549) causing thyrotoxicosis. |
| Relative | <ul style="list-style-type: none"> — failed medical treatment (due to relapse or severe drug reaction). — a small diffuse toxic gland if the patient states a preference for surgery. — cosmetic. — carcinoma (see text, p. 559). — thyroiditis, if diagnosis is uncertain etc. (see text, p. 564). |

Preoperative Preparation.—Thyrotoxic patients require careful preparation for about fourteen days. If the patient has been rendered euthyroid with medical treatment (p. 544), the drugs are discontinued and a pre-operative course of potassium iodide² is given for ten to fourteen days, to reduce the vascularity of the gland. Iodide controls thyrotoxicosis as effectively as thiouracil, but after fourteen days its beneficial effects diminish. It is also to be given in mild cases who have not received medical treatment. The effect of this preoperative preparation on thyroid histology is seen in the accompanying microphotograph (fig. 707).

Teeth.—This is a convenient place to emphasise that should the patient

¹ Iod = German for iodine + Basedow.

² Potassium Iodide tablets N.F. 60 mg. tds. have superseded Lugol's Iodide solution. Circa 1600 B.C. the Chinese used burnt sponge and seaweed for the treatment of thyrotoxicosis.

*Carl Adolphe von Basedow, 1799–1854. General Practitioner, Mersburg, Germany.
Jean Guillaume Auguste Lugol, 1786–1851. Physician, Hospital Saint-Louis, Paris. Henry S. Plummer, 1874–1936, Physician, Mayo Clinic, Rochester, U.S.A., was the first to use Lugol's solution in the preparation of thyrotoxic patients for operation.*

possess enlarged or infected tonsils or carious teeth, on no account must tonsillectomy or dental extraction be undertaken until the thyrotoxicosis has been fully controlled. Fatal thyroid crises (p. 549) may be precipitated by a minor operation upon a patient who is still thyrotoxic.

Indirect Laryngoscopy.—The vocal cords must always be inspected before operation, as in about 4 per cent. of patients symptomless paresis or paralysis is present, possibly due to toxic neuritis during exanthemas in childhood. The presence of an impaired vocal cord *must*, for the surgeon's protection, be recorded categorically in the notes before the operation.

Subtotal Thyroidectomy.—General anaesthesia is administered by an endotracheal tube. Local anaesthesia is sometimes employed in poor-risk and elderly patients.

Technique.—(a) *Incision and exposure of the gland.* An almost transverse incision is made following a crease of the neck. The upper flap of skin and platysma is reflected up to the level of the pomum Adami. The lower flap is reflected down to the suprasternal notch. A vertical incision is made through the musculo-fascial layers in order to expose the correct plane of the surface of the gland. By suitable retraction of these layers it is possible to expose the lateral lobes, but if there is any difficulty it is always advisable to divide the pretracheal muscles as high as convenient (the nerve supply enters the muscles inferiorly).



FIG. 708.—Ligation of the superior thyroid vessels.

(b) *Vascular Isolation.*—The middle thyroid vein and any accessory middle veins of Kocher are carefully dissected and then divided between ligatures, allowing the lateral lobe to be dislocated forwards: this step permits identification of the inferior thyroid vein, which is dealt with similarly. Next the superior pole is freed and the superior thyroid vessels are ligated (fig. 708). Injury to the superior laryngeal nerve (fig. 691) is best avoided by dissecting the medial aspect of the superior thyroid artery and the upper pole of the thyroid from below upwards (injury causes loss of strength—timbre—of the voice). In most cases the inferior thyroid artery is ligated in continuity, but care must be taken to place the ligature around the artery only and laterally well away from the gland, to avoid the recurrent laryngeal nerve.

(c) *Identification and preservation of the recurrent laryngeal nerve* (figs. 691 and 709). Most operators ascertain the position of the nerve as it lies between the trachea and the oesophagus. It can be felt with the finger, and then it is exposed carefully along its course to where it disappears behind the gland. Complete dissection of the nerve behind the gland is not usually carried out, because as shown in fig. 709 the posterior portions of the lateral lobes are preserved. Furthermore, extensive dissection may cause troublesome oozing and so increase the likelihood of nerve damage, and also interference of the blood supply to the parathyroids (fig. 691). In some instances of retrosternal prolongation of a part of the thyroid, special care must be taken to ascer-

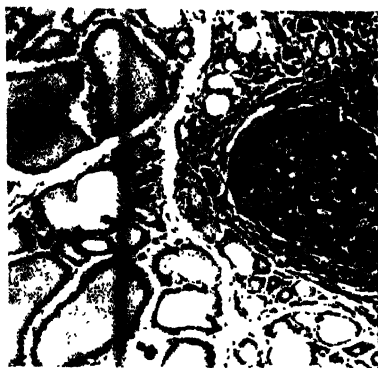


FIG. 707.—'Prepared' thyrotoxicosis. The acini are lined by low cuboidal epithelium and contain more colloid. Compare with figs. 700 and 701. Note the lymphocytic infiltration with lymph follicle formation, which is a feature of the thyrotoxic gland. $\times 100$. (Dr. Bernard Fox, London.)

tain that the nerve has not been displaced into an abnormal position, e.g. overlying the prolongation. Injury to one recurrent laryngeal nerve is sometimes recognised at operation (a) by stridor; (b) by a change in the patient's voice, if the operation is being performed under local anæsthesia. Injury to both nerves may bring about sudden laryngeal obstruction, necessitating immediate tracheostomy; at other times the injury is not recognised until the early post-operative period (see below).¹

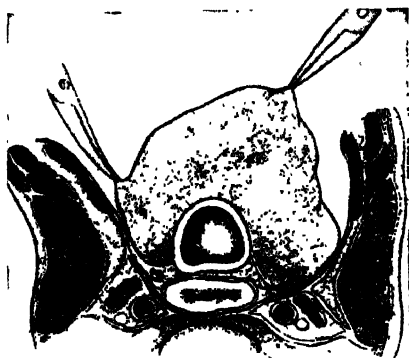


FIG. 709.—Transverse section showing the relative amount of the lateral lobes removed in toxic goitre, and the portion that remains protecting the parathyroid glands and the recurrent laryngeal nerves.

receive the fluid intravenously. If the necessary amount of fluid cannot be administered by mouth, via a transnasal intragastric tube, or per rectum, an intravenous drip is indicated.

Post-operative Complications:

Hæmorrhage.—The nurse should be instructed to watch for excessive hæmorrhage, particularly at the back of the dressing, for the blood tends to trickle posteriorly. Reactionary venous hæmorrhage is the usual type, particularly if the patient coughs or vomits. Hæmorrhage may be concealed owing to clotting. Any undue bulging of the neck must be reported and dealt with immediately.

Deep arterial or venous reactionary hæmorrhage (fig. 710) comes on without warning, usually within six hours of operation. The patient holds her head in a peculiar fixed position, and finds it impossible to move her head without exaggerating the symptoms of suffocation. *So sudden and severe is this type of respiratory obstruction that more often than not it is impossible to get a doctor in time. Consequently the nurse must be taught how to open the wound, cut the muscle sutures, separate the muscles, and remove the clot—in this desperately urgent situation it is justifiable to carry out this measure with unsterilised hands.* At the same time the resuscitation 'alert' will be put into action as for cardiac arrest. If bleeding continues, the patient must be taken to the operating theatre and the bleeding vessel ligated.

Dyspnœa.—Urgent dyspnœa can result from blood-clot pressing upon the trachea or from bilateral damage to the recurrent laryngeal nerves. If reopening the wound fails to relieve the dyspnœa, tracheostomy must be performed forthwith.

Alteration in the Voice.—After an extensive thyroidectomy, nearly all patients speak in a whisper for a few days, to spare undue movement in the region of the wound. If hoarseness or aphonia persist, it is possible that there has been damage to one or both recurrent laryngeal nerves.

Recurrent Laryngeal Nerve Damage (p. 637).



FIG. 710.—Tension hæmatoma deep to the pre-tracheal muscles. (After V. H. Riddell, F.R.C.S., London.)

¹ It is essential that pre- and post-operative laryngoscopy be carried out as a routine.

Tracheitis frequently follows thyroidectomy. It is relieved by medicated steam inhalations.

Parathyroid Tetany (p. 568).

Thyrototoxic Crisis (thyroid storm) (p. 549).

NON-TOXIC GOITRE

PHYSIOLOGICAL HYPERPLASIA¹ (*syn.* GOITRE OF PUBERTY)

Clinical Features.—The only symptom is a swelling in the neck (fig. 711). The condition is almost confined to females. The thyroid gland is enlarged evenly and feels comparatively soft. Sometimes the deformity is considerable. Usually the enlargement subsides gradually, and has all but disappeared by the twentieth to twenty-second years. However, any enlargement that does not subside completely in the intermenstrual phase must be considered as abnormal, and as constituting a potential colloid goitre.

Treatment.—Satisfactory results are obtained by prescribing L-thyroxine (0.3 mg. daily) which should be maintained for at least six months. The use of iodine preparations is discouraged, for not a few examples of thyrotoxicosis have been produced by their exhibition in these cases. (See Jod-Basedow, p. 550.)



FIG. 711.—Physiological hyperplasia of the thyroid gland (goitre of puberty).

COLLOID GOITRE

The patient usually presents between the ages of twenty and thirty years, i.e. after physiological

hyperplasia should have subsided. The whole of the thyroid gland is affected, and, as a rule, by the time the patient seeks advice the deformity is most obvious (fig. 712). To the palpating fingers the swelling feels elastic and tolerably smooth. Symptoms of pressure upon the trachea are infrequent. Occasionally this goitre becomes toxic.

Microscopically, the thyroid vesicles are found to be greatly distended with colloid and lined by flattened epithelium.



FIG. 712.—Colloid goitre.

Treatment.—Thyroid extract causes a decreased avidity of the gland for iodine, and suitable doses of this preparation combined with small doses of sodium

iodide give better results than either alone (Werner). If there is not a dramatic decrease in the size of the thyroid, operation (p. 551) is required.

MULTINODULAR GOITRE (*syn.* Adenoparenchymatous Goitre < Endemic (commonest) Sporadic)

Endemic goitre.—Iodine deficiency.—The hypothesis that iodine deficiency is a major factor in the production of endemic goitre is firmly established. In nearly all districts where endemic goitre is prevalent it has been proved that there is a very low iodine content in the water and food.

¹ Some physiological hyperplasia also occurs during pregnancy and at the menopause.

Geographical Distribution

(a) *Mountains and Limestone Country*.—Endemic goitre abounds on the slopes and in the valleys of mountain ranges, such as the Alps, the Rocky Mountains, the Upper Mississippi Valley, the Andes and the Himalayas. There is also a relationship between goitre and chalk or limestone country. For example, in England the goitre belt (fig. 713) includes the Mendip hills of Somerset, the Chilterns, the Cotswolds, and the Pennine chain of Derbyshire and Yorkshire. (The endemic goitre of Derbyshire is known as the Derbyshire neck.) In Ireland, the limestone county of Tipperary is another example.



FIG. 713.—The goitre belt of England and Wales.

(b) Yet in spite of the emphasis that has been placed on mountainous districts, it should be realised that some goitre regions are little, if at all, above sea level; the reason why these lowlands are goitrous is that they depend upon faraway mountain ranges for their water supply, e.g. Holland, the Plain of Lombardy, Egypt, the Congo, and the Great Plains east of the Southern Alps of New Zealand.¹

(c) *Fluoride excess* in drinking water has also been deemed to be a major cause in the production of goitre in the north-west of Cape Province, South Africa.

Water Pollution.—Water contaminated with human or animal excreta can cause enlargement of the thyroid (McCarrison).

Goitrogens in Foodstuffs².—Members of the Brassica family (cabbages, kale and rape) contain substances such as thiocyanates and thio-oxazolidone in their leaves and seeds, which may cause goitres in sheep and cattle. The amounts normally ingested by humans are unlikely to have any deleterious effect.

Prophylaxis.—By supplying table salt containing one part of potassium iodide to 10,000 parts of sodium chloride the incidence of goitre has been strikingly reduced in Switzerland, Detroit, Sweden, and New Zealand. In the State of Michigan and in County Tipperary, small doses of sodium iodide are given to schoolchildren during the winter months, with the same beneficial effect, whereas in Holland notable results have accrued from adding an iodising apparatus to the municipal water supplies in affected districts.

The incidence of goitre in Great Britain has diminished considerably in recent years probably due to the improved distribution of fish, with its high iodine content, and also to better water supplies.

Pathology.—Nodular goitre passes through a stage of diffuse epithelial hyperplasia, followed by involution and the formation of a colloid goitre. As a rule recurring cycles of hyperplasia and involution continue, and the unequal response by different portions of the gland results in gross nodularity. The nodules, although circumscribed by a delicate capsule, are difficult or impossible to enucleate. By the time the gland comes to be examined, most of the nodules are cysts filled with brown, green, or black watery fluid, or similarly coloured jelly-like material (fig. 715). Cholesterol crystals are generally present in large numbers in the contents of cysts. In other cases of some standing the stroma shows an overgrowth of fibrous tissue, and as time passes irregular areas of calcification are wont to occur in it.

Clinical Features.—Let us consider a district where endemic goitre is of average severity. By the age of six, about 20 per cent. of the boys and 30 per

¹ Paradoxically, the soil of New Zealand is heavily impregnated with iodine, but this does not prevent goitre in man, sheep, and other animals (Sir Charles Hercus).

² Concerning proprietary drugs, Felsol powders, used for asthma, contain iodopyrine compound which may cause goitre by inhibiting the organic binding of iodine by the thyroid.

Sir Robert McCarrison, 1878-1960. Major-General Indian Medical Service (Rtd.), Consulting Physician, Radcliffe Infirmary, Oxford.

Sir Charles Ernest Hercus, Contemporary. Professor of Public Health, University of Otago, New Zealand.

cent. of the girls present a visible and palpable smooth, soft, and symmetrical enlargement of the thyroid gland. As the children become older, the number with goitre increases; so does the average size of the gland. After the age of puberty the total number presenting goitres declines, for in some of the boys the thyroid enlargement disappears. Among the girls the number and the size of the goitres continue to increase to the age of eighteen. After that time only exceptionally does an enlarged gland decrease in size to become unnoticeable. Sometimes it remains stationary; often it continues to enlarge, and becomes multinodular (fig. 714).

Multinodular goitre is encountered most frequently in patients over thirty years of age. The whole gland is studded with rounded swellings of varying size. The unsightly swelling, when it has reached a distressing size, is the most common symptom that brings the patient to seek relief.

Complications :

Pressure upon the trachea (fig. 715) may develop. When the goitre is mainly unilateral, the degree of tracheal displacement is sometimes fantastic. Nevertheless, it is not this type of deformity that produces dangerous dyspnoea; it is rather the bilateral, deep, but not obviously great, enlargement. Here the continuous compression of the sides of the trachea decreases its transverse diameter. If the goitre is retrosternal, it may cause superior mediastinal block (p. 558).

Secondary Thyrotoxicosis.—Jod-Basedow (p. 550).

Carcinoma.—Multinodular goitre of long standing, whether sporadic or endemic, is the seat of a malignant change in about 8 per cent. of cases.

Treatment.—The only effective treatment of multinodular goitre is by operation, and partial thyroidectomy (fig. 716) should be recommended not only to rid the patient of the deformity, but to prevent the complications considered above.

SOLITARY NODULAR GOITRE

(syn. ADENOMA OF THE THYROID < Solid Cystic)

Solitary adenoma is rare in endemic districts, but common elsewhere.

Pathology.—This discrete swelling has a



FIG. 714.—Multinodular goitre.

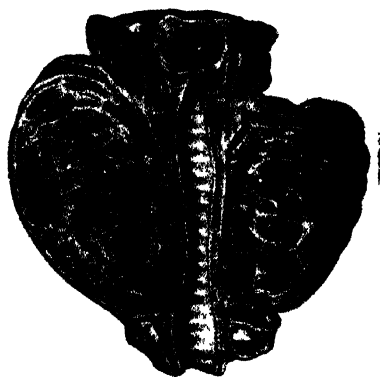


FIG. 715.—Scabbard trachea caused by a multinodular goitre.



FIG. 716.—Showing the approximate amount of the thyroid gland that is removed in nodular goitre without thyrotoxicosis. Shaded portion remains.

complete capsule composed of fibrous tissue, which in old-standing cases sometimes shows calcareous or, very rarely, osseous changes. The colour of the interior is often variegated from pale yellow to deep purple. Apart from the capsule, the chief microscopical difference between a solid adenoma of the thyroid and the gland proper is the presence in the latter of well-developed fibro-vascular trabeculae. Some believe that a solitary encapsulated nodule occurring in an otherwise healthy thyroid is a benign new-growth—to wit, an adenoma; others that it is a solitary nodular goitre.

Cystic Degeneration.—Within the adenoma one or more cystic cavities are wont to occur; should they coalesce, a cyst is produced. At first filled with clear straw-coloured fluid, not infrequently hæmorrhage occurs into it from one of the thin-walled blood-vessels that so richly supply the scanty stroma; consequently the content is often brown, and may contain cholesterol crystals.

Clinical Features.—The adenoma is nearly always solitary; occasionally two are present. The favourite site is at the junction of the isthmus with one lateral lobe. Females greatly predominate and the patient may present at any age over ten years, with one or more of the following:

Swelling (fig. 717) is the most common symptom.



FIG. 717.—A large adenoma of the thyroid.

Dyspnœa occurs when the adenoma presses upon the trachea. This is the second most common complaint and by far the most important. In the early stages dyspnœa is noticed only upon exertion; later it worries the patient at night. *Urgent dyspnœa* sometimes comes unheralded from hæmorrhage into a cystadenoma and this may cause sudden death.

Thyrotoxic symptoms tend to develop in old-standing solid adenomas about the age of forty. These symptoms (p. 543) are to be expected in at least 20 per cent. of such cases: some would go as far as to state that sooner or later some toxic features develop in every case (Linnell).

Alteration in the voice due to pressure on a recurrent laryngeal nerve.

Treatment.—Unquestionably, removal of an adenoma of the thyroid should be urged in all cases for the following reasons:

- (i) There is a possibility of a malignant change, even in youth (p. 560).
- (ii) The possibility of thyrotoxic complications.
- (iii) Danger of suffocation (see below).
- (iv) For cosmetic reasons.

If the adenoma is well encapsulated, the operation of resection-enucleation (fig. 718) with careful hæmostasis is adequate, but if there is any doubt the whole of the affected lobe and the isthmus should be excised.

Before leaving this subject, we must deal with

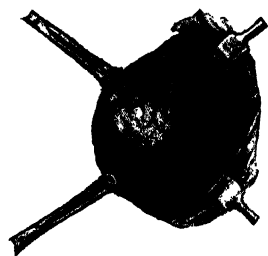


FIG. 718.—Thyroid gland exposed, showing an adenoma suitable for resection-enucleation.

the important question of what to do in a case of impending suffocation from a sudden hæmorrhage into a cystadenoma of the thyroid. The treatment must be immediate and, as a first-aid measure, cold applications are applied and the patient sits up with the head flexed. Aspiration of a cyst with a wide-bore needle is often effective, but in desperate cases an incision over the swelling, *dividing the pretracheal cervical fascia* thereby allowing the adenoma to bulge into the wound (instead of pressing upon the trachea), has proved a life-saving measure.

Hürthle-cell adenoma, though rare, is of considerable interest and importance, for it represents a proven example where an adenoma, if not removed intact within its capsule, becomes a carcinoma. These tumours probably arise from normal thyroid epithelium. Microscopically they consist of large eosinophilic, finely granular, or foamy polygonal cells.

RETROSTERNAL GOITRE

Most retrosternal goitres are acquired. A few are congenital, due to the thyroglossal bud being carried too far downward (p. 566). The acquired form arises as an extension downward of a nodule in the lower pole of either lobe. There are three varieties :

1. **Substernal.**—There is a prolongation of a cervical goitre downwards behind the sternum: this is the most common form which may be palpable in the suprasternal notch, especially if the patient swallows. On rare occasions the prolongation proves to extend not only downwards, but backwards behind the trachea, and sometimes even behind the œsophagus.

2. **Intrathoracic.**—The whole goitre—indeed, in the congenital variety, the whole thyroid—is situated within the thorax between the great veins, and resting upon the aorta.

3. **Plunging Goitre.**—The goitre is wholly intrathoracic, but from time to time it is forced into the neck by raised intrathoracic pressure due to coughing.

Ætiology of acquired cases. Cervical goitres of all kinds are much more common in women ; retrosternal goitre is more common in men, particularly stocky, short-necked individuals. The sternohyoid and sternothyroid muscles prevent forward expansion, and direct the swelling into the superior mediastinum. For a long time the adenoma can rise with deglutition, and descend again through the thoracic inlet. Finally, because of increase in its size, it becomes imprisoned in the thorax.

Clinical Features.—As a rule an intrathoracic goitre remains symptomless for years, and consequently it is exceptional for the patient to present before the meridian of life has been passed.

Masked.—A goitre wholly within the thorax is frequently overlooked, and, when it becomes toxic, the symptoms to which it gives rise may be mistaken for asthma or heart disease.



FIG. 719.—Intrathoracic goitre, showing the reason for the dyspnœa. (After Frank Lahey.)

Dyspnœa and cough are the chief presenting symptoms. Oft-times dyspnœa is severe, and is associated with *stridor*, which is due to deviation and compression or kinking of the trachea. Particularly characteristic is *nocturnal dyspnœa*. The patient may complain that an attempt to sleep on one side, usually the right, produces such difficulty in breathing (fig. 719) that he always sleeps on the other side.

Dysphagia is not uncommon. It is due to pressure on the œsophagus. It is seldom severe enough to be incapacitating.

Hoarseness due to pressure on a recurrent laryngeal nerve, usually the left, occurs in approximately 10 per cent. of cases.

Venous Engorgement.—As a retrosternal goitre enlarges, dilatation of the superficial thoracic veins over the upper part of the chest wall (fig. 720),



FIG. 720.—Engorgement of the superficial thoracic veins due to intrathoracic goitre. (The late Professor Rendle Short, Bristol.)



FIG. 721.—Retrosternal goitre causing deviation and compression of the trachea. (Professor Carl Krebs, Aarhus, Denmark.)

due to pressure upon one or both innominate veins, is liable to occur. Occasionally venous obstruction gives rise to œdema of the face.

Hæmoptysis, unmixed with sputum and possibly copious, occasionally occurs, due to rupture of an engorged tracheal vein (Burgess).

Radiography is particularly valuable as a means of displaying deviation and compression of the trachea (fig. 721). Owing to years of delay in diagnosis, calcification sometimes occurs in an intrathoracic goitre, rendering radiological diagnosis unquestionable.

However, radiographs are usually of little value in differentiating a retrosternal goitre from a thymoma or other neoplasm of the mediastinum, a not infrequent problem.

Radio-iodine studies with a Geiger counter may be helpful in distinguishing an intrathoracic goitre from a mediastinal neoplasm, provided, of course, the retrosternal goitre possesses sufficient normal thyroid tissue to take up ^{131}I .

Treatment.—On no account should treatment with antithyroid drugs or ^{131}I be even considered, as these cause the goitre to swell. Operative treatment is indicated in all cases giving rise to symptoms as soon as thyrotoxicosis, if present, is under control. Regional anæsthesia is par-

icularly desirable in this instance because of the danger of post-operative rachetis, and other pulmonary complications engendered by the passage of an endotracheal tube that may be difficult to introduce. When compared with that of a cervical goitre of corresponding size, removal of a large intrathoracic goitre is hazardous. The risks are greater because of the location, the greater hæmorrhage that may be difficult to control, and the resultant shock. Also the patients are usually older, and may have cardiovascular disease.

Through a standard collar incision the tumour is approached as for subtotal thyroidectomy (p. 551). The blood supply of a retrosternal goitre comes from above. The first step, therefore, is to ligate and divide the middle thyroid veins; the second, to ligate and divide the superior thyroid vessels. Usually, after the cervical portion of the goitre has been mobilised, the delivery and removal of the retrosternal extension presents no insuperable difficulty. When they can be found, the inferior thyroid arteries are ligated well away from the thyroid gland. Gentle traction is exerted on the lateral lobes. The isthmus of the thyroid is brought into view and divided between hæmostats. By further gentle traction on the mobilised affected lobe, it is now possible to deliver the retrosternal portion of the goitre on to the surface in the great majority of cases. Should it be necessary to aid delivery, it is safe to insert a finger below and behind the retrosternal extension (fig. 722). In a few instances when the tumour cannot be brought through the thoracic inlet, its upper part can be incised and enough of its interior removed piecemeal to render it small enough to be delivered and resected. Should there be considerable oozing from the cavity, it can be packed with absorbable gauze followed by ribbon gauze. In other circumstances the wound is closed with drainage. When packing has been employed, it is removed after forty-eight hours.

If the above technique is employed, splitting the sternum (fig. 747) in order to obtain access is rarely necessary.

CARCINOMA OF THE THYROID

Little is known about the pathogenesis of this relatively uncommon condition, which is three times commoner in women.¹ The predominance in women rather suggests that the physiological cycle of hyperplasia-involution may play an important role. Experimental work in rats treated with thyroid-blocking agents suggests that one sequence of events is hyperplasia → adenoma formation → differentiated carcinoma → undifferentiated carcinoma. Forty per cent. of patients with proven thyroid carcinoma have had a goitre for at least five years.

Pathology.—Exact histological classification is difficult, when it is realised that a malignant thyroid may contain several types of malignant cell. Three main pathological varieties are recognised:



FIG. 722.—A stage in the delivery of an intrathoracic goitre.

¹ In the Annual Report of Chief Medical Officer, Ministry of Health (1965), thyroid carcinoma accounted for 298 female and 86 male deaths. As a registered cause of death the incidence is approximately that of carcinoma of the tongue.

1. **Papilliferous** (35 per cent.)¹ occurs most frequently in young persons, even in children, a solitary nodule (adenoma) being the site of predilection. Thirty per cent. show one or more intraglandular seedlings (fig. 723). Often, while the primary growth is still small, this variety of carcinoma metastasizes to the cervical lymph nodes, where it may remain localised for many years prior to further dissemination. Consequently this variety is relatively benign.



FIG. 723.—Papilliferous carcinoma of the thyroid, showing an intraglandular seedling. This specimen demonstrates how necessary it is to remove the whole lobe, if not the whole gland. (F. F. Rundle, F.R.C.S., Sydney, Australia.)

2. **Follicular** (40 per cent.) usually arises in middle-aged persons, and is appreciably more malignant than the foregoing. Commencing as a circumscribed lesion macroscopically indistinguishable from an adenoma, or as a malignant change commencing in a nodule of a multinodular goitre, invasion occurs into and through the capsule, whence local spread is characteristically into the venules, where clumps of growth can often be seen microscopically in the lumen. This explains why this neoplasm metastasises via the blood-stream to bones and the lungs (see treatment).

3. **Anaplastic** (25 per cent.) occurs mainly in elderly people. Unlike other varieties, this tumour commonly arises in a normal thyroid gland. It spreads by direct extension and invasion on a broad front, and metastasises early to the cervical and mediastinal lymph nodes and the viscera. These growths consist for the most part of sheets of undifferentiated cells, either spheroidal, cuboidal, polygonal, or spindle-shaped. Some show considerable pleomorphism. Those exhibiting multinuclear giant cells are devastatingly malignant.

4. Rare forms of thyroid cancer are the amyloiditic and the medullary types.

Carcinoma of the Thyroid Resulting from Radiotherapy.—A considerable increase in the number of cases of carcinoma of the thyroid, often, but not necessarily, papilliferous in type, appearing under the age of eighteen is undoubtedly in part due to a wave of enthusiasm for radiotherapy for the treatment of tuberculous cervical adenitis, enlargements of the thymus, and even enlarged tonsils, that perhaps reached its zenith a decade or so ago. In many series of cases of carcinoma of the thyroid occurring in children no less than 50 per cent. had received X-ray treatment for one or other of the conditions listed.

Clinical Types.—1. Solitary nodule. 2. Local metastasis (lateral aberrant thyroid). 3. Metastatic disease with or without a goitre. 4. Histological surprise. 5. Obvious anaplastic carcinoma.

1. **Solitary Nodule.**—The profession is becoming more carcinoma-conscious with regard to localised swellings of the thyroid. A solitary nodule (adenoma) is looked upon with suspicion, especially when it occurs in a male (such nodules are relatively more often carcinomatous in the male).

¹ For an unknown reason papilliferous carcinoma is becoming more common, and in some series it heads the list.

Likewise a similar swelling in childhood or youth is a potential source of malignancy; indeed, 33 per cent. of such nodules are carcinomatous (Pemberton). It is coming to be recognised that the doctrine 'No removal unless symptoms' is a very dangerous one (fig. 724), and the practitioner is exhorted to exercise the same vigilance in urging the removal and histological examination in the case of these swellings as he has been taught to do in the case of a lump in the breast. The target is to recognise carcinoma of the thyroid early or even in its premalignant state, when a cure can be anticipated. Doubtless a great improvement in results will accrue if and when this principle is accepted generally.

In cases where the thyroid is enlarged, and there is one area more indurated than the remainder of the gland, particularly when the major enlargement is not of long duration, malignancy should be assumed (Cattell). Fixation of the gland (when not due to thyroiditis) causing limitation of movements on deglutition, is an unfavourable sign. Hoarseness usually denotes a fairly advanced stage.

2. **Local Metastasis as a First Symptom.**—Sometimes the patient presents with enlarged lymph nodes, usually unilateral, and possibly the primary growth in the thyroid is so small that it is impalpable. (See Lateral Aberrant Thyroid, p. 566.) Biopsy is then the only method of arriving at the correct diagnosis.

3. **Metastatic Disease with or without a Goitre.**—It is not very unusual for a patient with a carcinoma of the thyroid to present on account of a tumour of a bone (fig. 725) or a pathological fracture thereof. Quite often the primary in the thyroid is overlooked through forgetting the diagnostic rule *Always examine the thyroid, breasts, kidneys and prostate,*

and think of the *bronchi* when confronted with a case of this kind.

As a rule, such a metastasis is apparently solitary, osteolytic, intensely vascular, may pulsate, and frequently gives rise to great pain. Occasionally metastases of a carcinoma of the thyroid (fig. 726) exhibit sufficient secretory activity to produce mild hyperthyroidism; often they contain enough functional thyroid tissue to enable them to be identified by a tracer dose of radio-iodine.



FIG. 724.—Carcinoma of the thyroid that commenced in an adenoma of many years' standing. (The late Professor Rendle Short, Bristol.)



FIG. 725.—Metastasis in the left parietal bone from a carcinoma of the thyroid. (Professor A. K. Toufess, Lahore, Pakistan.)



FIG. 726.—Metastasis from a carcinoma of the thyroid in a humerus. (Dr. S. Devadatta, Vellore, South India.)

4. **Histological Surprise** (e.g. *Carcinoma Secondary to Multinodular Goitre*).—As a rule the patient has had an irregular enlargement of the thyroid for over twenty years. She seeks advice because of recent progressive increase in the size of the swelling (fig. 727) not infrequently accompanied by dyspnoea or dysphagia, and occasionally by pain. Often, however, it is impossible to diagnose a malignant change in these grossly irregular goitres until they have been subjected to histological scrutiny.



FIG. 727.—Carcinoma that originated in a multinodular goitre.

5. **Obvious Anaplastic Carcinoma**.—It is seldom that there is difficulty in diagnosing an anaplastic tumour of the thyroid, for it is hard, fixed, bulging (fig. 728), and usually associated with pressure symptoms of recent origin. One-third of these patients have unilateral vocal cord paralysis when they first present (Taylor).



FIG. 728.—Anaplastic carcinoma of the thyroid. Stony hard neoplasm implicating right sternomastoid. Four months' history.

Stump Recurrence after Operations for Carcinoma of the Thyroid.—Generally a stump recurrence must be regarded as a surgeon's failure; there has been insufficiently wide extirpation of the gland, or the surgeon has shrunk from the risk of injuring the recurrent laryngeal nerves and parathyroids, or carcinoma was unsuspected and histological examination of the specimen was not undertaken. The frequency of local recurrence after operation for papillary carcinoma is high. From a radio-therapeutic clinic Windeyer reported that a quarter of the patients were referred because of local recurrence after previous operation for papillary carcinoma. The interval between the operation and recurrence sometimes is a matter of years.

TREATMENT OF CARCINOMA OF THE THYROID

Operation.—In all instances where carcinoma is even a remote possibility, resection of the lump with a wide margin of healthy thyroid tissue should be followed by an immediate frozen section and pathological report, in the same way as doubtful lumps of the breast are examined. When there are no facilities for this procedure, or the pathological report is equivocal, paraffin sections should be available in forty-eight hours. In event of carcinoma being found as the result of the second method of histological examination, the only safe course is to re-operate forthwith before adhesions make the venture hazardous. The principles in operating for carcinoma of the thyroid are as follows:

No Palpable Cervical Metastases.—Total extracapsular thyroidectomy, with preservation of as much extracapsular parathyroid tissue as possible, is performed. When the pretracheal muscles are implicated, they should be excised, together with the thyroid. Total removal of the thyroid is only practicable if preservation of the recurrent laryngeal nerve is feasible.

Heroic operations for advanced growths gain no advantage and only cause more suffering.

In cases of papillary carcinoma bilateral dissection of the lymph nodes and adjacent tissues is carried out as far laterally as the carotid sheath.¹

When the growth is of the follicular type the internal jugular vein may be resected on the side of the growth, being ligated at the outset, so as to diminish the risk of neoplastic embolus (p. 133).

Palpable and Mobile Cervical Metastases.—In addition to total thyroidectomy, block dissection of the neck (p. 535) may be performed on the side affected, including the pretracheal muscles but sparing the submaxillary triangles, which are never implicated.

Radio-Iodine Treatment.—Radio-iodine treatment is the method of choice only if a thyroid neoplasm takes up radio-iodine. Follicular carcinoma concentrates iodine better than any of the other neoplasms, but not so efficiently as normal thyroid tissue. A test dose is given (100mc.—larger than a diagnostic tracer dose, p. 539), and a whole body scan is performed at six and twenty-four hours. In this way the presence of metastases will be detected. Should the metastasis take up ¹³¹I, a total thyroidectomy is the next step. Six weeks after operation, by which time the patient is developing myxœdema and the output of TSH by the pituitary is high (p. 538), a large ablation dose of ¹³¹I is given (at least 100 mc.). The ablation dose may be repeated after a few months, and in the interim the patient is maintained on L-thyroxine in adequate dosage.

Great care must be taken in the handling of the patient's excreta and bedclothing, because of the high radioactivity. The code of practice for safety in the use of radioactive substances must always be strictly enforced.

Untoward Reactions.—A few patients suffer from irradiation sickness (fatigue, headache, loss of appetite, and vomiting). Irradiation parotitis, including dryness of the mouth (consequent upon diminished secretion of the salivary glands which always concentrate radio-iodine), painful swelling of the thyroid, if present, and of the metastases, and the rheumatic pains referred to on p. 546 are rather frequent.

Deep X-ray Treatment.—Anaplastic growths (which do not take up radio-iodine for that treatment) often show a good initial response but early recurrence is almost inevitable. So-called prophylactic irradiation has no general place after an operation for malignant disease of the thyroid (Windeyer). The trachea and pharyngoesophagus always receive a heavy dose, causing great and prolonged discomfort. Should the X-rays be directed on a recently healed operation wound, breaking down with discharge of suture material is likely to occur.

Thyroxine.—The administration of L-thyroxine, just short of clinical toxicity, depresses the pituitary thyrotropic hormone (p. 538), and, in some cases, may discourage recurrences.

SECONDARY NEOPLASMS OF THE THYROID

The most common primary neoplasms to produce secondaries in the thyroid gland are: hypernephroma, carcinoma of the colon, rectum, breast and lung, and tumours of the lymphatic system.

In suspected cases, and when there is no evidence of secondary deposits in other organs, thyroidectomy is recommended, not only to prove the diagnosis but to prevent the inevitable infiltration and progressive obstruction of the trachea and œsophagus. Palliative X-ray therapy may be indicated, especially in inoperable cases, and terminal tracheostomy is occasionally necessary.

PRIMARY RETICULOSARCOMA OF THE THYROID

The existence of this uncommon condition has been established only in recent years and its differentiation from the small-celled anaplastic carcinoma is difficult.

¹ McClintock advises that the sternum be split, to enable *en bloc* excision of the lymphatic field in the superior mediastinum.

John C. McClintock, *Contemporary*. Associate Clinical Professor of Surgery, Albany Medical College, Albany, New York.

Brian Wellingham Windeyer, *Contemporary*. Dean of the Faculty of Medicine and Professor of Radiology University of London (Middlesex Hospital).

There are no distinctive diagnostic features and a clinical diagnosis is extremely difficult to make. The pathological types are as set out on p. 155.

Treatment.—The diagnosis is made after routine thyroidectomy. All cases should have a course of X-ray therapy as reticulosarcoma is a very radio-sensitive tumour, and the field of irradiation should include the thyroid gland, the lower cervical lymph chains, and the superior mediastinum.

THYROIDITIS

Hashimoto's Disease (Struma Lymphomatosa, Lymphadenoid Goitre).—Hashimoto's disease is not an inflammation. It is an auto-immune disease, the nature of which and the role of antibodies in its production was defined by Roitt and Doniach in 1956.

Pathology.—Macroscopically the colour of the thyroid is pale pink ranging to yellowish white, according to the amount of fibrosis present. In early

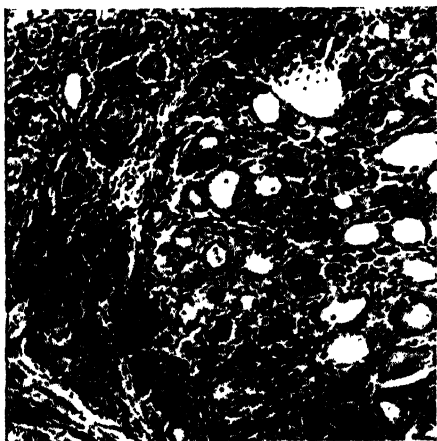


FIG. 729. — Hashimoto's disease (struma lymphomatosa). $\times 100$. Intense lymphocytic-plasma cell infiltration, acinar destruction, and fibrosis. Compare with the normal histology (fig. 700). (Dr. Bernard Fox, London.)

cases it gives the impression of being semi-translucent. Microscopically, no normal thyroid tissue is present. Throughout the gland lymphoid follicles and lymphoid cells abound (fig. 729).

Clinical Features.—The disease nearly always affects females (often middle-aged), and frequently produces no symptoms until hypothyroidism appears. The goitre may be diffuse, but one lobe, the isthmus, or the pyramidal lobe may be predominant. The gland feels firm, like india-rubber, and usually is not tender. The surface is often irregular (bosselated).

Investigations.—The diagnosis is confirmed by demonstrating the presence of thyroid antibodies in the serum. The most sensitive test is the tanned red cell agglutination titre test (TRCT), as a result of which titre values up to one in five million may be found. If both the TRCT and a complement fixation test are used in conjunction, 90 per cent. of the cases will be diagnosed.

The radio-iodine neck uptake may be normal, raised, or low, and is of no actual diagnostic value; it will, however indicate the extent of the thyroid reserve, for as the auto-immune process destroys the gland, the reserve falls to zero. This is mirrored by the neck uptake of radio-iodine falling from a hyperfunctioning range (during the period when there is regeneration of unaffected thyroid tissue) to zero. The process takes many years. The chemical PBI (p. 539) is normal until this last stage is reached, when it falls to a hypothyroid level.

Treatment.—When the diagnosis is certain the only treatment indicated is an adequate dose of thyroid hormone and careful follow-up.

Operation.—In cases where the diagnosis is in doubt, where there are

pressure symptoms, or when there is no response to treatment and the possibility of carcinoma cannot be ruled out, a partial excision-biopsy, subtotal or total thyroidectomy is the correct course.

Complications and other Features.—Carcinoma develops, usually papillary and of low malignancy, in 10 per cent. of cases. Occasionally reticulosarcoma and lymphosarcoma occur. Hürtle-cell adenoma (p. 557) is found with greater frequency than in other varieties of goitre. Luxton, in a series of thirty-five cases found that seven had Paget's disease of bone (p. 246), and in one-quarter of the cases the liver and spleen were enlarged. Finally there may be association with gastric antibodies and pernicious anaemia, and also Addison's disease (p. 578).

Riedel's Thyroiditis (Struma Fibrosa).—A rare disease of obscure ætiology, though recent evidence shows that the thyroid involvement is but one manifestation of a widespread collagen disorder, the main feature of which is extensive and severe perivascular fibrosis. The goitre, which may enlarge very rapidly, is very firm and may be tender. It becomes adherent to the surrounding structures (suggesting a carcinoma), and it may cause obstructive symptoms due to the severe degree of fibrosis. Associated with the thyroid enlargement there may be retroperitoneal fibrosis (Chap. 45) and peripheral vascular deficiency.

Investigations show a raised E.S.R. absence of thyroid anti-bodies, and a non-functioning goitre in respect of radio-iodine uptake.

Operation.—The diagnosis is confirmed by biopsy—excision of the affected lobe and adherent muscles. This procedure also relieves obstructive symptoms if present. Should there be any question of difficulty, especially where the recurrent laryngeal nerve is concerned, discretion is the better part of valour, and having removed an adequate portion for histology, the wound should be closed with drainage.

Acute and subacute thyroiditis (de Quervain's disease) is a rare condition characterised by sore throat and pain on swallowing due to the small tender goitre. There is a fever and a raised E.S.R. A mild degree of thyrotoxicosis usually co-exists due to the release of thyroid hormones as a result of the inflammatory reaction.

Investigations (p. 538) show a raised B.M.R. and chemical PBI, but a low or absent radio-iodine uptake. The main differential diagnosis is from a subacute form of Hashimoto's disease, which is distinguished by the presence of thyroid antibodies, not seen in de Quervain's disease. *Treatment.*—The disease responds very well to prednisone 10 mg. 6-hourly, and remits within a few weeks.

ECTOPIC THYROID AND ANOMALIES OF THE THYROGLOSSAL TRACT

Ectopic and Aberrant Thyroid Tissue.—The whole thyroid gland may be situated in some part of the thyroglossal tract, in which case the third and fourth rings of the trachea, which are normally covered by the isthmus can be palpated easily.

Lingual thyroid is the most common of these rare abnormalities. This variety of ectopic thyroid gland gives rise to a rounded swelling at the back of the tongue beneath the foramen cæcum (fig. 730), and produces dysphagia, impairment of speech, and occasionally compromises the airway. In about 10 per cent. of cases removal of a lingual thyroid is followed by myxœdema, for the abnormally situated gland is the only thyroid tissue present.

Median (Thyroglossal) Ectopic Thyroid.—In the case of an ectopic thyroid situated in the upper two-thirds of the neck (fig. 731 B), the swelling it causes is usually mistaken for a thyroglossal cyst (p. 566), which is much more common. When performing an operation for a supposed thyroglossal cyst, if the swelling is found to be solid and composed of thyroid substance, a little of the thyroid tissue should be spared in order to prevent myxœdema. This type of ectopic thyroid is often the only thyroid tissue present.



FIG. 730.—Lingual thyroid.
(H. Wapshaw, F.R.C.S., Glasgow.)

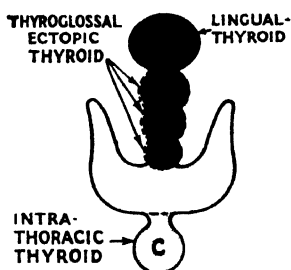


FIG. 731.—Ectopic and aberrant thyroids. A and B are ectopic thyroids, C intrathoracic aberrant thyroid (nearly always acquired).

thyroid tissue in the ovary can function and maintain euthyroidism, if not hyperthyroidism; it can also undergo non-toxic nodular changes, as well as malignant degeneration with metastases. Thyrotoxicosis due to struma ovarii should be suspected if there is no ^{131}I uptake in the week and a high 48-hour PBI 131 (p. 539).

ANOMALIES OF THE THYROGLOSSAL TRACT

Thyroglossal cyst may present in any part of the thyroglossal tract (fig. 732). The common situations, in order of frequency, are beneath the hyoid

FIG. 732.—Possible sites of a thyroglossal cyst: (1) Beneath the foramen cæcum. (2) In the floor of the mouth. (3) Suprahyoid. (4) Subhyoid. (5) On the thyroid cartilage. (6) At the level of the cricoid cartilage.



(fig. 733), in the region of the thyroid cartilage (fig. 734), and above the hyoid bone (fig. 735). Such a cyst occupies the middle line, except in the region of the thyroid cartilage, where the thyroglossal tract is pushed to one side, usually to the left.

Thyroglossal cysts are the seat of recurrent attacks of inflammation



FIG. 733.—Subhyoid thyroglossal cyst. The commonest variety.



FIG. 734.—Thyroglossal cyst in relation to the thyroid cartilage.



FIG. 735.—Suprahyoid thyroglossal cyst.

(fig. 736), due to the fact that the wall contains nodules of lymphatic tissue, which communicate by lymphatics with the lymph nodes of the neck. When inflamed they are often mistaken for abscesses and incised, which is one way in which a thyroglossal fistula arises.



FIG. 736.—Inflamed thyroglossal cyst.

of the neck as a whole and that of the thyroglossal tract), is characteristic. A thyroglossal fistula is lined by columnar epithelium, discharges mucus, and is the seat of recurrent attacks of inflammation.

Treatment of a thyroglossal cyst and a thyroglossal fistula is essentially the same. Every vestige of the thyroglossal tract must be removed right up to the foramen cæcum, otherwise a discharging fistula is almost inevitable. Because of difficulty in defining the tract in the region of the hyoid bone, the centre

Thyroglossal fistula is never congenital and it follows inadequate extirpation or incision of a thyroglossal cyst. Long-standing fistulæ are inclined to be situated low down in the neck, and fig. 737 shows an example that had been present for twenty years. The hood of skin, with its concavity downwards (due to uneven rates of growth

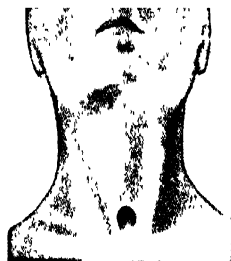


FIG. 737.—A long-standing thyroglossal fistula. The semilunar fold is characteristic.

of this bone is resected in the course of the dissection (Sistrunk's operation) (fig. 738). Before embarking upon the removal of a supposed thyroglossal cyst, it is well to make sure that there is a thyroid gland present in the normal position, for the swelling in question may be an ectopic thyroid (p. 565).



FIG. 738.—Complete extirpation of the thyroglossal tract. A portion of the body of the hyoid bone has been removed, and the dissection is proceeding towards the foramen cæcum.

CHAPTER 25

THE PARATHYROID GLANDS, THE THYMUS, AND THE ADRENAL GLANDS

THE PARATHYROID GLANDS

Anatomy and Physiology.—Like man, most animals have four parathyroid glands, except the rat, which has two. These small glands, oval in shape, each about 0.5–1 cm. in size are arranged in pairs. They are pink in the young, coffee-brown to reddish-yellow at puberty, and from puberty onwards varying shades of yellow, depending upon the amount of fat deposition. The superior pair are developed from the fourth pharyngeal pouches while, paradoxically, the inferior pair arise (together with the thymus) from the third pharyngeal pouches (footnote p. 537), and migrate caudally. The blood supply of both sets of glands comes from the inferior thyroid artery.

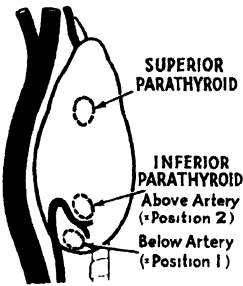


FIG. 739.—Posterior view of the left lobe of the thyroid, depicting the typical locations of parathyroid glands. (After the late Sir James Walton.)

The parathyroid glands are situated outside the thyroid capsule, although occasionally a superior parathyroid lies deeply within a furrow of the thyroid gland. In 8 per cent. of subtotal thyroidectomies a portion of parathyroid tissue is found within the extirpated specimen. The inferior pair is inconstant in position (a) in relation to the inferior thyroid artery (fig. 739), (b) because they may be found in the superior and posterior mediastinum (fig. 741).

Histology.—Each gland is composed of (1) principal (basophilic) cells; (2) water-clear cells; (3) oxyphilic cells. The last variety increases with age.

Function.—The parathyroids produce parathormone, a polypeptide with a molecular weight of 8600 (Rasmussen) which controls the intra- and extracellular disposition of calcium. Bone and kidney are the chief target organs; calcium and phosphate are mobilised from bone by direct action; phosphate is excreted by the kidneys because of depressed tubular reabsorption or by increased phosphate secretion. There is a reciprocity between phosphates and calcium. In hyperparathyroidism there is phosphaturia, a rise in the serum calcium (removed from the bone-storehouse), and then a rise in the urine calcium.

Calcitonin (Copp) may be the second parathyroid hormone, though it is also known as Thyrocalcitonin as it has been extracted from thyroid venous blood. It lowers the serum calcium and effects calcium storage in bones; quite the opposite action to parathormone.

Blood Chemistry.—The normal serum calcium level is 4.5 to 5.5 mEq. per litre (9 to 11 mg. per 100 ml.). Serum phosphorus levels should be 1.8 to 2.7 mEq. per litre (3 to 4.5 mg. per 100 ml.). The product of $\text{Ca} \times \text{P}$ is held at about 40. Levels are slightly higher in children.

HYPOPARATHYROIDISM

Parathyroid tetany is a rare complication of thyroidectomy (1 per cent.) which occurs most frequently from one to five days after operation, but occasionally mild forms are not recognised for several weeks. Partial parathyroid deficiency occurs after 7 to 10 per cent of thyroidectomy patients. In 50 per cent. of cases in which parathyroid tetany occurs, two or more parathyroids are found embedded in the specimen removed; in the remainder it is assumed that the glands are deprived of their blood supply at operation.

Clinical Features.—The first symptoms are tingling and numbness of the lips, nose, and the extremities, sometimes accompanied by circum-oral pallor. The serum calcium is below normal levels.

Chvostek's Sign.—With a percussion hammer gently tap the seventh nerve as it courses in front of the external auditory meatus. In tetany, the tapping of the hyper-excitable nerve provokes a brisk muscular twitch on the same side of the face.

Trousseau's Sign.—A pneumatic tourniquet is placed around the arm, and the pressure is raised to 200 mm.Hg. If tetany is present, typical contractions of the hand are seen within three to five minutes. The fingers are extended, except at the metacarpophalangeal joints, and the thumb adducted to produce the 'obstetrician's hand' (fig. 740).

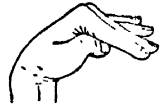


FIG. 740. — The 'obstetrician's hand' seen in parathyroid tetany.

In severe cases painful cramp of the hands, feet, and indeed any of the muscles of the body occurs. Strong adduction of the thumbs is almost always present, and this, coupled with plantar-flexion of the feet, constitutes the so-called carpo-pedal spasm which typifies parathyroid tetany. Generalised fits may occur. Occasionally spasm of the muscles of respiration results in severe dyspnoea, and the patient is not only in great pain, but is in mortal dread of suffocation. Blurring of vision due to spasm of the intra-ocular muscles is common; even if the symptoms are mild, cataracts due to prolonged hypocalcæmia are a frequent late complication of the condition.

Differential Diagnosis.—Tetany, a state of neuromotor excitability dependent upon hypocalcæmia, also occurs occasionally (a) in the new-born idiopathically, (b) in adults with long-continued vomiting due to pyloric obstruction, (c) rarely after operations upon the stomach, or (d) as a late complication of hypertrophic pyloric stenosis or diarrhoea and vomiting of infants. Tetany in all these conditions, unlike that of hypoparathyroidism, is due to alkalosis, consequent upon loss of hydrochloric acid from the stomach.

The symptoms of hypoparathyroidism may bear a remarkable resemblance to epilepsy, and the former condition should be considered in idiopathic convulsions.

Treatment.—*Prophylactic Treatment.*—When a parathyroid gland is excised inadvertently during subtotal thyroidectomy for a benign condition, the gland should be separated from the extirpated thyroid tissue and embedded in a sternomastoid muscle. The graft survives only a short time, but it may tide the patient over a critical period.

Immediate treatment of parathyroid tetany is to inject 10 to 20 ml. of a 10 per cent. solution of calcium gluconate intravenously. The injection should be made slowly, and it causes almost immediate relaxation of the spasms. A few minutes after the intravenous injection a similar injection of calcium gluconate is given intramuscularly, to give a more prolonged effect.

When spasms make intravenous injection difficult, 5 to 10 ml. of paraldehyde can be injected intramuscularly twenty minutes before the intravenous injection of calcium. In an unusually severe case it is necessary to repeat the injections until satisfactory control of the spasm has been obtained. If the patient can swallow, soluble calcium aspirin in doses of 30 G is effective in mild cases.

Maintenance treatment

1. **Vitamin D₂ (Calciferol) and Oral Calcium.**—These substances are the mainstay of treatment in doses of 50,000 to 200,000 units of calciferol, calcium lactate or soluble calcium aspirin. Serum calcium estimations will be required at intervals. In addition to vitamin D₂ and calcium, it is most important to place the patient on a low phosphorus-containing diet. The intake of fish, relatively high in phosphorus, should be limited.

After a varying time, some patients become completely insensitive to calciferol, and reliance must be placed on calcium lactate or calcium aspirin.

2. **Parathyroid Extract (parathormone).**—Parathyroid extract has the disadvantages that it is expensive, it must be given by injection, and it loses its effect after a few weeks or months. Thirty units are given intramuscularly twice daily. While treatment is in progress, either a diet rich in calcium is given, or eight grammes of calcium lactate is added to the diet.

Precautions to be taken in all Forms of Treatment of Parathyroid Tetany.

—Control of the treatment by serial calcium estimations of the blood is important because hypercalcaemia can give rise to calcification, renal damage, and toxæmia culminating in coma and death. Fortunately, in over half the cases the tetany disappears spontaneously in about a month. In a few cases it is found that treatment can be dispensed with after a longer period, but in the remainder it must be continued indefinitely. A mother should not suckle her child, as this causes loss of calcium in the milk.

HYPERPARATHYROIDISM

Hyperparathyroidism can result from an adenoma (rarely a carcinoma) of one, exceptionally of two, parathyroids, or, on much rarer occasions, from primary hyperplasia of all.

A **parathyroid adenoma** is a small, well-encapsulated tumour 2 to 4 cm. diameter, dark red in early cases, yellow in those of some standing. An *inferior* parathyroid gland has been the seat of this neoplasm in 80 per cent. of the reported cases. In 20 per cent. of cases the neo-

plasm occupies an aberrant position, viz. in the mediastinum, within the thyroid gland, and behind the œsophagus, in that order (fig. 741). In structure the neoplasm closely resembles normal parathyroid tissue. The sex incidence of the neoplasm is three females to one male.

Parathyroid hyperplasia presents a characteristic histological picture—a large proportion of water-clear cells. There is considerable enlargement of all the parathyroid glands (2 to 4 cm.).

Clinical Features.—Hyperparathyroidism comes in many guises:—

1. *Osteitis fibrosa cystica* (von Recklinghausen's disease) (p. 248).

2. The so-called 'brown tumour' of hyperparathyroidism, particularly of the mandible and maxilla (a variety of 'giant-cell tumour' of bone).

3. *Urinary calculi.* While hyperparathyroidism is responsible for only 5 per cent., investigations into the possibility of this condition should be undertaken in all cases of renal calculi. More cases of hyperparathyroidism are discovered in this way than all the others put together (Albright). *Nephrocalcinosis*, radiologically demonstrable as calcified streaks in the line of the tubules, is characteristic.

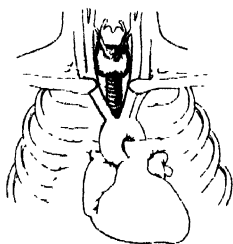


FIG. 741.—A parathyroid adenoma may be situated in any part of the area shaded yellow. Ectopic positions are explained by (1) embryological migration or (2) thoracic suction. (After W. F. Rianhoff Jnr.)

4. *Diagnostic problems*—patients with nausea, vomiting, anorexia and weakness.
5. *Peptic ulceration* is a not uncommon complication of hyperparathyroidism, occurring in 15 per cent. of cases. The only characteristic symptom is that the pain is made worse by alkalis. Fatal hæmatemesis is relatively frequent.
6. *The 'arthritic' presentation*—simple osteoarthritic effusion into one or more joints.
7. *Renal rickets and renal dwarfism*, and other types of renal osteodystrophy. In these conditions the associated parathyroid hyperplasia is *secondary* to the renal condition.
8. *Steatorrhæa* from any cause, e.g. fibrocystic disease of the pancreas, with its loss of calcium from the alimentary canal in the form of soaps may, by reason of low blood calcium and the body's call to raise the low calcium level, provoke *secondary* parathyroid hyperplasia.

In most cases symptoms have been present for two or more years before the correct diagnosis is made and confirmed. The average age of the patients at the time of establishing the diagnosis is forty-four. As it is usually impossible to detect a parathyroid tumour by palpation (if a lump is felt it is frequently a coincident thyroid adenoma), and as such a tumour is rarely sufficiently dense to cast a shadow on radiological examination, and as there are no local signs in the case of parathyroid hyperplasia, the only methods of confirming a possible diagnosis of hyperparathyroidism are by:

Radiographic Skeletal Changes.—The most characteristic X-ray finding is subperiosteal resorption of bone, best seen in the middle phalanges; this change is often accompanied by reabsorption of the tufts of the terminal phalanges (fig. 742). These changes are also well seen in the lamina dura of the tooth sockets. The bones show small clear cystic spaces and the skull may have a characteristic homogenous 'ground-glass' or granular pattern of decreased density. There may be soft tissue metastatic calcification.



FIG. 742. — Reabsorption of the tuft of a terminal phalanx. (D. R. Davies, F.R.C.S., London.)

Biochemical Investigations.—These show:

1. An increased excretion of phosphorus in the urine.
2. An increased excretion of calcium in the urine.
3. Elevation of serum calcium above the normal 9 to 11 to as much as 20 mg. or more per 100 ml. Repeated estimations are necessary to confirm the raised serum level.
4. Diminution of serum phosphorus below 3 mg. per 100 ml. (reliable when renal function is good).
5. A raised serum alkaline phosphatase when there is bone involvement.
6. *The Sulkowitch Test.*—The patient is given a diet containing 125 mg. of calcium daily for three days. The test is positive when more than 200 mg. of calcium per day is excreted in the urine. In normal individuals less than 100 mg. is excreted under these conditions.

Treatment.—The only curative treatment is parathyroidectomy.

Parathyroidectomy.—The patient takes 4 gm. (60 grains) of calcium lactate and 0,000 units of calciferol, together with 1 litre (2 pints) of milk daily for three days before the operation, and for six months afterwards. The thyroid gland is exposed as for thyroidectomy (p. 551), and the parathyroid tumour is sought by sight



FIG. 743.—Parathyroid tumour exposed.

and touch. If it is not soon apparent, the superior pole is mobilised by ligating and dividing the superior thyroid vessels first on one side and then, if necessary, on the other. *If a parathyroid tumour is found* (fig. 743), it is dissected out or, should it be buried in thyroid tissue, the lateral lobe of the thyroid in which it is embedded is resected: this completed, the operator must not rest content. It is essential to continue a systematic search, because sometimes more than one parathyroid is the seat of an adenoma. *When a parathyroid tumour is not found*, and all or several of the parathyroid glands are larger than normal, parathyroid hyperplasia must be assumed, for should a hidden parathyroid tumour be present, the other parathyroids would be not larger but smaller than normal (from disuse atrophy). If hyperplasia

seems assured, three, or even three and a half, of the glands should be excised.

When the parathyroids appear normal, or smaller than would be expected, and an ectopic parathyroid tumour cannot be located by palpation and blunt dissection in the region of the cervical oesophagus, the sternum is split and the tumour is sought in the anterior mediastinum, removing the thymus if necessary (p. 574). After parathyroidectomy for adenoma, tetany is not uncommon, and precautions must be taken by giving calcium and parathormone (p. 570), until the remaining parathyroids recommence to function. Frequent serum calcium estimations should be performed.

Prognosis.—When a parathyroid tumour is found and removed in the comparatively early stages of the disease, the outcome is often striking. Skeletal decalcification, if present, gives place to recalcification; a 'brown tumour' of bone-marrow resorbs, and it is improbable that urinary calculi, if removed *in toto*, will recur. On the other hand, extirpation of parathyroid glands that are the seat of hyperplasia, although ameliorating symptoms of hyperparathyroidism, is rarely curative.

Carcinoma of a parathyroid is very rare. The tumour may be palpable. Usually these tumours secrete parathormone; they metastasise early. Radiotherapy has been employed for recurrences, without success.

THE THYMUS

Anatomy.—The gland originates as a diverticulum of the third and sometimes the fourth pharyngeal pouch on each side. Epithelial in origin, it soon assumes a lymphoid character. The body of the thymus consists of two lobes closely applied to one another in the middle line, overlying the upper part of the pericardium and the great vessels, and extending upwards into the base of the neck. Each lobe is overlapped by pleura, and above this level has a slender pole passing to the isthmus of the thyroid. The blood supply of the gland is derived mainly from the internal mammary arteries. After puberty, the thymus commences to atrophy, and is replaced by fat, but even up to, and after, the age of fifty, the gland still contains a considerable amount of characteristic lymphoid tissue with epithelial elements, notably Hassall's corpuscles.

The Thymus and Immunity.—The thymus is believed to exert an influence over the reticulo-endothelial system, with respect to the development of immune antibodies. Possibly thymic cells are seeded into the lymph nodes, bone-marrow, and spleen. Certainly, thymectomised rabbits and mice have a lowered resistance to infection, and they will readily accept skin and tumour homografts (Miller).

Hyperplasia of the thymus occurs in toxic goitre, acromegaly, in some cases of Addison's disease, after castration, after bilateral adrenalectomy, and in myasthenia gravis. In infancy the gland may be enlarged (fig. 744). In the past this condition was called 'status thymo-lymphaticus', and thought to be an explanation of sudden death under anaesthesia in children. Anaesthetists now deny the possibility.

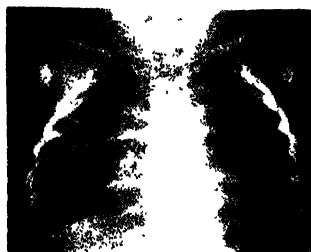


FIG. 744.—Enlargement of the thymus in infancy. (Professor Carl Krebs, Aarhus, Denmark.)

Arthur Hill Hassall, 1817–1891 Physician, Royal Free Hospital, London.

Jacques Francis Miller, Contemporary. The Walter and Eliza Hall Institute of Medical Research, Melbourne, Australia.

Thomas Addison, 1793–1860. Physician, Guy's Hospital, London.

MYASTHENIA GRAVIS

This is a disease in which transmission of motor-nerve impulses at the neuromuscular junctions is blocked by interference with the action of the transmitter—acetylcholine—upon the muscle fibres.¹

Clinical Features.—The disease occurs in both sexes and commences, as a rule, in early adult life. The essential symptoms are profound fatigue after modest exertion, and transient paresis of voluntary muscles. Muscles

supplied both by the cranial and spinal nerves are affected, the extrinsic ocular muscles being involved most constantly. Usually ptosis (fig. 745), a squint, and diplopia are the first symptoms. Later manifestations are drooping of the mandible, with weakness of mastication and difficulty in swallowing. The patient exhibits extreme fatigue on



FIG. 745.—Myasthenia gravis. Before neostigmine. (Arthur Makey, F.R.C.S., London.)

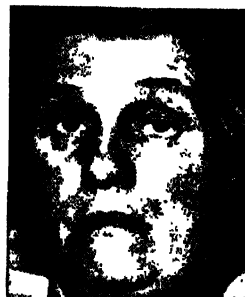


FIG. 746.—Myasthenia gravis. One hour after neostigmine. (Arthur Makey, F.R.C.S., London.)

the least exertion, rendering her incapable of almost any sustained activity. The voice becomes weak, and in some cases death occurs within a few months from involvement of the muscles of respiration. Usually the disease runs a chronic progressive course, but partial remissions occur. The reflexes remain normal, and there is no sensory loss. Occasionally myasthenia gravis complicates primary thyrotoxicosis. Should a patient with myasthenia gravis become pregnant, remission of symptoms during pregnancy often occurs.

Confirmatory Tests.—(1) Two mg. of neostigmine with $\frac{1}{100}$ grain (0.65 mg.) of atropine sulphate (to check increased peristalsis), are injected hypodermically. In about half an hour (in comparatively early cases) pareses disappear (fig. 746) for a few hours. (2) The affected muscles often show the myasthenic reaction—fatigue with faradic, but not with galvanic, stimulation.

Radiography, particularly a lateral radiograph, of the superior mediastinum should always be undertaken. In 12 per cent. of cases a thymic neoplasm is revealed. Apart from tumours, there is no X-ray evidence of thymic enlargements in myasthenic patients.

Treatment.—Medical.—Orally, from 4 to 15 tablets (15 mg. each) of neostigmine can be given in the twenty-four hours. They should be so spaced as to enable meals to be taken without difficulty in mastication. Pyridostigmine is a related longer-acting compound which can be used in combination with, or instead of, neostigmine. In critical cases neostigmine may be given in a slow drip infusion. Oral potassium salts

¹ At a postgraduate course in 1934, Mary Walker, a Scottish doctor working in London, learned that physostigmine counteracts curare, and gave the former to a myasthenic patient. Luckily this patient was spared the side-effects (vomiting). This breakthrough met with fierce opposition, but she was vindicated by the successful series of Dr. L. P. E. Laurent, Physician, West London Hospital (who had reported remission during pregnancy—above), and her introduction of prostigmine (neostigmine) which has less side-effects.

Alfred Blalock, 1899–1964. Surgeon-in-Chief Emeritus, Johns Hopkins Hospital, Baltimore, U.S.A., was the first to perform thymectomy successfully for myasthenia gravis in 1936.

given in large dosage (12g per day) are often beneficial. It must be emphasised that surgery is considered only if medical treatment fails.

Operative.—Thymectomy.—A longitudinal incision is made in the middle line from just below the cricoid cartilage to the level of the fourth costal cartilages. The

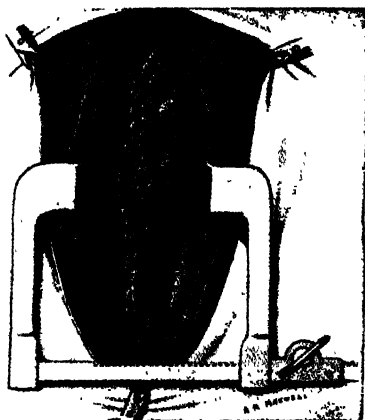


FIG. 747.—Exposure of a tumour of the thymus by splitting the sternum. (Sir Geoffrey Keynes, F.R.C.S., London.)

sternohyoid muscles are separated, the index finger is inserted under the manubrium, and on each side the layers of the pleura are displaced postero-laterally. The sternum is split by sawing through the outer table with a Hey's or circular saw, and dividing the inner table with a sternum splitter. The divided bone edges are separated with bone hooks and the pleura is pushed away from the sternum until the level of the fourth costal cartilages is reached. The sternum is then divided transversely at the level of the third interspace, which permits the divided edges of the sternum to be separated by a strong self-retaining retractor, exposing the anterior mediastinum (fig. 747). The pleurae are dissected still farther away from the middle line, thus displaying the thymus, which is attached by connective tissue that is not very dense to the ascending aorta and the pericardium. Thymic arteries and veins from the internal mammary and inferior thyroid vessels require

ligation. The gland is liberated, avoiding damage to the pleura and the innominate vein. A pneumothorax imposes a further burden on respiration. The divided sternum is approximated with encircling sutures, and the soft parts are reunited. Special attention is given to prevent and treat any chest infection, because of the severe muscle weakness.

Results of Thymectomy.—The best results are obtained in the younger patients with long histories. In about half the cases without an adenoma, the thymus on histological examination proves to be hyperplastic; in the other half it appears normal. Thirty per cent. are cured completely; of the remainder, many are improved considerably. The operative mortality is about 10 per cent. If an extract made from these apparently normal glands is injected into normal animals or persons, temporary muscular paresis results (Wilson).

X-ray Therapy.—In cases where a neoplasm of the thymus is demonstrated by radiography, radiotherapy is given for three months before thymectomy is undertaken (Geoffrey Keynes), otherwise the mortality is high.

Tumours of the thymus are not uncommon. Classification is difficult, but Thompson and Thackray describe three main histological types: (1) Epithelial. (2) Lymphoid. (3) Teratomatous. The epithelial type is the commonest, and is made up of the following sub-groups: (a) differentiated or epidermoid, (b) oval or spindle celled, (c) lympho-epithelioma, (d) granulomatous, (e) undifferentiated. The differentiated or epidermoid tumour is composed of sheets of large polygonal cells, stemming from the Hassall's corpuscles, and is infiltrated by lymphocytes. Clinically, it is associated with myasthenia.

Thymic tumours are diverse in behaviour. Some are simple growths, while others are either locally malignant or show a propensity to metastasise to lymph nodes and abdominal viscera. The lymphoid tumours are lymphosarcomas, while the teratomas are equally malignant and include examples of chorion-carcinoma.

Cyst formation, lobulation by fibrous tissue, necrosis, and calcification are frequently seen.

Clinical Features.—Many tumours are symptomless and are discovered accidentally on X-ray examination of the thorax (fig. 748); occasionally the presenting feature is

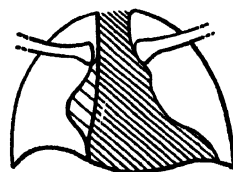


FIG. 748.—Tracing of a radiograph showing unilateral thymic tumour. The patient was radiographed because he had pneumonia. (After H. E. Sorensen.)

William Hey, 1736-1819. Surgeon, the General Infirmary, Leeds.

Andrew Wilson, Contemporary. Professor of Pharmacology, University of Liverpool.

Sir Geoffrey Langdon Keynes, Contemporary. Consulting Surgeon, St. Bartholomew's Hospital, London.

Robert Edward Mercey Thompson, Contemporary. Reader in Bacteriology, Bland-Sutton Institute of Pathology Middlesex Hospital, London.

Alan Christopher Thackray, Contemporary. Professor of Morbid Anatomy, University of London (Middlesex Hospital).

myæsthenia. Large tumours cause stridor, dysphagia, prominent veins over the thoracic inlet, and sometimes cyanosis of the face and upper extremities.

Treatment.—After a course of X-ray treatment an attempt to remove the tumour should be made. Most thymomas are radio-sensitive, but are not radio-curable. Tumours situated on one side of the mediastinum only can be removed through a standard thoracotomy incision with resection of one rib: when necessary the incision can be extended by transection of the sternum. A tumour that projects into both sides is removed through a sternum-splitting incision, but it is not always possible to remove the whole growth. Post-operative irradiation is advisable in all cases.

Is Hodgkin's Disease a Thymic Tumour?—Some believe that Hodgkin's disease is a tumour arising in the thymus gland, and metastasising from this site. "Are not many mediastinal masses seen at radiography thymic tumours, rather than enlarged mediastinal lymph nodes?" asks A. D. Thomson. It is possible that some examples of Hodgkin's disease arise in the thymus (fig. 749) or in ectopic thymus tissue in the neck or the thorax. Certainly the 'owl eye' cells of a granulomatous thymoma are indistinguishable from the Dorothy Reed cells of Hodgkin's disease. The thymic hypothesis of the origin of Hodgkin's disease raises the question of cure of early cases of this fell disease by extirpation of the primary growth, together with metastases (see also 'The Thymus and Immunity' p. 572).

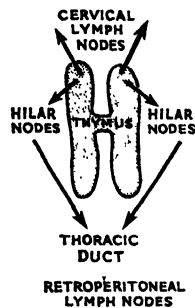


FIG. 749.—Lymphatic drainage of the thymus. (After A. D. Thomson.)

THE ADRENAL GLANDS

Surgical Anatomy.—At birth, the adrenal glands have attained nearly adult proportions. Fully developed, each weighs about 4 gm., but the left is a little larger than the right. A deeper yellow colour and a firmer consistency enables the gland to be distinguished from the adjacent fat. Each rests on the superior, anterior, and medial aspects of the superior pole of the corresponding kidney, and presents the appearance of a French Liberty cap worn at a rakish angle.

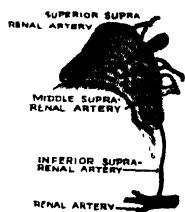


FIG. 750.—Right adrenal gland, viewed from in front.

The intimate anatomical relationship of the cortex to the medulla of an adrenal gland is the only good reason for considering the two portions of the gland as one and the same structure. Embryologically, histologically, pathologically and functionally, the adrenal cortex and the adrenal medulla are separate internal secretory glands.

The adrenal glands are supplied by several adrenal arteries (fig. 750), rendering them remarkably vascular, but only one vein drains each gland. On the right side the adrenal vein is short and enters the inferior vena cava just distal to the hepatic vein, while on the left it empties into the left renal vein (which communicates through the azygos vein with the left intercostal, internal mammary, and vertebral veins (Anson)). The dissimilarity of the right and left venous flow determines, to some extent, the location of metastases from malignant tumours of these glands.

Radiography of the Adrenal Glands

Delineation of an Adrenal Tumour.—Because of their protected position, very few adrenal tumours are palpable. In some advanced cases pyelography shows a deformity of the upper calyx of the corresponding kidney but this is sometimes impossible to distinguish from a renal neoplasm.

Paracoccygeal (fig. 751) injection of oxygen into the retroperitoneal tissues is free from danger and, combined with excretory pyelography, is helpful in defining the tumour. The clearest visualisation of the adrenal gland has been obtained by a combination of aortography and the retroperitoneal injection of oxygen.

Calcification in an adrenal gland is difficult to interpret in a radiograph. It is liable to be confused with a renal calculus. Areas of calcification in the adrenal glands may be present in Addison's disease (p. 578).

Thomas Hodgkin, 1798–1866. Curator of the Museum, Guy's Hospital, London.

Andrew Douglas Thomson, Contemporary. Director of Pathology, Royal Masonic Hospital, London.

Dorothy Reed (Mrs. Mendenhall), Contemporary. Formerly Fellow in Pathology, Johns Hopkins Hospital, Baltimore, U.S.A.

Henry J. Anson, Contemporary. Research Professor, Department of Otolaryngology and Maxillofacial Surgery, College of Medicine, University of Iowa, U.S.A.

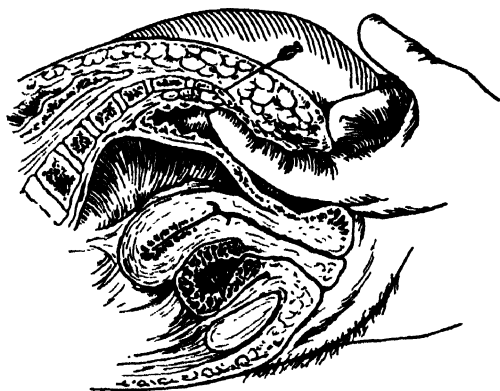


FIG. 751.—Method of introducing a lumbar puncture needle into the retroperitoneal space for insufflation of oxygen via a pneumothorax apparatus. Five hundred ml. of oxygen is injected. (After F. H. Rothfeld.)

and hypopotasæmia. **Aldosterone** is the most important of these 'salt regulating' hormones (see Conn's syndrome, p. 581).

2. **The cortisones** are concerned with the metabolism of proteins and carbohydrates, favouring the formation of the latter from the body's storehouse of the former. This conversion is known as gluconeogenesis. Consequently hormones belonging to this group are called glucocorticoids. The best known of these are **hydrocortisone** (also known as **cortisol**), and **cortisone** (which is converted in the body to hydrocortisone). The therapeutic application of these hormones falls into two headings:

(a) *In Endocrine Deficiencies*.—Cortisone is the logical need in adrenocortical insufficiency.

(b) *In Non-endocrine Disease*.—Cortisone is used in the treatment of a diversity of diseases, including allergic conditions, granulomatous disorders, blood diseases and the collagenoses. Used initially with apparent dramatic success in rheumatoid arthritis, the long-term results have led many to conclude that the results of cortisone therapy are no better than those obtained by aspirin. Hydrocortisone is an effective anti-allergic agent in a number of skin diseases and eye conditions.

3. **Sex Hormones**.—Both androgenic and oestrogenic hormones are produced by the adrenal cortex. Excessive secretion of androgens causes virilism in females, while on rare occasions excessive secretion of oestrogens brings about effeminacy in males.

Inter-hormonic Action.—The anterior lobe of the pituitary gland secretes adrenocorticotrophic hormone (ACTH) which stimulates the adrenal cortex, whereas the cortisol of the adrenal cortex inhibits the secretion of ACTH (fig. 752).

Tests of Adrenal Cortical Activity.—

(i) *Estimation of the 24-hour output of urinary steroids (cortisol metabolites)*.—The 17-ketogenic steroid and the 17-hydroxycorticosteroid excretions are similar—10–20 mg./24 hrs for men, 5–15 mg. for women, and 2–4 mg. for a child of six to eleven years. Adult levels are reached at about eighteen years. The average excretion is lower in coloured than in white males. After the age of forty, urinary steroid excretion gradually falls, and at seventy is approximately halved.

(ii) *Plasma cortisol level*.—Normally 8–28 micrograms/100 ml.

(iii) *ACTH stimulation test*.—An intramuscular injection of corticotropin gel (40 units twice daily)¹ stimulates the adrenal cortex, and so the urinary steroid levels are

THE ADRENAL CORTEX

The adrenal cortex is made up of the following layers from without inwards; the zona glomerulosa, the zona fasciculata, and the zona reticularis.

Physiology.—At least fifty steroid compounds have been isolated from the adrenal cortex. These hormones exhibit various types of activity which, for practical purposes, can be arranged in three groups.

1. **Salt Regulating Hormones** which are concerned in the maintenance of water and electrolytic balance. A deficiency of these hormones produces sodium diuresis, potassium retention and dehydration; an excess results in hypertension, oedema, cardiac dilatation,

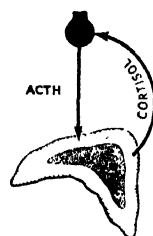


FIG. 752.—Pituitary ACTH stimulates the adrenals. Cortisol of the adrenals inhibits the output of ACTH.

¹ A single injection of 250 microgrammes of a synthetic corticotropin (Synacthen) may be used instead. Normally this will double the plasma cortisol level in the space of thirty minutes.

increased. Patients with adrenal hyperplasia are said to be very responsive and may give a level of 20–60 mg./24 hrs above the prestimulation control levels of the two urinary steroids mentioned in (i).

(iv) *Adrenal Suppression*.—Injections of cortisol or dexamethasone will suppress adrenal function (see Inter-hormonic Action and fig. 752, p. 576), as shown by the depression of the urinary steroids, especially in patients with adrenal hyperplasia. Adrenal tumours, however, are autonomous, so the excretion of urinary steroids in these cases is not suppressed.

It must be remembered that, as a rule, the result of each test alone is not a clear indication of the state of the adrenals, and therefore any conclusions must be drawn from the results of all the tests.

Disorders of adrenocortical function

The two most common disorders of adrenocortical function are chronic insufficiency (Addison's disease, p. 578) and chronic over-activity (Cushing's syndrome, p. 579).

Acute Hypocorticism:

1. *Adrenal Apoplexy in the Newborn*.—Extensive hæmorrhage into one (fig. 753) or both adrenals can be a cause of death in infants who succumb within the first few days of birth. The condition is prone to occur after long and difficult labour, and particularly when traumatic resuscitative procedures have to be employed to combat asphyxia neonatorum. The hæmorrhage into the adrenals follows necrosis of the innermost layer of the cortex, which always occurs at birth, possibly as a result of sudden withdrawal of the female sex hormone (œstrogen). Adrenal crises in the newborn produce signs of profound shock. A mass may be palpable in one or both renal regions. Intravenous fluid therapy with hydrocortisone, or, failing the latter, cortisone intramuscularly, offers the only hope of survival.

2. *Waterhouse-Friderichsen Syndrome*.—Massive bilateral adrenal cortical hæmorrhage, occurs mainly in cases of fulminating meningococcal septicæmia and in some cases streptococcal, staphylococcal, or pneumococcal septicæmia. The majority of cases occur in infants and young children, but it is not exceptional in adults. The onset is catastrophic, with rigors, hyperpyrexia, cyanosis, and vomiting. Petechial hæmorrhages into the skin which coalesce rapidly into purpuric blotches are a constant feature. Profound shock follows, and before long the patient passes into coma. The condition is one of overwhelming sepsis that pursues a galloping course, death occurring in most cases within forty-eight hours of the onset of symptoms unless correct treatment is given without delay.

Unilateral hæmorrhage causing a lesser degree of systemic upset and not associated with infections has been described. This type of case resembles a perinephric abscess or other upper abdominal acute condition.

Confirming the Diagnosis.—It is futile to await the result of a blood culture. Bilateral tenderness 2 in. (5 cm.) below the costal margin, and well lateral to the umbilicus, clear urine (oliguria is often present), and an absence of signs in the lungs help to call attention to the adrenal glands. In meningococcal infection the diplococcus can be demonstrated by smears obtained from a punctured petechial spot.

Treatment.—Antibiotic therapy must be given intensively by the intravenous route. In addition, an intravenous infusion of dextrose-saline solution containing hydrocortisone 100 to 200 mg. per litre is given. In the absence of hydrocortisone, cortisone can be given intramuscularly, but its action is slower and less certain. Oxygen should also be administered. Following such treatment, improvement often sets in within three hours, and a number of patients have recovered.

3. Crises of Infantile Hypercorticism (p. 579).



FIG. 753.—Kidney and adrenal of a newly born infant showing hæmorrhage into the adrenal gland.

4. **Following Bilateral Adrenalectomy.**—If precautions are taken (p. 581), acute hypocorticism is unusual in the post-operative period. In the months that follow, crises may occur if the correct dose of cortisone replacement is not given. The treatment is to support the blood pressure with D.O.C.A. (desoxycorticosterone acetate, 5 to 20 mg. intramuscularly), and then administer 3 litres of dextrose-saline solution per twenty-four hours, 50 to 200 mg. of hydrocortisone being added to each flask. Failing hydrocortisone, give cortisone intramuscularly.

Chronic Hypocorticism

Addison's disease is due to adreno-cortical insufficiency consequent upon progressive destruction of the zona reticularis, the zona fasciculata, the zona glomerulosa, and the medulla of the adrenal glands, in that order. Tuberculosis accounts for 50 per cent. of cases, the remainder being due to atrophy, amyloidosis, and other lesions that destroy the cortex. The medulla, which may or may not be implicated, appears to play no part in the clinical disturbance.



FIG. 754.—Addison's disease in a spinster aged twenty. Scattered pigmented areas are shown, especially in relation to a pressure point of her girdle. (Dr. Leonard Simpson, London.)

Clinical Features.—Addison's disease usually commences in the third or fourth decade. Sometimes it is the terminal event in cases of adrenogenital hyperplasia. The sex distribution is about equal. The leading features are muscular weakness and a low blood pressure. Irregular dusky pigmentation of the skin, due to deposits of melanin, appears at points of pressure (e.g. garter, belt) (fig. 754) and in the flexion creases. Pigmentation of mucous membranes, particularly of the mouth, is often striking. When fully established, the course of the disease is punctuated by crises of acute adrenocortical insufficiency (see above).

Confirmatory Diagnostic test.—The 24-hour urinary excretion of the 17-hydroxycorticosteroids by a patient with Addison's disease are usually less than 5 mg./24 hrs if the patient is a man and less than 1 mg./24 hrs in the case of a woman. The excretion fails to increase after ACTH administration (p. 576).

Especially during a crisis, the plasma sodium is decreased, the chlorides low, and the potassium elevated above 5 mEq./litre.

Treatment is medical, using cortisone, and relevant cases should include chemotherapy for tuberculosis.

Prognosis.—By the use of cortisone, the expectation of life of a patient suffering from Addison's disease has been extended from up to three years to at least seven years.

Hypercorticism

The various forms of adrenal cortical hyperfunction are classified according to the age of onset. (1) Infantile. (2) Pre-pubertal. (3) Adult, otherwise known as Cushing's syndrome—the commonest type. (4) Post-menopausal.

Primary aldosteronism (Conn's syndrome) can occur at any age (p. 581).

1. Infantile Hypercorticism.—Androgenic excess during intrauterine life is one form of pseudo-hermaphroditism in the *female child*. The condition is present at birth; sometimes the enlarged clitoris and a varying degree of hypospadias make it difficult to determine the infant's sex. The 17-ketosteroid content of the urine may be sufficiently elevated to substantiate a diagnosis of a female with adrenal hyperfunction. If this is not the case, it is justifiable to perform sex determination by a skin biopsy before the age of one year. Female pseudohermaphroditism with virilism is invariably associated with disease of the adrenal cortex, usually bilateral hyperplasia of the cortex. Hormonal studies have shown that there is a congenital failure of the adrenal glands to synthesise gluco-corticoids, mainly compound F. Due to this lack, these infants are liable to acute phases of adrenal insufficiency during stress or infection, or to suffer from periodic hypoglycaemic attacks. They need cortisone, not only in the emergency, but as long-term therapy, thereby inhibiting the secretion of excessive androgens. In the absence of such treatment the epiphyses join early, the patients are dwarfed, menstruation does not occur, and the breasts do not develop. These tendencies are corrected by cortisone, given orally, 25 mg. or more daily, the dose being determined by 17-ketosteroid estimations (Leonard Simpson). Hirsutism is moderated, but not necessarily abolished. The treatment should be commenced early if good results are to be obtained.

2. Pre-pubertal Hypercorticism.—There is never any doubt as to the sex of the infant at birth, and during the very early years of life the child is normal. The symptoms commence about the age of five or six years.

In the Female.—Pubic and axillary hair appear, but there is no gross enlargement of the clitoris. The child is short in stature, the legs being especially stunted, but she looks much older than she is. Puberty is often precocious, menstruation, if it occurs, being scanty. There is a deepening of the voice at this time.

In the Male.—The term 'infant Hercules' is descriptive. He is extremely short, muscular, and hirsute. The genitalia assume adult proportions, and spermatozoa are often present in the seminal fluid.

In both sexes, 17-ketosteroid content of the urine is increased. A very high reading supports the diagnosis of an adrenocortical tumour, which must always be excluded. In both males and females, with a later onset or the passage of time, the features of Cushing's syndrome become super-added.

Treatment.—This is identical with that of Cushing's syndrome.

3. Post-pubertal or Adult Hypercorticism (Cushing's syndrome) is due to an excessive production of glucocorticoids, mainly hydrocortisone (compound F). It is the commonest type of hypercorticism. In 35 per cent. of cases an adrenal neoplasm is present; in half of these the tumour is malignant. In 60 per cent. of cases the patient has bilateral adrenal hyperplasia; in one-third of these a basophilic tumour of the anterior pituitary gland—often microscopic in size—is present. In 5 per cent. of cases there is no discernible structural alteration in the adrenal glands, but in a small proportion of these there is a tumour of the pineal gland or the thymus.

In its most typical form, Cushing's syndrome is seen in patients treated with large doses of cortisone over long periods for non-endocrine diseases, particularly rheumatoid arthritis.

Clinical Features.—The female : male ratio is at least 3 : 1. The great majority of cases (excluding those induced by cortisone therapy) occur in females between fifteen and thirty years of age, in whom it produces highly characteristic features. Although the patient's weight is not necessarily increased, there is a deposition of fat in certain situations. The face becomes rubicund, rounded like a full moon, and the lips are pursed. The

abdomen becomes protuberant, the neck thick, the supraclavicular fossæ obliterated, and a roll of fat appears over the region of the vertebra prominens (buffalo hump). The arms, and especially the legs, are relatively thin, the muscular development is poor, and the patient complains of increasing weakness. As the disease progresses, so the general contour becomes more and more



FIG. 755.—Cushing's syndrome in a woman aged twenty-three years. Adrenal hyperplasia. (Dr. Leonard Simpson, London.)

that of a lemon on match-sticks—→ Consequent upon the inhibitory effect of the hypercorticism on fibrous tissue, the skin becomes of tissue-paper consistency, and inelastic. Exceedingly characteristic are purple-red striæ distentiæ, mostly on the abdomen (fig. 755), of a texture that can be likened to an over-stretched garter. Ecchymoses are frequent and bruising occurs on the slightest trauma. Acne is common, and there is a low resistance to skin infections. Often there is increased growth of lanugo hair, but hirsutism is usually absent. Amenorrhœa is usual or, in the male, impotence. Due to a negative calcium balance, the matrix of bone becomes thin, and severe osteoporosis results. Pathological fractures, particularly compression fracture of a vertebra, are common, and this is sometimes the first reason for the patient seeking advice.



Mild glycosuria is often present. Hypertension is frequent, and eventually congestive heart failure supervenes. In about 60 per cent. of cases various psychoses occur.

Cushing's syndrome is rare in children; when it occurs, the patient is nearly always a female and an adrenal tumour is usually the cause.

A sub-group, probably due to an excessive secretion of adrenal androgens (*adreno-genital syndrome*), commences between the ages of fifteen and twenty-five and is confined to females. One of the first indications of its onset is amenorrhœa or oligo-menorrhœa. There follows an excessive growth of hair on the face (fig. 756), acne, atrophy of the breasts, alteration in bodily contour and muscular development, deepening of the voice, and enlargement of the clitoris. Jewish and Spanish women are more prone to this affliction than those of other races.



FIG. 756.—Adreno-genital syndrome in a woman of twenty-eight. (Dr. Leonard Simpson, London.)

Arrhenoblastoma of the ovary.—This rare condition occurs between puberty and the menopause and also causes hirsutism. It may also arise in a suprarenal 'rest'.

Laboratory Findings.—Polycythæmia, lymphopænia, and eosinopænia are common. A fasting eosinophil count over 30 per c.mm. of blood is good evidence against the diagnosis. The basal metabolic rate is low and the serum cholesterol is elevated. The dextrose tolerance is impaired, and the insulin tolerance test reveals a resistance to the action of insulin. Urinary 17-ketosteroids are somewhat high, and urinary ketogenic steroids are above normal levels. There is a negative calcium balance and radiography of the skeleton reveals osteoporosis, most marked in the spine and pelvis.

Treatment.—In cases of cortical hyperplasia, total adrenalectomy on one side and resection of seven-eighths of the adrenal gland on the other side (usually after an interval) is the best course. Obviously the only treatment for neoplastic cases is excision of the adrenal gland bearing the tumour.

If a pituitary tumour causes oculomotor symptoms or enlargement of the pituitary fossa as seen on X-ray, it should be removed surgically (p. 392); in other circumstances any form of treatment for the pituitary is best avoided.

Very rarely, the *adreno-genital syndrome* appears in youths and men. Owing to excessive production of œstrogenic hormones by the adrenal cortex, gynæcomastia, atrophy of the testicles, and psychic signs of effeminacy appear (Adrenal Feminism).

4. **Post-menopausal hypercorticism** is usually characterised by the growth of a beard (the bearded woman of the circus), and is often accompanied by mental aberration. A lesser degree of hirsutes is almost a natural accompaniment of the aging process, particularly in dark-haired females, and it is difficult to draw the line between the normal and the pathological. Thus it is that operative treatment is usually disappointing.

Primary aldosteronism (Conn's syndrome) is due to a rare adreno-cortical tumour producing aldosterone (p. 576). Excess of this leads to sodium retention and a fall in serum potassium. The latter causes the typical features of the syndrome, namely episodes of muscular weakness associated with polyuria and polydipsia. The serum sodium is high and the potassium is low, but simple administration of potassium does not relieve the condition. Special, complex urinary tests demonstrate an increase of aldosterone. Effective treatment involves removal of the causative tumour, but there is the usual difficulty in ascertaining on which side it lies (see under operation, below).

ADRENALECTOMY FOR HYPERCORTICISM

It is essential that all patients who are to be subjected to extirpation of adrenal cortical tissue be prepared adequately for the operation, and supported post-operatively by adrenocortical hormone replacement therapy, irrespective of the extent of adrenal resection.

Cortisone Therapy

Pre-operative During the Operation and Immediate Post-operative Period	{	Cortisone acetate, 100 mg. intramuscularly twice a day for two days. One dose is given two hours before operation. Hydrocortisone, 100 mg. slowly by intravenous drip. Then a similar quantity during the subsequent twelve hours, to be followed by cortisone 50 mg. intramuscularly six-hourly. If the blood-pressure remains below 100 systolic, then blood-transfusion and/or noradrenaline is given (p. 583).
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Post-operative { **Day 1.**—Cortisone 50 mg. intramuscularly six-hourly.
Days 2 and 3.—Cortisone 50 mg. intramuscularly eight-hourly.
Days 4 and 5.—Cortisone 50 mg. intramuscularly every twelve hours.
Days 6 and 7.—Cortisone 25 mg. by mouth every eight hours.

Thenceforward cortisone by mouth should be reduced slowly to maintenance level in cases of total adrenalectomy, or to zero in subtotal adrenalectomy.

N.B.—If hydrocortisone is not available, 200 mg. of cortisone instead of 100 mg. should be administered intramuscularly twelve hours before operation. Cortisone is cheaper than hydrocortisone.

After total adrenalectomy the patient should always carry a card stating the dosage of cortisone she is receiving. Any stress (a further operation or infection) is an indication to increase the dosage.

Operation: (a) When an adrenal tumour has been demonstrated pre-operatively, excision of that adrenal gland alone is carried out. (b) If a tumour has not been demonstrated, the patient is prepared for bilateral exploration.

Because the difficulties are usually less on the left than on the right side, exploration of the left adrenal gland is undertaken first. If a tumour is found, adrenalectomy is carried out. If the left gland is found to be atrophic, it is highly probable that there is a tumour on the right side, which should be explored forthwith. Should the gland be hyperplastic or normal, subtotal (90 per cent.) adrenalectomy is indicated. If, after this has been performed, it is apparent that the patient will not tolerate a bilateral operation well, exploration of the contralateral side should be postponed.

Technique.—An ample postero-lateral incision, such as is used for exploration of the kidney (Chap. 45), is commonly employed. After subperiosteal resection of the twelfth rib the lower border of the pleura is defined and protected. The incision is extended through the bed of the twelfth rib to reveal the perinephric fat, within which the adrenal gland is identified, as described below. Sometimes an approach through the bed of the eleventh rib, reflecting the pleura upwards is preferred.

On the right side the suprarenal vein is short and may be torn from the vena cava if it is not identified and ligated at an early stage of the dissection. By finger and gauze dissection, keeping close to the gland, the gland is freed from below and behind, upwards, ligating and dividing bleeding vessels as they are encountered, until it is suspended only by its main vascular pedicle near its apex. If subtotal adrenalectomy is to be performed, the gland is cut across with scissors so as to leave a small triangular fragment of the apex well supplied with blood-vessels. Bleeding should be controlled by swab pressure, as diathermy coagulation leads to necrosis.

BILATERAL TOTAL ADRENALECTOMY FOR INOPERABLE CARCINOMA OF THE BREAST OR PROSTATE, OR THEIR METASTASES

In somewhat less than 50 per cent. of patients, total adrenalectomy results in a surprising degree of reduction of pain, a feeling of well-being, occasional radiographic evidence of recalcification of osseous metastases, and apparent resorption of metastases in other situations. In the case of carcinoma of the breast, the operation may be combined with removal of both ovaries (p. 612). Hormone therapy is employed before, during, and after adrenalectomy as described above. The majority of patients tolerate a single-stage bilateral adrenalectomy exceedingly well.

Operation.—The anterior route is to be preferred in these cases, and the adrenal glands are approached through a curved transverse incision. The left adrenal gland is approached first by cutting along the lateral leaf of the lieno-renal ligament and then curving downwards and medially, so as to enable a wide peritoneal flap to be reflected. By retracting the spleen downwards and medially, the

adrenal gland comes into view. The fascia over its lateral border is incised, and by gauze dissection the blood-vessels of the gland are defined, ligated, and divided, thus freeing the gland, which is removed. The right adrenal gland is more deeply situated. The peritoneum is incised lateral to the duodenum and above the upper pole of the kidney. The flap of peritoneum is raised to expose the anterior surface of the adrenal gland as it lies against the bare surface of the liver. The fascia covering the lateral surface of the gland is incised. A finger can then be inserted above the upper pole of the gland into the space between the two layers of fascia enclosing the gland (fig. 757). This prevents the gland from becoming displaced upwards, which otherwise it is prone to do. The anterior fascial layer is then incised transversely and the gland can be dissected under vision, as on the left side. After removal, each gland should be inspected to check its completeness, and each adrenal bed must be searched for the presence of accessory adrenal tissue, which is present in 32 per cent. of cases. If this important step is omitted, failure of the operation is not unlikely. The abdomen is closed with small drainage tubes passing to the adrenal fossæ on either side.

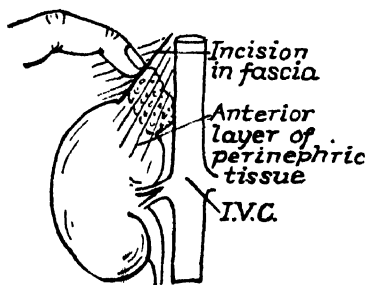


FIG. 757.—Incision in the fascia lateral to the right adrenal gland. Insertion of the finger into the space above the gland prevents its upward displacement. (After J. C. McKeown and A. Ganguli.)

Alternative Approach.—Some surgeons prefer to perform bilateral adrenalectomy for carcinoma of the breast through two incisions, resecting the twelfth or eleventh ribs.

THE ADRENAL MEDULLA

The medulla of the adrenal glands (chromaffin tissue), which is developed, together with sympathetic nerves, from ectoderm, is grey in colour and connected intimately, both anatomically and functionally, with splanchnic nerves. Chromaffin tissue is so-called because the large polyhedral cells of which it is composed contain granules that stain yellow with chromic acid. These granules are the internal secretion of the adrenal medulla itself, for they can be observed being extruded *in toto* into radicles of the adrenal vein. The secretion consists of adrenaline and nor-adrenaline. In health, 80 per cent. of the output is adrenaline, and 20 per cent. is noradrenaline. However, in hyperfunctioning medullary tumour (phæochromocytoma) this ratio is completely reversed. Fear, anger, pain, and effort give rise to an increased output in response to the stimuli received via the splanchnic nerves.

It would appear that one of the principal functions of chromaffin tissue is to liberate quickly hormones that permit a more rapid response of the body to noxious stimuli. Noradrenaline causes an over-all vasoconstriction, thereby raising the blood pressure. Its action is short-lived.

Noradrenaline drip.—The method of administration is to give very slowly indeed (30 drops per minute, gradually reduced to 7 drops) dextrose-saline solution intravenously,¹ to which has been added 4 ml. of 0.1 per cent noradrenaline to each 1,000-ml. flask of the solution, the aim being to adjust the drip to maintain the diastolic blood pressure between 70–80 mm. Hg. The disadvantage of noradrenaline is that it is of little value or even harmful in established shock. If effective at first, subsequent doses have diminishing effect. Alternatively, when effective, withdrawal is difficult without allowing the blood pressure to plummet down again.

TUMOURS OF THE ADRENAL MEDULLA

Neoplasms of the sympathetic neurones	$\left\{ \begin{array}{l} \text{Ganglioneuroma} \\ \text{Neuroblastoma (sympatheticoblastoma)} \end{array} \right.$
Neoplasm of chromaffin cells = Phæochromocytoma	

The saphenous vein should not be used, as the vasoconstriction may cause skin necrosis.

Those occurring at any age :

A **ganglioneuroma** is relatively benign. This neoplasm is symptomless, grows to a large size, and constitutes one of the varieties of retroperitoneal 'sarcoma' (Chap. 37). Only 15 per cent. involve the adrenal, the remainder occurring in any position along the sympathetic chain. If removed completely at a comparatively early stage, a cure can be expected.

Those occurring in infants and children :

Neuroblastoma of the adrenal medulla is a reddish-grey tumour that is highly malignant. It soon breaks its confines and invades neighbouring organs, e.g. the kidney and the pancreas, and metastasises by lymphatics, and even more frequently by the blood-stream.

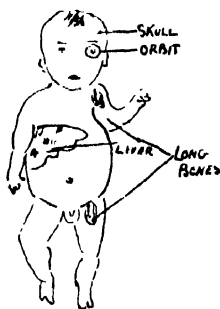


FIG. 758.—The common sites for metastases from neuroblastoma of the adrenal. Bones are involved more frequently than the liver.

Clinical Features.—Distributed equally between the sexes, 80 per cent. of these comparatively rare tumours occur below the age of five years. Usually the child is brought on account of an abdominal swelling. Pallor and loss of appetite are frequent accompaniments. The knobby contour of an adrenal neuroblastoma helps to differentiate it from a Wilms' tumour (Chap. 45), which remains smooth even after it has attained a great size. Although unilateral, the growth, as it enlarges, extends across the middle line. About 60 per cent. of patients have metastases by the time they are seen (fig. 758).

Pepper's type of the disease.—The primary and secondaries are on the right side, with large liver metastases. In *Hutchison's type*, a left-sided primary spreads upwards by lymphatics and deposits are found in the orbit and skull. Metastases in the skull mimic the spicular osteogenic sarcoma; those in the long bones resemble Ewing's tumours.

Investigations should include a complete radiographic examination of the skeleton.

Treatment.—Unless there are secondary deposits, exploration (before or after radiotherapy) should be undertaken; in comparatively early cases the tumour can be removed completely. When total removal is not feasible, as much as possible of the neoplasm should be excised, followed later by a course of deep radiotherapy.

If complete excision is possible, the prognosis is enhanced, but an extraordinary feature of these cases is that from time to time a patient survives when the tumour is found to be so advanced that only a piece is removed for section, and no treatment of any kind is given; sometimes even secondary deposits disappear. There is some evidence that cases which are histologically borderline between ganglioneuroma and neuroblastoma become converted to the former with high doses of *folic acid* with subsequent betterment of the prognosis. With *operation*, followed by *X-ray therapy*, about 25 per cent. of the patients recover, and if they are free from recurrence by the end of one year, it is almost certain that they are cured permanently (Gross).

Those occurring in adults (rare in children) :

Phæochromocytoma is a soft brownish benign tumour, usually less than 2 inches (5 cm.) in diameter (fig. 759), composed of large differentiated sympathetic ganglion cells, and a few fibres enclosed in a delicate capsule. It owes its name to the presence of chromaffin granules. In about 15 per cent. of cases the tumour is bilateral. This tumour, occurs in both sexes, usually during early adult life or middle age. It produces, either intermittently or continuously, an excess of adrenaline, and especially of noradrenaline:

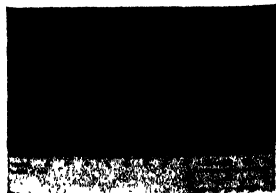


FIG. 759.—Phæochromocytoma excised successfully.

the ratio of the latter to the former often being as high as 20:1 causing *hypertension* which is either *paroxysmal* or *persistent*. The latter predominates statistically and probably indicates a late stage of the disease. Consequently all patients under sixty years of age who suffer from sustained arterial hypertension deserve routine tests to confirm or exclude a phæochromocytoma. While not more than 0.5 per cent. of cases of hypertension are caused by a phæochromocytoma, at the Mayo Clinic, where routine diagnostic procedures are undertaken to confirm or exclude the presence of this tumour in all cases of hypertension, the percentage has been stated to be nearly 3 per cent. Untreated, it progresses to a fatal termination.

Clinical Features.—The most common symptoms, in order of frequency, are: headache (55 per cent.), palpitation, vomiting, sweating, dyspnœa, weakness, pallor—i.e. the symptoms of adrenaline overdosage. A typical attack is associated with hypertension. When the blood pressure is persistently high the patients are liable to all the complications of hypertension and may present with them. Ten per cent. of the patients have diabetes mellitus.

Radiography.—Pyelographic examinations sometimes reveal displacement of the upper pole of the kidney. Other radiographic investigations are those described on p. 575, that is, retroperitoneal oxygen insufflation with an intravenous pyelogram and tomography. Aortography shows a vascular 'blush' at the site of the tumour. It also reveals the vascular state and size of the kidneys. If a 'blush' is not seen and one kidney is diseased, this is the likely explanation of the hypertension.

Laboratory Tests

1. *Colorimetric examination of the urine* for a metabolite of noradrenaline, vanillyl-mandelic acid, or fluorimetric determination of the catecholamines, can be relied upon to exclude the diagnosis of phæochromocytoma.

2. *Biological assay* (injection of an extract of the urine into an animal, usually a cat) determines accurately if there is a pressor substance excreted in the patient's urine.

3. *Direct Blood Examination.*—Another test is to pass a polythene tube into the inferior vena cava and test samples of blood for their concentration of noradrenaline.

4. The response to a carefully given diagnostic dose of Rogitine¹ is also confirmatory evidence of hypersecretion of chromaffin tissue.

Operation.—Twenty to 40 mg. (for an adult) of Rogitine (1 to 2 tablets) should be given after meals t.d.s., while the patient is being prepared for operation. The hazardous phases in the operation are: during the induction of anæsthesia, positioning of the patient on the operating table, when the tumour is manipulated, and immediately after removal of the tumour. Five mg. of Rogitine is given intravenously, prior to commencing the anæsthetic. With an intravenous drip infusion of dextrose-saline running, the operation is commenced. If pre-operative investigations have not revealed the side on which the tumour is situated, the procedure detailed on p. 582 is carried out. As some 10 per cent. of phæochromocytomas are ectopic there is something to be said for exploration via the anterior route (p. 582). The blood-pressure rises sharply when the affected adrenal gland is handled, and to counteract this rise another 5 mg. of Rogitine is given into the tube of the intravenous drip. Should it be necessary, yet another dose is given. As soon as the adrenal gland has been extirpated, a considerable fall in the blood pressure is to be expected, and is combated by giving an injection of noradrenaline (p. 583), which, during the first week following the operation, should always be at hand for immediate intravenous injection in case of need. With these precautions, the earlier operative mortality has been lowered. If symptoms persist after unilateral adrenalectomy, a tumour in the contralateral gland is highly probable. In this instance the second tumour, which is

¹ Rogitine (Ciba) (phentolamine B.P.), a substance that suppresses temporarily the pressor activity of the secretion of chromaffin tissue.

usually well-defined, must be dissected from the healthy portion of the gland. Some consider this to be the better technique for all cases, particularly because these tumours are almost always benign and the operation is curative.

The excised specimen, when fixed in bichromate solution, stains brown.

Hyperplasia of the adrenal medulla, although often more in evidence on one side than the other, is usually bilateral. Paroxysmal hypertension, clinically identical with that produced by a phæochromocytoma, is present. Unilateral adrenalectomy brings about amelioration, but for a cure of the condition, after an interval, the remaining adrenal should be removed.

CHAPTER 26

THE BREAST

Comparative and Surgical Anatomy.—Mammals are distinguished and so-called because they are provided with mammary glands. The cow, sheep, goat, mare and the elephant have an udder surmounted by teats, while other animals are furnished with breasts, the number of pairs of which vary with the species, and is related to the average number of offspring in each litter. Thus the sow has six to nine pairs, rodents six or seven pairs, while, like man, the anthropoid apes, the lioness, the sea-cow and, as John Hunter first noted, the whale, have but a single pair.

In anatomical works the protuberant part of the human breast is generally described as overlying the second to the sixth ribs, and extending from the lateral border of the sternum to the anterior axillary line. Actually a thin layer of mammary tissue extends considerably farther on all sides, viz: to the clavicle above, to the seventh or eighth ribs below, to the mid-line medially, and to the edge of the latissimus dorsi posteriorly. This fact is of importance to a surgeon when he seeks to remove the whole breast. The full extent of the breast is apparent in cases of milk engorgement.

As age advances, the parenchyma of the breast undergoes considerable atrophy and becomes loose in texture, making the detection of lumps within it easier.

The axillary tail of the breast is of considerable surgical importance. In some normal cases it is palpable, and in a few it can be seen in the pre-menstrual phase and during lactation. A well-developed axillary tail is sometimes mistaken for a mass of enlarged lymph nodes or a lipoma.

The lobule is the basic structural unit of the mammary gland (fig. 760). In the human breast the number and size of the lobules vary exceedingly: they are largest and most numerous during early womanhood. From ten to more than a hundred lobules empty by means of ductules into a lactiferous duct, of which there are from fifteen to twenty. Each lactiferous duct is provided with an ampulla—a little reservoir for milk or abnormal discharges.

The ligaments of Cooper are hollow conical projections of fibrous tissue filled with breast tissue, the apices of the cones being attached firmly to the deeper layers of the skin overlying the breast. These ligaments account for the dimpling of the skin overlying a scirrhus carcinoma, or other lesions of the breast accompanied by fibrosis.

The Areola.—The subcutaneous tissue contains involuntary muscle arranged in concentric rings as well as radially. The areolar epithelium contains numerous glands of three kinds—sweat glands, sebaceous glands, and accessory mammary glands. The sebaceous glands (known as the glands of Montgomery) enlarge strikingly during pregnancy and serve to lubricate the nipple during lactation. The accessory mammary glands are minute, inconstant, and possess ducts that open on the areola.

The Nipple is covered by a thick and rather crinkled skin. Near its apex, and very difficult to see because of the cutaneous corrugations, lie the orifices of the lactiferous ducts. The nipple and the areola of a nullipara are pink; with succeeding pregnancies they become pigmented by deposits of melanin. The nipple contains smooth muscle fibres arranged concentrically and longitudinally; thus it is an erectile structure and, for the convenience of the infant in arms, points forwards and outwards.

Lymphatics.—The lymphatic vessels of the breast drain into (1) nodes that lie between the greater and lesser pectoral muscles; (2) the thoracic chain of lymph nodes along the lateral thoracic artery; (3) the subscapular chain that extends from the lateral thoracic wall to the axillary vein, and thence along the axillary vein,



FIG. 760.—Showing the lobules and the lactiferous ducts, with their ampullæ.

most of the lymph nodes being on the caudal aspect of that vessel; (4) the central group, which is the largest, lying at the apex of the axilla. The highest of these lymph nodes is sheltered beneath the clavicle.

It is highly important to know that : (a) there is free communication between the subclavicular and the supraclavicular lymph nodes which are involved in 33 per cent. of cases in which the axillary lymph nodes are the seat of secondary deposits of carcinoma (Andreassen) ; (b) that the postero-cervical chain of lymph nodes are linked with those of the axilla, and that (c) the lymph nodes along the internal mammary artery are involved in 48 per cent. of cases in which the axillary lymph nodes are implicated (Handley).

THE NIPPLE

Absence of the nipple is very rare, and usually it is associated with amazia.

Supernumerary nipples occasionally occur along a line extending from the anterior fold of the axilla to the fold of the groin. This constitutes the milk line of lower mammalia. Unless the clinician is familiar with the anomaly, supernumerary nipples are liable to be mistaken for moles or warts.

Retraction of the nipple is of two exceedingly important varieties : (a) that occurring at puberty (remote), and (b) that occurring during womanhood (recent).

(a) **Retraction occurring at puberty** is due in most instances to a developmental abnormality ; the nipple, for some unknown reason, does not develop *pari passu* with the breast. It is possible that in a few instances the retraction is an aftermath of mastitis of infancy. In about one-quarter of the cases the condition is bilateral.

Non-protuberance of the nipple hinders an infant suckling at the breast. Clinical experience shows that a breast with long-standing retraction of its nipple is prone to fibroadenosis and, especially during lactation, to infection and abscess formation.

Treatment.—During and soon after puberty: if the patient draws out the nipple between finger and thumb daily for about three weeks the condition is usually remedied. In somewhat older patients, the regular application of a large warmed test-tube over the nipple may create sufficient suction to rectify the deformity.

(b) **Recent Retraction.**—The importance of long-standing retraction of the nipple is dwarfed by the ominous diagnostic significance of recent retraction (fig. 761), which is a frequent accompaniment of scirrhus carcinoma (p. 605).

Therefore the all-important question to put to the patient is : "How long has this nipple been retracted?"

Cracked Nipple.—Want of care in the preparation for lactation and neglect of the hygiene of the nipple during lactation are the chief causes of this not uncommon condition. Its main importance lies in the fact that the crack is the forerunner of acute infective mastitis.

Prophylaxis.—During the last two months of pregnancy the nipples and their areolæ should be

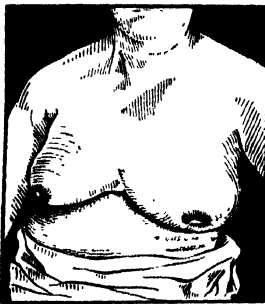


FIG. 761.—Recent retraction of the left nipple.

washed, dried, and anointed with a little lanoline each day. The same routine may be continued, with advantage, after suckling¹.

Treatment.—At the first sign of soreness the nipple is rested for twenty-four to forty-eight hours and the breast emptied with a breast-pump, as necessary. The nipple is washed and Bepanthen (5 per cent. Panthenol) Cream applied. When the soreness ceases, the baby is put to the breast for one minute at first and normal feeding is gradually resumed.

A cracked nipple is rested until healed; Tr. Benzoin Co. is applied locally. The breast is expressed and feeding gradually established as for a sore nipple. Especially if the nipple is partially retracted, the use of a nipple shield (fig. 762) will prevent recurrence.

Papilloma of the nipple presents the features of a cutaneous papilloma. Sometimes it grows to the size of a cherry, but the pedicle is always narrow. The treatment is excision together with a tiny disc of the skin from which it grows.



FIG. 762.—Rubber nipple shield.

Retention Cyst of a Gland of Montgomery.—These glands, situated in the areola, secrete sebum and, as a result of an orifice of one of the glands becoming blocked, a sebaceous cyst forms.

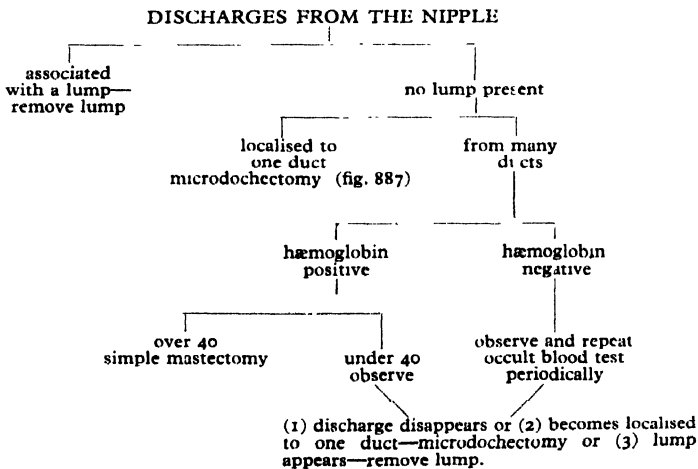
Chancre of the Nipple.—The majority of chancres of the nipple occur by infection from a syphilitic buccal mucous patch in the mouth of a member of the opposite sex. Although wet-nurses are now seldom, if ever, employed in this country, when this form of infant feeding is used the nipple is sometimes infected from the mouth of a syphilitic baby. The mother of such an infant is immune to re-infection from her own child.

Eczema of the nipple is a rare condition and is bilateral, and presents features common to eczema elsewhere.

Paget's disease of the nipple (p. 606) must be distinguished from the foregoing.

ABNORMAL DISCHARGES FROM THE NIPPLE

The discharge occurs from one (rarely more) of the lactiferous ducts.



Scheme for management of discharges from the nipple. ¹ (After Hedley Atkins, P.R.C.S., London.)

The management depends on the presence of a lump, or of occult blood (as tested by the benzidine test) or on whether it is localised to one duct. All such cases must be kept under regular observation.

¹ In some maternity hospitals the nipple is cleansed before and after suckling by wiping with a sponge removed from 70 per cent. alcohol solution kept in a jar labelled with the patient's name.

A **clear serous discharge** is associated usually with a retention cyst, consequent upon fibroadenosis.

A **blood-stained discharge** is pathognomonic of duct papilloma or duct carcinoma. The former condition is more common than the latter.

A **black or green discharge** may be due to altered blood from the foregoing, but is much more frequently an accompaniment of retention cysts of fibroadenosis. In cases of a dirty green discharge, when the breast is removed it is usually found to be riddled with cysts containing the same material.

THE BREAST

CONGENITAL ABNORMALITIES

Amazia.—Congenital absence of the breast may occur on one (fig. 763) or both sides. It is sometimes associated with an absence of the sternal portion of the pectoralis major. *Amazia*, which is rare, is more common in males.



FIG. 763.—Congenital absence of the right breast.



FIG. 764.—An accessory and functioning breast on the left thigh. (After G. J. A. Witkowski.)

Polymazia.—Accessory breasts have been recorded in the axilla, groin, buttock, and thigh, the most frequent site being the axilla. They have been known to function during lactation (fig. 764).

DIFFUSE HYPERTROPHY

Diffuse hypertrophy of the breasts occurs sporadically in otherwise healthy girls at puberty and, much less often, during the first pregnancy. The breasts attain enormous dimensions (fig. 765), and may reach below the knees when the patient is sitting. This tremendous overgrowth of the mammary glands is due, apparently, to their extreme sensitivity to oestrogenic hormone. Sometimes the hypertrophy is unilateral.¹ Unilateral hypertrophy may be easily mistaken for the breast which is pushed forward by a large soft fibroadenoma of the retromammary area. If diffuse hypertrophy causes real distress, a plastic operation is indicated.

PENDULOUS BREASTS

Flabby breasts that hang loosely and are frequently, but not necessarily over-size, is a common cosmetic mammary complaint. The cause of the condition is regression from a former fullness, due to (a) over-development at adolescence, (b) a terminated pregnancy, (c) lactation, or (d) obesity. For the more extravagant cases, plastic reconstruction of the breasts can be undertaken.

¹ In tropical countries this must be distinguished from filarial elephantiasis of the breast

UNDER-DEVELOPMENT OF THE BREASTS

In patients between the ages of twenty and thirty some increase in size can be expected following a two months' course of an œstrogen preparation.

INJURIES OF THE BREAST

Injuries of the breast are rare and comparatively unimportant.

Hæmatoma, particularly a resolving hæmatoma, gives rise to a lump which, in the absence of overlying bruising, is difficult, if not impossible, to diagnose correctly until an exploratory incision has been made.

Traumatic fat necrosis may be acute or chronic, and usually occurs in stout, middle-aged women. Following a blow, or even indirect violence (e.g. contraction of the pectoralis major), or the administration of subcutaneous infusion into the breast, a lump, often painless, appears. In the absence of a definite lead, the swelling, which is often attached to the skin, is usually diagnosed as a carcinoma. A definite history of injury, especially that inflicted by subcutaneous infusion, should bring the condition to the clinician's mind. On incising the lump a chalky white area of necrotic fat is found resembling necrosis seen in cases of subsiding acute pancreatitis.

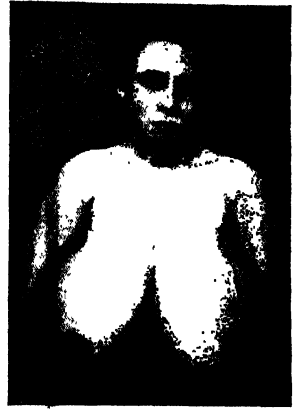


FIG. 765. — Diffuse hypertrophy (virginal).

ACUTE AND SUBACUTE INFLAMMATIONS OF THE BREAST

Mastitis of infants is at least as common in the male as in the female. Its ætiology is closely related to the lactation of infants. On the third or fourth day of life, if a breast of an infant is pressed lightly, a drop of colourless fluid can be expressed; a few days later there is often a slight milky secretion, which finally disappears during the third week. This is popularly known as 'witch's milk'. The explanation of this phenomenon is that the hormone which stimulates the mother's breast reacts also upon the mammary tissue of the foetus. Mastitis of infants is essentially a physiological activity. It may lead to a true mastitis by retrograde infection. This true mastitis usually resolves; occasionally it suppurates.

Mastitis of puberty is encountered rather frequently, usually in males. The patient, aged about fourteen, complains of pain and swelling in the breast. In 80 per cent. the condition is unilateral but the opposite breast may be affected later. The breast is enlarged, tender, and slightly indurated.

Treatment is symptomatic. Suppuration never occurs. The tenderness subsides in fourteen days or so, but induration often persists for several weeks.

Mastitis of mumps is usually unilateral, and more common in females.

Mastitis from local irritation, which is commonly subacute and seldom suppurates, may be produced from a too tight elastic brassière. The condition is by no means rare in men (p. 615).

Mastitis from milk engorgement is liable to occur about weaning time; and sometimes in the early days of lactation when one of the lactiferous ducts becomes blocked with epithelial debris. In the latter instance a sector only of the breast becomes indurated and tender.

Treatment.—Unless the engorgement can be relieved with a breast pump, or by the sucking of the infant (stagnant milk being such a good medium for bacterial growth), bacterial infection is liable to supervene. Antibiotic treatment as detailed below, if commenced early, will abort the infection in most instances. The advisability of weaning the infant and inhibiting secretion of milk (see later) should receive consideration.

Bacterial mastitis, which is by far the most common variety of mastitis, nearly always commences acutely. Although often referred to as mastitis of lactation, it is incorrect to assume that acute mastitis in women is necessarily lactational. Of a hundred consecutive cases of breast abscess, thirty-two occurred in women who were not lactating (de Jode); probably some were due to infection of a hæmatoma. In almost every case the infecting organism is a staphylococcus, and in a high percentage it is penicillin-resistant. In cases where the infection is acquired in hospital no less than 90 per cent. of the infecting staphylococci are insensitive to penicillin. There are two varieties of this condition:

Intramammary mastitis (80 per cent.) frequently progresses to suppuration. In lactational cases the sufferer is often in the *first* month of her *first* lactation, or, less frequently, is suckling an infant six or more months old, when its incisor teeth are developing.

Ætiology.—In maternity wards, staphylococcal infections of the breast sometimes assume almost epidemic proportions, due, no doubt, to a staphylococcal carrier among the nursing staff. Usually the intermediary is the infant; after the second day of life 50 per cent. of infants harbour staphylococci in the nasopharynx.

‘Cleansing the baby’s mouth’ with a swab is also an ætiological factor. The delicate buccal mucosa is excoriated by the process; it becomes infected, and organisms in the infant’s saliva are inoculated on to the mother’s nipple.

There seems little doubt that in the great majority of cases the precursor of intramammary mastitis is failure of secretion to escape because one (rarely more) of the lactiferous ducts becomes blocked with epithelial debris—a hypothesis that is strengthened by the fact that, whether they are lactating or not, intramammary mastitis and abscess of the breast are relatively frequent in women with a retracted nipple. While stasis in some part of the lactiferous tree is a major factor in the production of this condition, undoubtedly the older hypothesis—ascending infection from a sore or an infected cracked nipple—must not be spurned. Once within the ampulla of the duct, staphylococci cause clotting of milk. Within the clot organisms multiply rapidly.

Clinical Features.—The affected breast, or more usually mainly one quadrant of it, presents the classical signs of acute inflammation, and what is aptly called ‘the cellulitic stage’ of a breast abscess has been reached.

Treatment during the Cellulitic Stage.—The patient must be confined to bed. Tetracycline 250 mg. four times a day for seven days should be started immediately. Support to the breast with a firm many-tailed bandage

and local heat will help to relieve the pain, and permit examination of the inflamed breast daily, which is essential.

To wean or not to wean is a question of great importance. Except when the patient has been suckling the child for over nine months, undoubtedly it is better not to wean. Feeding is continued from the uninfected breast, and the infected breast is emptied after each feed by manual expression, carried out by the patient herself; this is more effective than a breast pump, which does not always clear obstructed ducts. The milk expressed from the inflamed breast is boiled for five minutes before being given to the child in a feeding bottle. In patients with bilateral acute mastitis the baby can be fed entirely on the milk obtained by expression and boiled; this is not detrimental to the baby.

In cases where the child has already been breast-fed too long, or the mother finds manual expression too painful, weaning is advisable. Stilbœstrol 10 mg. three times a day is given after the breasts have been emptied and this usually inhibits lactation completely.

Formation of an 'Antibioma'.—It is absolutely essential that an antibiotic should *not* be given if pus is already present. If an antibiotic is given, the pus in the abscess frequently becomes sterile and a large brawny œdematous swelling remains in the breast and takes many weeks to resolve. Sometimes there is excessive fibrosis and this, with the absence of tenderness, has caused the diagnosis to be revised in favour of a carcinoma. It is better to explore the mass with a wide-bore aspirating needle than to cause an 'antibioma' with its attendant pain, chronicity, and ill health. Most 'antibiomas' are due to (1) the patient stopping breast feeding with inadequate suppression of lactation and (2) late, inadequate, and ineffective antibiotics.

Indications for Operation.—The breast should be incised when, after emptying, an area of tense induration is felt and/or when œdema of the overlying skin is found. Usually the area of induration is sector-shaped, and in early cases about one-quarter of the breast is involved (fig. 766); in many later cases the area is more extensive (fig. 767).

Drainage of an Intramammary Abscess.—An incision following the cutaneo-alveolar margin (fig. 768) has a high cosmetic value and permits access to the whole of the interior of the affected segment. The incision passes through the skin and the superficial fascia. A long hæmostat is then inserted into the abscess cavity. Every part of the abscess is palpated against the point of the hæmostat, and its jaws are opened. All loculi and 'lactoceles' that can be felt are entered. Finally, the hæmostat having been withdrawn, a finger is introduced and any remaining septa are disrupted. Unless the abscess cavity is situated at the very highest sector of the breast a counter-incision should be made at the most dependent part of the breast and a drainage tube inserted. In this, almost more than any part in the body, *dependent drainage* is essential. Dressings are carried out aseptically on the second day, when the tube is removed, and subsequently every other day. Antibiotic therapy is given for at least one week.

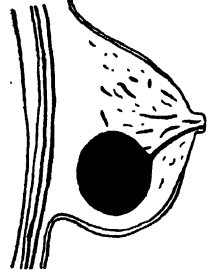


FIG. 766.—Intramammary breast abscess.



FIG. 767.—Intramammary breast abscess. The abscess should always be drained before it becomes subcutaneous. (T. A. Bouchier-Hayes, F.R.C.S.I., Dublin.)



FIG. 768.—Incision exactly at the areolar margin.

Subareolar mastitis (20 per cent.) is really not a true mastitis but results from an infected (sebaceous) gland of Montgomery, or from a furuncle on or near the areola. The inflammation develops insidiously, usually without constitutional symptoms. When the patient presents early, there is often an area of induration no larger than a pea. No matter how small, if a lump can be felt, pus is present (fig. 769), and the abscess should be drained without delay. Spontaneous rupture, if allowed to occur, does not cure the condition; it merely results in recrudescence or chronicity.

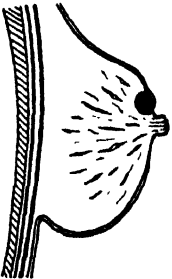


FIG. 769.—Subareolar abscess.

Chronic intramammary abscess is often a very difficult condition to diagnose: when encapsulated within a thick wall of fibrous tissue, the condition cannot be distinguished from carcinoma. Consequently it occasionally happens that the abscess is only discovered after the excised breast has been transected.



FIG. 771.—Slitting up a chronic subareolar abscess. In this case the patient has a retracted nipple of long standing. (After H. T. Caswell and A. W. E. Burnett.)

Chronic Subareolar Abscess (leading to a *milk-fistula*).—A recurrent subacute or a chronic abscess may occur apart from lactation in women of the child-bearing age. The condition is a frequent complication of long-standing retraction of the nipple. The abscess ruptures and subsides, only to repeat the cycle over and over again at intervals of a few months when it forms a chronic milk-fistula (fig. 770) which continues to discharge, and flares up when the fistula closes.



FIG. 770.—Milk-fistula originating in a chronic subareolar abscess.

Treatment.—Antibiotic therapy followed by incision and drainage is useless. The fistula must be treated in the same way as a fistula-in-ano, i.e. the track is laid open and saucerised.

Operation.—The only treatment that succeeds is to slit up the fistula (fig. 771) together with the communicating duct to its orifice on the nipple, which often lies in the sulcus of an inverted nipple. The next step is to excise thoroughly the fibrous walls of the abscess. This should be followed by packing and delayed closure.

Tuberculosis of the breast, which is comparatively rare among Western races but more common in some other parts of the world, is, as a rule, associated with active pulmonary tuberculosis, or secondary to tuberculous cervical adenitis¹. Tuberculosis of the breast (fig. 772) occurs more often in women who have borne one or more children and usually takes the form of multiple chronic abscesses. The diagnosis rests on bacteriological and microscopical examinations. Treatment with streptomycin, isoniazid and P.A.S. should be given (p. 23). If healing does not occur within two months, in most cases it is advisable to amputate the breast.

Actinomycosis of the breast is rarer still, except among peasant women who work in the fields of Europe. The lesions present the essential characteristics of facio-cervical actinomycosis (p. 531). If healing does not occur as a result of the systemic treatment of actinomycosis recommended on p. 532, the affected breast should be amputated.

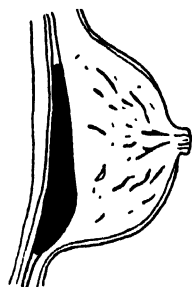


FIG. 773.—Retro-mammary abscess.

Syphilis of the Breast.—A primary chancre of the nipple has been referred to on p. 29. Secondary lesions of syphilis in the form of mucous patches are sometimes found in the submammary fold. In the second stage also both breasts may become swollen and painful, the condition being known as diffuse syphilitic mastitis.

Gumma of the breast is very rare, and almost impossible to diagnose unless there is other evidence of syphilis.

Retromammary Abscess (fig. 773).—Here the pus is situated in the cellular tissues behind the breast, and in the great majority of cases the abscess has no connection with the breast proper. Usually a retromammary abscess originates from a tuberculous rib, infected hæmatoma, or possibly from a chronic empyema necessitatis (p. 651), and treatment must be directed to the relief of these conditions. A submammary incision allows the breast to be retracted as necessary from the field of operation.

MONDOR'S DISEASE

Mondor's disease is thrombophlebitis of the superficial veins of the breast and anterior chest wall (fig. 774).

In the absence of injury or infection, the cause of the thrombophlebitis—like that of spontaneous thrombophlebitis in other sites—is obscure. The essential characteristic physical sign is an indurated subcutaneous thrombophlebitic cord about 3 mm. in diameter, of varying length, situated in the subcutis of the breast. Usually it is attached to the skin, and of a consistency that has been likened to that of the vas deferens. When the skin over the breast is stretched by



FIG. 772.—Tuberculosis of the breast with secondary suppurating axillary lymph nodes. (Professor A. K. Toufeeq, Lahore, Pakistan.)



FIG. 774.—Mondor's disease: a self-limiting thrombophlebitis of veins coursing over the upper chest towards the axilla. (Dr. J. H. Farrow, New York.)

¹ In 1829 Sir Astley Cooper, Surgeon to Guy's Hospital, London, described scrofulous swellings in the bosoms of young women, most of whom suffered from tuberculous cervical adenitis.

raising the arm, a narrow, shallow subcutaneous groove alongside the cord becomes apparent. The great importance of the condition is that those unfamiliar with Mondor's disease are likely to diagnose the condition as one of lymphatic permeation from an occult carcinoma of the breast. The only treatment required is restricted arm movements, and in any case subsidence occurs without recurrence, complications, or deformity.

FIBROADENOSIS¹

Ætiology.—Fibroadenosis is an aberration of those physiological changes that occur in mammary tissue at puberty (evolution) and at the menopause (involution). Usually it occurs between the ages of forty and fifty-five. Although it is called an 'aberration' it may be nothing more than the *normal* involutional change. Some women, however, worry about the lumps or the pain which it sometimes causes and thus it has become a disease 'entity'. Many women are affected without being aware of it.

Pathology.—When sectioned with a knife the affected areas in the breast are white or yellow, but they never present the grey tones of carcinoma. Microscopically the disease consists essentially of five features which vary in extent and degree in any one breast—these are:

(1) *Microcyst Formation*.—These cysts are often long standing and vary very much in size. They contain dark mucoid material.

(2) *Adenosis*.—As shown by an overall increase in acinar tissue.

(3) *Fibrosis*.—The interstitial tissues are swollen and there is round-celled infiltration. Fat and elastic tissue disappears and is replaced by dense white fibrous trabeculæ. This fibrous tissue compresses the ducts and leads to cyst formation.

(4) *Epitheliosis*.—Hyperplasia of epithelium in the lining of the acini may occur.

(5) *Papillomatosis*.—These may be small branching papillomas inside the cysts or small ducts.

After decades of discussion most surgeons and pathologists are now convinced that fibroadenosis as such, is not a precancerous condition. Others are emphatic that fibroadenosis *with epithelial hyperplasia* is definitely a precarcinomatous condition. Both fibroadenosis and carcinoma of the breast are common conditions; by coincidence, they can coexist, which in point of fact occurs in 3 per cent. of cases of fibroadenosis.

Clinical Features.—Fibroadenosis may occur at any age after puberty; it is particularly common about the time of the menopause. Spinsters, childless married women, and multiparous women who have not suckled their children are the usual sufferers. This suggests that the condition is prone to appear in breasts that have been denied their intended function. The patient usually complains of pain in *one* breast, worse before menstruation, or after using the arm. On examination both breasts are inclined to be what can be described as 'finely nodular'—the nodules being about the size of rice grains. As a rule it is possible to define the saucer-like edge of the periphery of the

¹ Fibroadenosis must not be confused with fibroadenoma, the most common benign tumour of the breast.

breast. When the breast complained of is examined between finger and thumb, more often than not, an indefinite lump can be made out, but with the flat of the hand this can be felt only vaguely. The lump is neither adherent to the pectoral fascia nor to the skin. There is no recent retraction of the nipple, but occasionally there is a serous or dark-green discharge therefrom. Fibroadenosis is sometimes more in evidence in one quadrant of the breast (fig. 775) than in the remainder. The presence of a palpable cyst or cysts is inclined to obscure the clinical findings of fibroadenosis *per se*. Frequently the axillary lymph nodes are slightly enlarged; they are not hard but are often tender.



FIG. 775.—Sector-shaped fibroadenosis. The patient, a spinster of twenty-eight, had a retracted nipple on the left side for many years.



FIG. 776.—A method of supporting the breast by adhesive strapping.

Treatment.—Reassurance is probably the most important part of treatment. It must be explained that these changes occur at this stage of life, that they are not precancerous and a certain amount of pain is to be expected. Œstrin will relieve the pain for a while but it will recur when it is withdrawn. Support for the breast by strapping (fig. 776) or a firm brassiere is essential. When the menopause is complete the pain will cease and the lumps disappear. From the surgeon's point of view it is important to keep the patient under review until it is quite sure that no carcinoma is present.

When the Diagnosis of Carcinoma is in Doubt.—There will always be cases where the clinician cannot be sure whether a particular lump in the breast is a patch of fibroadenosis or an early carcinoma. In doubtful cases it is wise to advocate operation without delay. The first step is to excise¹ that part of the breast containing the lump, and to look at its cut surface (fig. 777). In many cases a naked-eye inspection will suffice to indicate the nature of the mass. *If it is fibroadenosis*, nothing more than local removal of the affected area is indicated. *If it is carcinoma*, the incision is closed, gloves and instruments are changed, and the surgeon performs a radical mastectomy.

Sometimes, even by looking at the cut surface, no conclusion can be reached. If facilities for immediate histological diagnosis by frozen section are not available, the surgeon must either proceed to treat the case as one of carcinoma, or close the incision and await a histological report. The latter course may necessitate a second operation, that possibly may be refused.

¹ Many surgeons condemn pre-operative drill biopsy on the grounds that: (1) the procedure is open to the grave possibility of disseminating carcinoma; (2) the needle may miss a neoplastic area altogether; and (3) often it is difficult to give a positive diagnosis on the tiny specimen.



FIG. 777.—Macroscopic examination of a doubtful lump in the breast. In the lower part of the specimen the dead-white appearance of fibroadenosis is evident. In the upper quarter of this area there is a small carcinoma which cuts like an unripe pear, is greyish-white in colour and the surface of which is concave.

Indications for Surgery in Fibroadenosis.—Surgery should only be undertaken for severe and persisting anxiety about the lumps and even in extreme cases when the patient says the pain is intolerable the results are disappointing.

Submammary Excision of the Breast.

A satisfactory method of removing the mammary gland in these cases is through a submammary incision. The breast, including its axillary tail, is dissected out, leaving the skin and nipple intact. After the mammary gland has been excised, a purse-string suture on the under-surface of the areola ensures eversion of the nipple.

This operation (fig. 778), originated by Gaillard Thomas, gives a very good cosmetic result (fig. 779), especially in women with comparatively small, firm breasts.



FIG. 778.—Submammary excision of the breast completed.



FIG. 779.—Result of submammary excision of both breasts for fibroadenosis.

CLASSIFICATION OF CYSTIC SWELLINGS OF THE BREAST

Cysts of the breast occur either in the ducts or in the stroma.

Ducts —Fibroadenosis < Solitary.
Multiple.

Intracystic papilliferous carcinoma (p. 606).
Galactocele.

Stroma —Papillary Cystadenoma (p. 601).

Serocystic disease of Brodie (p. 601).

Colloid degeneration of carcinoma (p. 603).

Lymphatic cyst.

Hydatid.

(a) *Simple Solitary Mammary Cyst.*—The size of the cyst varies from $\frac{1}{4}$ inch to 2 inches (6 mm.—5 cm.) in diameter, the seat of election being the upper and outer quadrant. Macroscopically the solitary variety (fig. 780) presents as a blue-domed cyst (Bloodgood), the colour being due to its fluid content. Although, clinically, the condition is a solitary cyst, as a rule the breast parenchyma contains a number of tiny satellite cysts. That the

swelling is a cyst may sometimes be confirmed by transillumination; exceptionally aspiration is required to establish the diagnosis.

(b) *Multiple Cysts*.—This is the commonest type; the cysts are usually several in number. Sometimes one sector of the breast is affected alone. If a main duct near the nipple is obstructed, a duct papilloma should be suspected.

Treatment.—*To aspirate or not to aspirate? That is the question!*

Aspiration has its dangers as an intracystic neoplasm may be missed. For this reason many surgeons regard aspiration as unsafe. It is only safe if (1) the cyst does not refill, (2) the fluid withdrawn is not blood stained, (3) there is no residual lump after aspiration (Patey). If any of these criteria are not fulfilled, biopsy excision must be done. A solitary cyst can be excised through an incision over the swelling following one of Langer's lines (fig. 783). Multiple cysts are best dealt with by sub-mammary excision of the breast.



FIG. 780.—The blue-domed cyst of Bloodgood. (After C. F. Geschickter.)

Galactocoele, which is extremely rare, usually presents as a solitary, subareolar cyst, and always dates from lactation. It contains milk, liquid or inspissated, and in long-standing cases its walls tend to calcify.

Lymphatic cyst is a curiosity, and its pathology is similar to the more common lymphatic cyst of the neck (p. 521).

BENIGN NEOPLASMS OF THE BREAST

Epithelial	Duct papilloma.	
	Pure adenoma (very rare).	
Connective tissue	Neurofibroma.	
	Lipoma.	
Mixed-	-Fibroadenoma	
	-Papillary Cystadenoma.	
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Duct Papilloma.—The majority of these tumours are single, but bilateral examples are not rare, and occasionally two or more ducts of the same breast are the seat of a papillary growth. The usual single papilloma often has a stalk, and is situated in one of the larger lactiferous ducts.

Clinical Features.—The condition is rare before the age of twenty-five, and usually occurs in women between thirty-five and fifty. In the majority of cases bright red blood or, less often, a dark blood-stained discharge from the nipple is the only symptom. Rarely, the discharge is serous and not blood stained. On examination, a cystic swelling can sometimes be felt

beneath the areola; pressure upon it will cause a discharge from the mouth of the affected duct on the nipple. No greater tragedy is enacted than when this eminently curable condition is neglected. After months or years the duct papilloma may be transformed into a duct carcinoma.

Treatment.—Amputation of the breast is usually unnecessary. It is important not to express the blood before operation as it then may be difficult to identify the duct in the theatre.

Microdochectomy (after Hedley Atkins and Brigitte Wolff).—In this operation magnifying binocular glasses have proved most helpful. A fine sewing needle, from which the point has been removed, is inserted into the duct from which the discharge is emerging (fig. 781A). The skin of the nipple is then fixed to the eye of the needle by fine silk stitches. The skin of the incision is then infiltrated with adrenaline in saline (1 ml. of adrenaline in 1000 in 80 ml. of saline). Using a pair of fine pointed

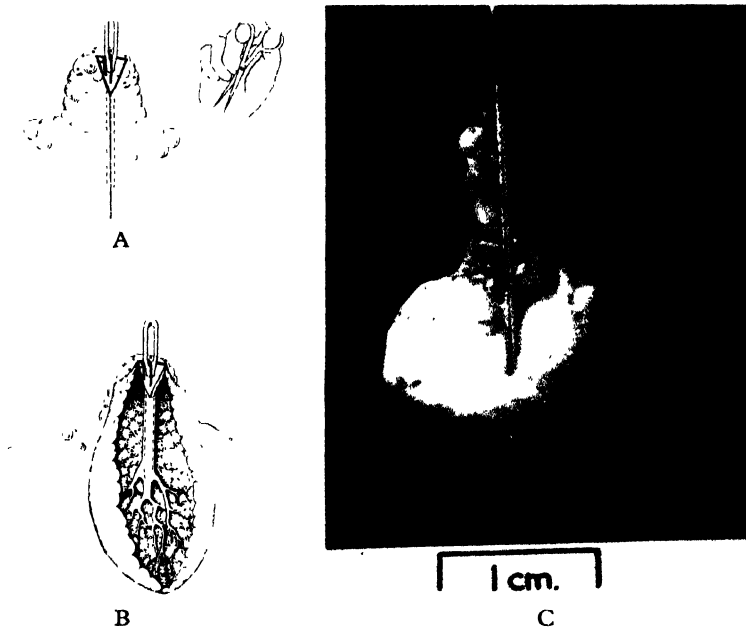


FIG. 781.—A. Blunt needle inserted into duct and stitched in position.
B. Reflection of skin flap to show needle inside duct.
C. Specimen removed intact.

(Atkins and Wolff. From *British Journal of Surgery*).

scissors, a triangular area is cut 1 mm. away from the eye of the needle (fig. 781A). Skin flaps are then reflected and the needle together with the duct is excised to produce a mass of breast tissue about 2.5 cm. in diameter. The lesion is nearly always situated within 4–5 cm. of the nipple orifice. The whole specimen (fig. 781C), together with the needle and triangular area of skin, is removed intact. After strict hæmostasis, the wound is closed without drainage. A firm pressure bandage is applied for 48 hours.

Nearly all excised specimens are reported upon by the pathologist as "Papilloma. No evidence of malignancy"—a major triumph resulting from a comparatively minor surgical procedure.

Fibroadenoma of the breast is divided clinically into two varieties, the hard and the soft. The consistency depends on the ratio of cells to stroma.

Hedley John Barnard Atkins, Contemporary. President, Royal College of Surgeons of England; Professor of Surgery, University of London (Guy's Hospital).
Brigitte Wolff, Contemporary. Pathologist, Guy's Hospital Medical School, London.

Histology.—Two types are usually described, pericanalicular and intracanalicular fibroadenoma. But in most tumours both arrangements are to be seen. A well-developed capsule is always present.

Hard Fibroadenoma.—As a rule the patient is between fourteen and thirty, the peak being about twenty-one years of age, and she complains of a lump in the breast. Exceptionally, there is more than one of these neoplasms present. On examination the lump is smooth, hard, and so freely movable that it has been termed a 'breast mouse'.

Treatment is enucleation of the tumour (fig. 782) through an incision which should be submammary or following one of Langer's lines (fig. 783); this is accomplished easily, because this neoplasm is entirely encapsulated.



FIG 782.—Fibroadenoma of the breast enucleated (actual size).



FIG. 783.—Langer's lines in the skin over the breast. (After C. D. Haagensen.)

Soft fibroadenoma, like the foregoing, is a definitely localised tumour. Much rarer than the hard variety, soft fibroadenomas occur in women about the age of thirty-five or forty. They are occasionally bilateral and usually lie deeply in the breast. Cystic and sarcomatous changes sometimes supervene.

Treatment.—As a soft fibroadenoma usually lies at some depth from the surface, it is best removed through an ipsilateral submammary incision (fig. 784).



FIG. 784.—Incision for removing a soft fibroadenoma in the medial half of the breast.

Papillary cystadenoma is a swelling composed of cysts in which papillomatosis is so extensive that the cysts are almost filled and feel solid. It may cause a bloodstained discharge from the nipple. The condition is entirely innocent and local excision is all that is necessary.

MASSIVE SWELLINGS OF THE BREAST

These are:

1. Diffuse hypertrophy, which is usually bilateral (p. 590).
2. *Soft fibroadenoma* of breast in young girls (about 15 to 25) may cause unilateral hypertrophy. This is usually retromammary, difficult to palpate, and can be removed by a submammary incision.
3. *Serocystic disease of Brodie* (*syn.* Cystosarcoma Phylloides)¹.—This may occur at any time from 40 years onwards. It is really a giant fibro-adenoma which rapidly attains such a large size that the overlying skin necroses and the

¹ Phylloides—from the Greek Φυλλωδης=leaf-like. There are branching projections of the tumour tissue into the cystic cavities of this neoplasm. It is not a sarcoma.

url von Langer 1819-1887. Professor of Anatomy, University of Vienna.
or Benjamin Brodie 1783-1862. Surgeon, St. George's Hospital, London.

tumour fungates through. It is not malignant even though the term cystosarcoma implies this. The surface of the tumour is unevenly bosselated (fig. 785), with areas of softening and even fluctuation in the larger convexities. The overlying skin is thin and tense, and large veins can be seen coursing beneath the integument. As a rule the tumour is adherent neither to the skin nor to deeper structures. On occasions there is a serous discharge from the nipple. The axillary lymph nodes are not enlarged, except secondarily to infection.



FIG. 785.—Serocystic disease of Brodie.

Treatment.—Simple mastectomy is all that is necessary.

MALIGNANT TUMOURS OF THE BREAST

Sarcoma of the breast is usually of the spindle-celled variety, and accounts for 0.5 per cent. of malignant tumours of the breast. Some of these growths arise in a soft fibroadenoma. It may be impossible to distinguish clinically a sarcoma of the breast from a medullary carcinoma, but areas of cystic degeneration suggest the probability of sarcoma. On incising the neoplasm its pale, friable consistency is characteristic. Sarcoma of the breast is met with most often in women between the ages of thirty and forty. Treatment is by simple mastectomy followed by radiotherapy. Surprisingly enough the outlook in this disease is good.

CARCINOMA OF THE BREAST¹

There is no known cause of mammary cancer, and although the patient frequently attributes the lump to a knock, it is extremely improbable that injury plays any part in the production of the condition. Cancer very rarely attacks both breasts simultaneously. Only 0.6 per cent. of all cases occur in males. Women between forty and fifty years of age are its most frequent victims, the disease having a higher incidence in the unmarried and in the less fertile married women. There is some evidence to support the view that suckling reduces the incidence of breast cancer. It is estimated that 1 in 25 of all female children born will develop cancer of the breast during their lifetime. While any portion of the breast may be attacked, the disease commences most frequently in the upper and outer quadrant (fig. 786). Unfortunately, so often the patient states that, although she noticed a lump in her breast while washing herself, she "took no notice of it" because it was painless. Probably the average time between the patient finding the lump and reporting it is eight months. Women should be urged to report to their doctors as soon as a lump in the breast is discovered.

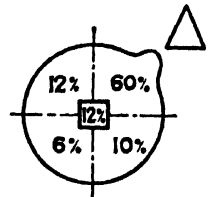


FIG. 786.—The relationship of carcinoma of the breast to the quadrants of the breast. (Marshall and Higginbotham's statistics.)

Pathological Classification.—All breast cancers arise in duct epithelium.² Some are so malignant and rapidly dividing that they transgress barriers readily and are

¹ The importance of this subject becomes more evident when it is realised that 7,500 women die annually from this condition in Great Britain. This is the highest incidence of any country in the world, except Austria and Denmark.

² Hedley Atkins, P.R.C.S.

almost everywhere seen to be extradochal. Such cells are polygonal (spheroidal) from mutual compression. Others are less malignant and retain their characteristic columnar form. Such growths have less tendency to transgress barriers (though they must do somewhere otherwise they would not be carcinomas) and are seen to be mainly intradochal. The extradochal growths excite a greater or less degree of fibrous reaction. A prominent feature of the histology is that in any one tumour there is usually an infinite variation in the pattern of the cancer cells, from the atrophic scirrhous with much fibrous tissue to the most cellular with little or none. Furthermore, while the breast lump may be an obvious scirrhous, the deposits in the lymph nodes may be highly cellular. It is possible, however, to distinguish those arising in the *nipple* (Paget's disease, p. 606), those arising in the *ducts* which are frequently papilliferous in form, and those arising in the *breast tissue* itself which are composed mainly of spheroidal cells with varying degrees of fibrous tissue response. A distinct type is the so-called *colloid* carcinoma where all the neoplastic cells are distended with colloid material. This tends to form a large growth but it is not highly malignant as might be expected.

From all this, it will be obvious that a distinct microscopical classification of any growth is very difficult as it may be scirrhous, trabecular scirrhous, medullary, or tubular. With such a wide variety of pattern in any one tumour it is impossible to give a prognosis on the histology alone.

THE SPREAD OF MAMMARY CARCINOMA

(a) **Local Spread.**—The tumour increases in size and invades other portions of the breast. It tends to involve the skin (fig. 789) and to penetrate the pectoral muscles, and even the chest wall.

(b) **Lymphatic spread** occurs in two ways: *by emboli*, composed of carcinoma cells, being swept along the lymphatic vessels by the lymph stream; and *by permeation*, that is, actual growth of columns of cancer cells along the lumina of the lymphatic channels. By these means the axillary lymph nodes and sometimes the internal mammary lymph nodes are involved comparatively early. Later, the supraclavicular lymph nodes, the opposite breast, and the mediastinum, are all possible resting places for itinerant carcinoma cells journeying by this route. Finally, they may be found in lymph nodes even farther afield.

(c) **Spread by the Blood-stream.**—It is by this route that skeletal metastases occur (in order of frequency) in the ribs, lumbar vertebræ, femur, thoracic vertebræ, and the skull, a pathological fracture occurring most often in a rib or a vertebra. In most instances it is by way of the blood-stream that metastases arrive in the liver or brain from the breast, but secondary deposits may also be carried to the liver via the lymphatics within the rectus sheath and the falciform ligament.

Clinical Types of Carcinoma of the Breast.—Again it is difficult to give any one growth a distinct classification but, in the main, it is possible to recognise certain clinical types. Commencing with the most malignant and (assuming that a diagnosis is made reasonably early) passing to the least malignant, the following clinical varieties are encountered:

Mastitis Carcinomatosa (2 per cent.).—Most of these cases occur during pregnancy or lactation. The diseased breast is often painful—an uncommon symptom in ordinary carcinoma of the breast. In 50 per cent. of cases there is retraction of the nipple. The reddened skin (fig. 787) feels abnormally warm and cutaneous oedema, which indicates blockage of the subdermal lymphatics with carcinoma cells, usually extends over a considerable area, i.e. over one-third or more of the breast. Enlargement of the axillary lymph nodes is present regularly. This galloping form of mam-



FIG. 787.—Mastitis carcinomatosa of the left breast.

mary cancer, which is the result of an unbridled proliferation of cancer cells with very little fibrous reaction, sometimes is mistaken for an abscess, and incised. The main distinguishing features between the two conditions are that in mastitis carcinomatosa there is (1) more widespread œdema, (2) (with few exceptions) an absence of pyrexia and leucocytosis, and (3) the 'inflammation' fails to respond to antibiotic therapy.

Treatment.—The widespread teaching that operation—radical mastectomy in particular—is always contra-indicated in cases of inflammatory carcinoma, is untenable. The only patients with this condition who have survived five years—and they are few enough in all conscience—have been subjected to radical mastectomy followed by radiotherapy. In many instances palliative treatment is all that is possible, and usually the patient is dead within a few months. Whether the pregnancy should be terminated is a great problem and each case has to be considered on its merits. Proof that benefit occurs is difficult to come by. If the pregnancy is terminated, oöphorectomy should be carried out as well.

Medullary (syn. anaplastic) carcinoma (17 per cent.) is usually found in women between twenty-five and thirty-five years of age with well-developed breasts (fig. 788). While presenting many characteristics of the scirrhus



FIG. 788.—Medullary carcinoma of the left breast. The nipple on the affected side is elevated. The breast contains an irregular firm mass. The axillary lymph nodes are impalpable.



FIG. 789.—Medullary carcinoma fungating through the skin. Note the *peau d'orange*.

variety, the epithelial element of the growth is in excess of its fibrous stroma. As a consequence the tumour does not feel so hard, and in advanced cases (fig. 789) it may present semi-solid areas due to degeneration of masses of ill-nourished cells. This variety of carcinoma is not so intensely malignant as one was often led to believe. Statistics from all clinics show that the

survival rate after operation is very little worse than that of scirrhus carcinoma. Perhaps the relatively large size and rapid growth of the tumour impel the patient to seek relief earlier than in the case of scirrhus carcinoma.

Scirrhus¹ carcinoma (63 per cent.) is met with principally in middle-aged and elderly women. Owing to an abundance of fibrous tissue the lump feels very hard, while its contour tends to be irregular. The most important physical sign in breast cancer is attachment of the lump to the skin, underlying muscle, or to the nipple (fig. 790). The clinician must spare no pains to be certain whether the lump he is investigating has any such attachment (fig. 791). The importance of recent retraction of the nipple has been alluded to already (p. 588). *Peau d'orange* of the skin overlying the tumour (fig. 795) is a late sign. In the early stages the axillary lymph nodes are impalpable, but they are invaded microscopically a considerable time before they can be felt clinically.

Untreated, the growth invades and eventually ulcerates through the skin and it may extend into the thorax (fig. 792), while dissemination via the lymphatic and blood-streams finally determines the fatal issue.

If a breast containing a scirrhus carcinoma is cut with a knife so as to section the tumour, the following macroscopical characteristics will be noticed :

1. The growth cuts like an unripe pear, and may grate whilst being cut.
2. Usually *both* cut surfaces are found to be concave.
3. The colour of the cut surface is definitely grey (fig. 777), and its appearance



FIG. 790.—Scirrhus carcinoma of the left breast ; Stage I. Nipple commencing to retract. (For staging of carcinoma of the breast, see p. 607.)



FIG. 791.— Scirrhus carcinoma ; Stage III. Nipple almost out of sight.




FIG. 792.—Stage IV. Nipple submerged, skin involved by the growth which is adherent to chest wall.

¹ Scirrhus—from the Greek *skiros* = hard.

has been aptly likened to the interior of an unripe pear. A most important gross finding is small granular whitish streaks within the tumour tissue.

4. On viewing the periphery of the sectioned tumour it will be found that there is not the slightest indication of a capsule. True to its namesake, the crab, its claws have penetrated hither and thither into the breast tissue, and it is impossible to separate the tumour from the breast.

Atrophic scirrhus carcinoma (5 per cent.) is seen principally in aged, thin women with small breasts. The cellular element of the growth is comparatively sparse, its main constituent being the fibrous stroma. Although steadily progressive, the disease runs a very chronic course, perhaps taking ten years to ulcerate through the skin, after which it is inclined to grow somewhat more rapidly. Stilboestrol (p. 613) may cause it to disappear.

Duct Carcinoma (8 per cent.).—As the leading symptom in both conditions is a blood-stained discharge from the nipple, it is often impossible to distinguish a duct papilloma from a duct carcinoma without additional help. In the case of a duct carcinoma sometimes a small lump can be palpated behind the nipple or areola, and emerging from this there is usually a sector-shaped area of induration, viz. : —————→ 

Obviously the cause of the latter is distension of those alveoli drained by the duct that is blocked by the growth.

Exploration in a Doubtful Case—On slitting up a lactiferous duct containing a duct carcinoma it is often possible to trace the main stalk of the neoplasm arising from the wall of the duct. From this stalk delicate tendrils, like fine seaweed, can be traced along smaller ducts, the latter and their corresponding alveoli being dilated into a fine sponge-work by the growth and blood-stained serous exudate.

The reason for the relative benignity of this form of carcinoma is that the lymph nodes are involved somewhat late in the course of the disease, and as a duct carcinoma gives rise to an alarming symptom (bloody discharge from the nipple), which impels the patient to seek advice early, the prognosis after radical mastectomy is usually good.



FIG. 793.—Intracystic papilliferous carcinoma. The breast is also the seat of fibroadenosis with multiple small cysts.

Intracystic Papilliferous Carcinoma (2 per cent.).—It is usually impossible to differentiate this condition from a simple cyst until its interior has been displayed or aspiration has revealed blood-stained fluid or a lump remains afterwards (p. 599). Rapid recurrence of the cyst after aspiration is also suspicious. Within the cyst there is a cauliflower-like growth (fig. 793). The prognosis after early operation is good.



FIG. 794.—Paget's disease of the nipple.

Paget's disease of the nipple (1 per cent.) is a persistent eczema-like condition that usually commences in patients over fifty years of age and does not respond to treatment. The nipple is eroded slowly and eventually disappears. As the disease progresses the areola becomes involved (fig. 794), and the erosion continues to spread peripherally for about two years, by which time a carcinoma within the breast becomes manifest. After many

years of controversy it is now agreed that Paget's disease of the nipple is due to a slowly growing duct carcinoma that infiltrates the epithelial covering of the nipple.

Microscopically the affected area is characterised by the presence of large vacuolated cells, with small, deeply staining nuclei, in the epidermis. In the majority of cases an exhaustive microscopic search reveals the presence of malignant changes in the ducts (Donald Teare). Sooner or later a carcinoma develops in the breast itself.

Differential Diagnosis:

Eczema	Paget's Disease
Bilateral.	Always unilateral.
Lactation.	Menopause.
Vesicles.	None.
Responds to treatment.	Does not respond.

Treatment should always be radical mastectomy. As a rule the prognosis is extremely good. Naturally, if the patient has a palpable lump as well as nipple erosion, the prognosis is less favourable. On no account should patients with this condition be subjected to radiotherapy alone, as Paget's disease of the nipple is radio-resistant.

CLINICAL STAGING OF CARCINOMA OF THE BREAST

When the patient is first examined, instead of categorising the growth in such vague terms as 'early', 'moderately advanced', or 'advanced', it is highly desirable to have some conventional method of expressing, in an explicit manner, the stage which has been reached. There is no better method than the Manchester¹ classification:

Stage I.—Growth confined to the breast. (a) An area of adherence to the skin smaller than the periphery of the tumour does not affect staging. Even ulceration of the skin in the area defined does not alter it.

Stage II.—(a) Same as Stage I but there are (b) affected *mobile* lymph nodes in axilla.

Stage III.—(a) Skin involvement larger than tumour and/or (b) lymph nodes are fixed and/or (c) tumour fixed to pectoral muscle and/or (d) affected lymph nodes in supraclavicular fossa.

Stage IV.—The same as Stage III plus distant metastases either blood borne or lymph borne; this includes metastases to opposite breast, axilla, supraclavicular fossa, or satellite skin nodules.

When Stage II has been passed, the prognosis deteriorates considerably.

The International Union against cancer has recommended a staging system known as T.N.M. (Tumour, Nodes, Metastases) (pp. 52 and 53). This system is becoming more and more accepted internationally. It is complicated and takes time but it is more precise.

PHENOMENA RESULTING FROM LYMPHATIC OBSTRUCTION IN CASES OF MAMMARY CARCINOMA

Peau d'orange (fig. 795) is due to cutaneous lymphatic œdema. Where the infiltrated skin is tethered by the sweat ducts it cannot swell. The characteristic pitted appearance, so well likened to orange peel by French observers, has become a classical physical sign of advanced carcinoma of the breast. But it should be

¹ Evolved by the staff of the Christie Hospital, Manchester.



FIG. 795.—Early *peau d'orange* is made more obvious by pinching up the skin.

remembered that occasionally the same phenomenon is seen over an abscess, particularly a chronic abscess, of the breast.

Early Post-operative Oedema of the Arm.—Oedema may occur within a matter of days after radical mastectomy. Its cause is uncertain, but it is wise to regard it as infective in origin and treat with a full course of antibiotics.

Late oedema of the arm (*syn. elephantiasis chirurgens*) is a frequent and troublesome complication of radical mastectomy. The swelling (fig. 796) appears at a time varying from several months to many years after operation. Obese patients are specially susceptible to it. It may be associated with a local infection of the arm or hand and if any such is demonstrable, it must be vigorously treated with antibiotics. It is certainly more common when a radical mastectomy is followed by deep X-ray therapy. The only treatment of value is elevation with the use of an elastic armlet from the wrist to the axilla. Massage and exercises may give considerable benefit. In obese patients drastic reduction in weight is helpful. Various operations to enhance lymphatic drainage have been advocated, but all fail in their purpose.

Brawny arm can result from advanced neoplastic infiltration of unremoved or incompletely removed axillary or supraclavicular lymph nodes. The oedema, which is persistent and brawny (it does not pit), is due to lymphatic blockage, but in some cases venous obstruction is a contributory cause.



FIG. 797.—Cancer *en cuirasse* in a patient with untreated carcinoma of the breast.



FIG. 796.—Late oedema of the arm following radical mastectomy three years previously.

A forequarter amputation is merciful in selected cases, especially in order to relieve the intense pain caused by involvement of nerves in the axilla.

Cancer en Cuirasse.—Here, in addition to a brawny arm, the affected side of the thoracic wall is studded with carcinomatous nodules and the skin is so infiltrated that it has been likened to a coat of armour (fig. 797). Usually, but not necessarily, the condition appears in cases with local recurrence after amputation of the breast. Various palliative measures can be tried, but the main objective is to relieve pain and mental distress for the few remaining months of the patient's life, e.g. Mist. Euphoria (p. 61) is strongly indicated.

Lymphangiosarcoma has been recognised recently as a complication of post-

mastectomy lymphoedema. Unless there is awareness of the condition, it is likely to be confused with recurrent carcinoma of the breast.

If considered humane and advisable, deep X-ray therapy may postpone the inevitably fatal course of this complication. The only alternative is to perform an interscapulo-thoracic (forequarter) amputation.

Serous effusion into the peritoneal or pleural cavities may be one of the terminal events in hopeless cases. The use of aspiration and replacement with a cytotoxin can alleviate symptoms in many of these patients.

BIOPSY FOR THE CONFIRMATION OF THE DIAGNOSIS OF CARCINOMA OF THE BREAST

In many cases (estimated at 25 per cent.) even an experienced clinician cannot be certain of the diagnosis of a lump in the breast. Biopsy, therefore,

assumes a position of importance unrivalled in any other part of the body. The possible dangers of drill biopsy have been alluded to already (p. 597, footnote).

Open biopsy is undertaken in the operating theatre, with the patient prepared for mastectomy. After the specimen has been obtained, the operative area is redraped, gloves and instruments are changed in readiness for mastectomy should the pathologist report that the section reveals carcinoma.

Excision is Safer than Incision.—Neoplastic tissue should never knowingly be incised *in vivo*. If the lesion is not small enough and superficial enough to be excised with a margin of apparently healthy tissue through an incision following one of Langer's lines (fig. 783), simple mastectomy will provide the best biopsy material. Such a step is especially justified in older women when the clinical diagnosis weighs in favour of carcinoma. Otherwise excision of the lesion through a submammary incision is advisable.

Macroscopical Examination of the Lump (fig. 777).—On bisection of the specimen, in nine out of ten cases it will be apparent to an experienced surgeon whether the lump is definitely malignant or not.

Frozen Section.—When a pathologist especially skilled in interpreting frozen sections microscopically is in attendance in the operating theatre he is able to give an opinion as to whether or not the growth is carcinomatous within a matter of minutes. In some cases, of course, he will be doubtful. When the services of a pathologist with special experience in this work are not available, or when he cannot express a definite opinion, it may be considered to the patient's advantage to close the wound and await the result of an examination of paraffin sections, which can be ready in forty-eight hours.

METHODS OF TREATMENT OF CARCINOMA OF THE BREAST

At the present time most authorities consider that radical mastectomy or extended radical mastectomy offers the best prospect of long survival, provided the neoplasm can be categorised as belonging to Stages I or II. When Stages III and IV are reached, the interests of the patient are best served by simple mastectomy, radiotherapy and, provided the tumour is hormone stimulated, endocrine therapy.

Radical Mastectomy.—The breast and associated structures are dissected *en bloc*, and the excised mass is composed of:

1. The whole breast.
2. A large portion of skin, the centre of which overlies the tumour, but always includes the nipple (fig. 798). When there is much skin involvement more skin must be sacrificed.
3. The fat and fascia from the lower border of the clavicle to, and including, the upper quarter of the sheath of the rectus abdominis, and from the sternum to the anterior border of the latissimus dorsi.
4. The pectoralis major and its fascial sheath (usually its clavicular head left).
5. The pectoralis minor and its fascial sheath.
6. The costocoracoid membrane.
7. All the fat, fascia, and lymph nodes of the axilla.



FIG. 798.—Incision for radical mastectomy. By temporarily retaining the portion of skin shaded in the inset, sufficient is left for providing skin for an immediate full-thickness graft, if required. (After R. H. Gardiner and A. D. Roy.)

8. The fascia over, and a few of the superficial muscle fibres of, the anterior part of the external oblique, serratus anterior, the subscapularis, the exposed portion of the latissimus dorsi, and the upper part of the rectus abdominis (fig. 799).

During the operation every effort should be made to preserve:

1. The axillary vein.
2. The cephalic vein.
3. The long thoracic nerve of Bell (nerve to serratus anterior).

The middle or long subscapular nerve (nerve to latissimus dorsi) can be sacrificed without ill effect, and this should be done without hesitation if its division enables a more thorough dissection to be carried out.

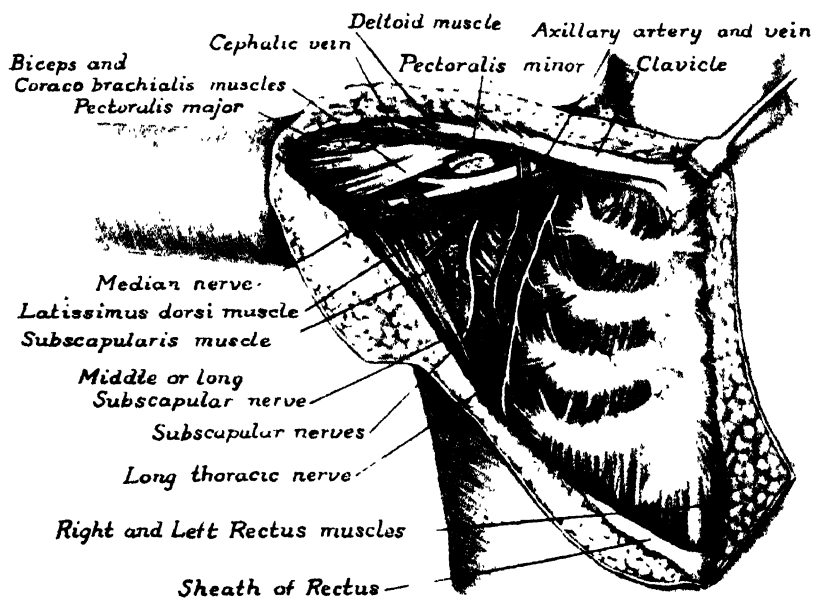


FIG. 799.—Radical mastectomy completed. It is desirable, but not essential, to preserve the nerves proceeding in a downward direction.

During the operation the exposed chest wall must be protected by towels wrung out in hot saline. At the completion of the operation the wound is drained.

If a wide area of skin has been sacrificed, it may not be possible to approximate the skin edges completely. A deficiency is left which is treated by immediate or subsequent skin grafting.

After operation the arm is supported upon a pillow until the wound has healed, when early movement of the arm is encouraged. The movements of the arm after so extensive a loss of muscle are surprisingly good.

Extended Radical Mastectomy.—As about 50 per cent. of cases of carcinoma in the medial quadrants and 25 per cent. in the lateral quadrants of the breast show metastases in the internal mammary lymphatic chain, an increasing number of surgeons feel that the internal mammary chain should be included in a radical mastectomy. A few incise the muscles of the second intercostal space and remove a node for frozen section; should it be involved, they regard the following step as not worth while. The second, third and fourth costal cartilages are divided with rib shears close to the sternum, and the intercostal muscles are severed vertically. A short lateral incision is made in the second and fourth intercostal spaces and the ribs are again divided half an inch (1.3 cm.) more laterally. The lower end of the trap-door thus fashioned is raised, and the internal mammary vessels are ligated as low as possible. The vessels, together with the lymphatics and adjacent tissue, are dissected upwards and are again ligated as high as possible in the second space. Some

Sir Charles Bell, 1774–1842. Surgeon, Middlesex Hospital, London, and founder of its Medical School.
Sir William Mitchell Banks, 1842–1904. Surgeon, Liverpool Royal Infirmary, and William S. Halsted, 1852–1922.
Professor of Surgery, Johns Hopkins University, Baltimore, were responsible for evolving the radical amputation of the breast as performed to-day. The operation is often known as 'a complete Halsted'.

times these vessels lie in the shadow of the sternum, when the sternum must be split near its lateral border to give access. Towards the close of the operation some surgeons drill holes through the ends of the severed bony structures and unite them with stainless steel wire. While firm pressure is applied to the lateral aspect of the thorax by an assistant, the sutures are tied. The frequency with which the pleura is damaged makes it desirable always to insert a drainage tube through the sixth intercostal space, and attach this to a water-sealed bottle.

Simple Mastectomy.—This may be justified when treating growths close to the sternum because deposits are more likely to have occurred in the internal mammary lymph nodes (Handley) and therefore the removal of the axillary contents is unwarranted.

THE RÔLE OF RADIOTHERAPY IN THE TREATMENT OF MAMMARY CARCINOMA

Pre-operative irradiation renders the tissues a little more vascular, but its main disadvantage is that the patient or her relatives hear that a 'mutilating' operation can be avoided, and consequently may refuse surgical treatment. On the other hand, pre-operative irradiation is especially valuable in Stage II cases where the skin is ulcerated, and occasionally it brings a Stage III case into the category of operability.

Post-operative irradiation is contraindicated when the radical mastectomy specimen reveals no metastases in the axillary lymph nodes. On the other hand, when given to patients who had axillary lymph node involvement in total amounts well over 3,500 r, there is a 5 per cent. increase in the five-year survival rate. Therefore, in all cases where the likelihood of recurrence can be predicted, post-operative irradiation, especially of the anterior mediastinum, is unquestionably a logical procedure.

Treatment of Recurrences and Secondary Deposits.—Deep X-ray therapy causes disappearance or retards the progress of recurrences and secondary deposits in about one-third of cases. Examples are encountered where extensive metastatic deposits are so radio-sensitive that they melt away with this form of therapy, unfortunately only to return in the same, or another, site.

McWhirter's Method of Treatment.—Because either the supraclavicular or the internal mammary lymph nodes are implicated in 48 per cent. of patients with axillary lymph node involvement (p. 603), R. McWhirter considers that it is illogical to clear the axilla of its lymph nodes and allow these oft-involved satellites to remain. He therefore advises simple mastectomy to rid the patient of the primary neoplasm, and relies on vigorous radiotherapy for the treatment of axillary, supraclavicular and internal mammary lymph node involvement. His aim is to give a dose of not less than 3,750 r to the operative area and whole lymphatic field during the three weeks immediately following the operation. He stipulates that the method is contraindicated in (1) very obese patients, (2) aged patients, (3) those with advanced arteriosclerosis, (4) those with tuberculosis, and (5) those with a primary neoplasm in the axillary tail of the breast.

Results.—Unlike some forms of malignant disease, where if the patient survives five years a permanent cure can be assumed, there is no such criterion in cases of carcinoma of the breast. Patients die of secondary mammary carcinoma as long as fifteen or even twenty years after operation. It is, however, abundantly clear that when the axillary lymph nodes are not involved, the prospect of many years of freedom from recurrence is much greater.

The effects of pregnancy after radical mastectomy for carcinoma are unknown but most surgeons advise against having children within 3 years of operation.

Robert McWhirter, Contemporary. Director of the Radiotherapeutic Department, Royal Infirmary, Edinburgh.

Recurrence-free Rates (all forms of treatment)

Stage	5 years	10 years
I	68 per cent.	54 per cent
II	40 " "	25 " "
III	15 " "	4 " "
IV	2 " "	4 * " "

* Includes several patients treated earlier but not accounted for in the five-year list. *(Statistics from the Royal Marsden Hospital, London.)*

Similarly the use of small doses of oestrogens for menopausal symptoms or in the contraceptive 'pill' may accelerate the growth of any remaining tumour tissue.

THE ENDOCRINE TREATMENT OF INOPERABLE AND RECURRENT CARCINOMA OF THE BREAST

Irrespective of their histological structure, mammary carcinomas apparently fall in one of two categories:

1. Hormone Dependent.—About 50 per cent. of mammary carcinomas are hormone dependent; that is to say unless they are nurtured by blood containing one or perhaps more than one of the hormones responsible for normal mammogenesis (fig. 800) they cease to proliferate or metastasise and many of their component cells perish. Unfortunately this regression following hormone deprivation is never permanent.

2. Autonomous.—The cells of which these are composed require no hormone stimulus to enable them to thrive, multiply, and metastasise. Even if carcinomas belonging to group one are deprived of the hormone or hormones that stimulate them, after a varying number of months they, too, become autonomous.

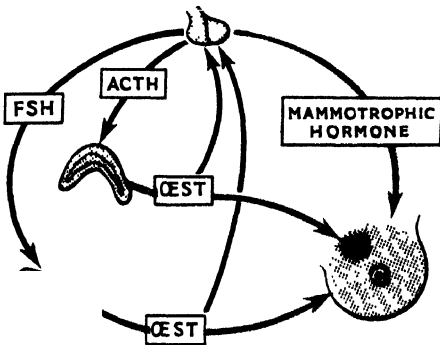


FIG. 800.—The hormonal influences of normal mammogenesis. ACTH=adrenocorticotrophic hormone; FSH=follicle stimulating hormone. *(After Jessiman and Moore.)*

Although the fact that some breast carcinomas are hormone dependent has been known for about seventy years, there is still no direct evidence as to which hormone is responsible. It has always been presumed that the term hormone dependent was synonymous with oestrogen dependent and that these cancers would cease to thrive if they were deprived of naturally occurring oestrogens. Indeed, most of the endocrine treatments for advanced cancer were aimed at those organs (i.e. the ovaries and adrenal glands) that are able to secrete oestrogens. During the past few years, however, since methods have become available for the accurate chemical estimation of oestrogens in the urine, no association has been found between oestrogen excretion and response to treatment. Treatment, therefore, is still on an empirical basis, and although it has been found that certain combinations or sequences of drugs have a beneficial effect, we still do not know why this is so.

Oöphorectomy

This was the first treatment designed to affect the endocrine status of patients with advanced carcinoma of the breast. It was first described in

1896 by Sir George Beatson, of the Western Infirmary in Glasgow, who published an account of four cases treated in this manner. Oöphorectomy or ovarian irradiation is still used extensively as a treatment of recurrent or advanced breast cancer and is beneficial in approximately 20 per cent. of cases. It is of value only before and up to five years after the menopause and the remission so produced can last up to about two years.

Androgens

The administration of androgens probably accomplishes the same as an oöphorectomy although it could be argued that the former would also counteract the adrenal secretion of oestrogens. The remission rate and duration of remission are similar for the two forms of treatment. Administration is usually by injection, either in the form of testosterone propionate, 100 mg. three times a week intramuscularly or one of the newer synthetic drugs, such as Durabolin 25 mg. twice a week intramuscularly. The great disadvantage of this form of treatment is the incidence of virilisation which, if severe, can be most distressing. With Durabolin, which has a more powerful anabolic and weaker androgenic effect, the incidence of side-effects is greatly diminished, and only a problem in 10 to 20 per cent. of cases. Androgens can be administered to all age groups, but are usually considered the treatment of choice for patients who are pre-menopausal or within five years of the menopause.

Oestrogens

Although the prescription of oestrogens may seem contradictory to the theories of hormone dependence, it is perhaps the most efficacious treatment. It should not be given unless the woman is more than five years post-menopausal. The response to the drug increases with age and may reach 60 per cent. in women over seventy. Over-all the remission rate is approximately 40 per cent. Side-effects are few, and although nausea or vomiting can be troublesome in the early stages, this usually subsides. If intractable, the use of a different type of oestrogen may well overcome the difficulty. It is important that the drug is administered in sufficient amounts, as there is some evidence that small doses may actually increase the rate of growth of the tumour. Stilboestrol is usually the drug of choice, and can be administered orally in doses of 50 mg. a day without severe side-effects. An efficient substitute is ethinyloestradiol 1 mg. daily by mouth.

Adrenalectomy¹ and Hypophysectomy

There can be little doubt that when successful these operations can give the greatest degree of benefit to a patient. Remissions for many years can be obtained, and patients are now living over ten years after one of these operations was carried out. In most series some 30 to 40 per cent. of patients have a really worthwhile objective improvement, and in a further 20 per cent. there will be relief of pain and subjective improvement. There seems little to choose between the two procedures.

Adrenalectomy is now usually carried out as a one-stage procedure through a transverse abdominal incision. This avoids the older and more painful two-stage operation where first the right and, after an interval of ten days, the left suprarenal is removed by an incision in the loin with removal of the eleventh

¹ Charles Huggins of Chicago was the first to carry out adrenalectomy successfully. He was the first surgeon to be awarded the Nobel Prize.

or twelfth rib. In all cases bilateral oöphorectomy must be carried out. It is best to do this through a transverse suprapubic (Pfannenstiel's) incision.

Hypophysectomy.—This is now done through the trans-sphenoidal trans-ethmoidal route (for details see textbooks of neuro-surgery). The pituitary is removed piecemeal after making a cruciate incision in the dura with diathermy.

Both procedures must be carried out under cortisone cover and all patients will require to take cortisone as replacement for the rest of their lives. Usually 50 mg. of cortisone acetate a day by mouth is sufficient. Nearly all hypophysectomy cases develop diabetes insipidus and it is necessary to prescribe pituitary snuff or injections of pitressin tannate in oil to overcome this. After about six months the condition spontaneously subsides.

As an alternative to surgical hypophysectomy, pellets of Yttrium 90 can be introduced into the pituitary fossa by the transnasal route. The early disadvantages of this procedure—of meningitis and blindness—seem now to have been overcome, but there is no information to compare the results of this procedure with surgical extirpation. The choice between adrenalectomy and hypophysectomy in any individual case probably depends on the facilities available. Surgical hypophysectomy requires the service of a skilled neurosurgeon if the operative mortality is to be kept within bounds, whereas adrenalectomy can be carried out by the general surgeon.

Cortisone and Ovarian Irradiation

It is known that most of the normal secretion of the adrenal gland can be diminished by the oral administration of large doses of cortisone. It is widely believed that this may accomplish the same result as can be obtained by surgical adrenalectomy, providing it is accompanied by ovarian irradiation or oophorectomy. The drug commonly employed is prednisone administered orally, in a dose of 5 mg. three times a day. Few observers believe, however, that the response to this treatment is as good or as prolonged as can be obtained by surgical hypophysectomy or adrenalectomy. It is, however, simple to administer and has a place in the treatment of advanced breast cancer, although normally reserved for those cases who are either too ill or too old for operative intervention.

SUMMARY OF TREATMENT OF ADVANCED BREAST CANCER (J. L. HAYWARD (IN ORDER OF PROCEDURE)

1. *Surgery.*—Localised lesions may be excised if solitary or causing symptoms.
2. *Radiotherapy.*—Especially for metastases in bone and skin.
3. *Hormones.*—(a) Up to menopause plus 5 years—ovarian ablation with or without androgens.
(b) After menopause plus 5 years—œstrogen therapy.
4. *Hypophysectomy or Adrenalectomy.*—For widespread metastases after hormone therapy has failed—corticosteroids if surgery not possible.
5. *Cytotoxins* (if hormone therapy and endocrine ablation fails)—especially for offensive lesions and pleural or peritoneal effusions.

THE 'FOLLOW-UP' OF CASES OF CARCINOMA OF THE BREAST

It is the duty of the surgeon to examine all his cases periodically for life, so that in event of recurrence he can inaugurate such treatment as is applicable without delay. The following indicates a routine suitable for this periodic examination :

History.—This includes an inquiry as to the physical energy, general health, and the presence of an unexplained cough. Symptoms that the patient usually ascribes to rheumatism, lumbago or sciatica (humerus, spine or femur) must be regarded with suspicion, for often skeletal metastases announce themselves in this way.

Examination.—The operation field is examined for nodules. The axillæ, supra-

clavicular lymph nodes, and opposite breast¹ are palpated. The hand and arm are examined for œdema. The chest is percussed and auscultated and the abdomen examined for evidence of enlarged liver or ascites. If considered necessary, a rectal or vaginal examination is made in order to detect pelvic or ovarian metastases.

Radiography.—In doubtful cases a radiographic examination is made of the chest, or of any bones suspected of harbouring metastases.

THE MALE BREAST

The mammary tissue of the male is the leading, if not the only, example of an external secretory mechanism that persists without actual or potential function.

Mastitis of puberty is discussed on p. 591.

Mastitis from local irritation is by no means rare in men; in civil life it usually occurs in manual labourers with ill-fitting braces (fig. 801). It is not uncommon among soldiers carrying heavy equipment across their shoulders. The treatment is to remove the cause; substitution of a belt for braces is sound advice.

Gynæcomazia.—(a) *Idiopathic.*—Hypertrophy of the male breast may be unilateral or bilateral. The breasts enlarge at puberty, and sometimes present the characteristics of a well-developed female organ. The subjects of this deformity are often virile. Chengwayo, chief of the Zulus (fig. 802), a gynæcomast, at the age of fifty-five, had forty wives and over a hundred children. Tribal tales of a father nurturing his motherless infant

with milk from his own breast belong to the realms of mythology, but pseudo-lactation has been observed in rare instances, and fluid akin to colostrum has been expressed.

(b) *Hormonal.*—Enlargement of the breasts often accompanies stilboestrol therapy, e.g. for carcinoma of the prostate (Chap. 47); it may also occur as a result of a teratoma or a chorion-epithelioma of the testis, in anorchism, and after castration.

(c) *Associated with Leprosy.*—Gynæcomazia is very common in male persons suffering from leprosy.

Possibly this is the result of bilateral testicular atrophy, which is a frequent accompaniment of leprosy (Bowesman).

(d) *Associated with Portal Hypertension.*—Gynæcomazia sometimes occurs in patients with portal hypertension due to failure of the liver to metabolise œstrogens.

(e) It is not uncommon in patients with *Klinefelter's syndrome* (a sexual anomaly giving a chromatin positive male) and in *carcinoma of the bronchus*.

Treatment.—Provided the patient is healthy and comparatively young, the treatment of gynæcomazia is mastectomy, for their possessor is subject to ridicule.

Fibroadenosis is the usual cause of unilateral gynæcomazia. The removed breast shows the same microscopical features as this condition in the female.

Fibroadenoma is not exceedingly rare, and presents the same clinical features as in the female.



FIG. 801.—A braces button being lost, the remaining button draws the brace inwards, and the buckle rubs against the nipple.



FIG. 802.—Chengwayo, from a photograph by Schujelot.



FIG. 803.—Carcinoma of the breast untreated for eight and a half years. The patient could not raise his arm completely because of cicatrisation around the axillary lymph nodes.

¹ A few surgeons recommend early simple mastectomy on the contralateral breast because 7.5 per cent. of patients with carcinoma of the breast develop carcinoma in the contralateral breast.

Carcinoma, which accounts for about 0.6% of all cases of carcinoma of the breast, has an evil reputation: more than one-third of cases are totally inoperable when the patient first presents. In the first place this is due to the fact that so often he does not seek advice for months or years (fig. 803). Secondly, the breast is so small that the enlarging growth reaches the extramammary tissues much sooner than in the female; this favours earlier metastasis.

Treatment.—In cases diagnosed during Stages I and II and treated by radical mastectomy and radiotherapy, the outlook should be favourable, but it is not as good as in the corresponding stages in the female.

In inoperable cases bilateral orchidectomy has an even more advantageous effect than oöphorectomy in the female, favourable results having been reported in as high as 70 per cent. of cases. Oestrogen therapy sometimes produces amelioration after the good effects of orchidectomy have waned. Conversely, prolonged oestrogen therapy employed in the treatment of carcinoma of the prostate has occasionally been associated with the development of mammary carcinoma.

Sarcoma, although uncommon, cannot be categorised as a pathological curiosity.

CHAPTER 27

THE PHARYNX

TONSILS AND ADENOIDS

Surgical Anatomy.—The lymphadenoid tissue of the nasopharynx is Nature's barrier to bacterial invasion. The ætiology of certain cervical inflammations can be better understood if Waldeyer's inner and outer rings (fig. 804) are studied. The *faucial tonsils* are the largest and most important moieties of the inner ring. The tonsils contain tortuous crypts, which extend throughout the tonsillar substance to the external capsule. These crypts can harbour pus and micro-organisms. Clothing the lateral two-thirds of each tonsil is the capsule, a well-defined structure composed of fibrous and elastic tissue, and muscle fibres. The medial third of the tonsil lies between the pillars of the fauces and, being bereft of covering, is accessible to clinical examination. The tonsil has an exceptionally good blood supply (fig. 805). It is well to bear in mind that a tortuous *facial artery* may be closely related to the lower pole. A vein unaccompanied by an artery—the paratonsillar vein—is often a source of serious venous bleeding following tonsillectomy. When divided, the bleeding end retracts into the upper part of the tonsillar fossa, and must be found and ligated before the patient leaves the theatre.

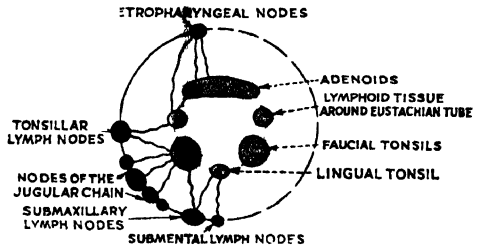


FIG. 804.—Waldeyer's rings. Inner ring—first barrier to infection; outer ring—second barrier.

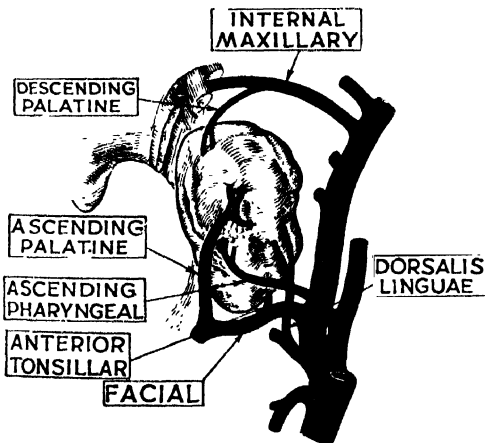


FIG. 805.—The arterial supply of the tonsil from branches of the external carotid. (After R. H. Fowler.)

ENLARGEMENT OF THE TONSILS AND ADENOIDS

Enlarged tonsils are not necessarily infected; a certain amount of hypertrophy is common in early childhood. As adult life approaches, the tonsils, together with other lymphoid tissues, tend to atrophy. Excessive hypertrophy is often bilateral. Occasionally, the tonsils are so large that they almost meet in the middle line.

Enlarged Adenoids¹.—The 'nasopharyngeal tonsil' is present at birth but undergoes atrophy at puberty, although remnants of it often persist into adult life. The most common period for hypertrophy is between the ages of

¹ To be exact—there is only one adenoid.

four and fourteen, and a damp climate favours the development. Enlarged adenoids consist of masses of lymphoid tissue covered by ciliated epithelium and supported by a delicate framework of fibrous tissue.

Considerable adenoid hypertrophy causes the patient to snore loudly at night and to breathe through the open mouth, giving that well-known vacant expression (fig. 806). Added to this, hearing is impaired by the hypertrophied lymph-adenoid tissue obstructing the orifices of the Eustachian tubes, and infections of the middle ear and upper respiratory tract occur frequently.



FIG. 806.—The adenoidal facies. Tonsillar hypertrophy is a usual accompaniment.

Acute follicular tonsillitis is a common condition characterised by pyrexia associated with a sore throat. The cervical lymph nodes are enlarged and tender. Pain occasionally radiates up to the ears. The most usual cause is *streptococcus pyogenes*. On examination the tonsils are swollen, and yellow spots, due to pus exuding from the tonsillar crypts, can often be discerned. The condition can be distinguished from diphtheria by a rapid bacteriological examination of a smear.

Treatment.—Aspirin is administered to relieve pain, and gargles of glycerol-thymol are soothing. When the infection has subsided, removal of the tonsils, or of portions of tonsillar tissue remaining after previous tonsillectomy, must be considered.

Chronic tonsillitis is sometimes associated with hypertrophy. During early childhood chronically inflamed tonsils are usually soft, but by the time puberty has been reached they have frequently become indurated and adherent, due to recurrent attacks of inflammation and subsequent fibrosis. The tonsillar lymph node of the jugular chain is usually palpable. Sometimes pus and debris can be expressed from infected tonsillar crypts to enable a bacteriological examination to be made.

TONSILLECTOMY¹

Indications.—1. A history of recurrent attacks of acute tonsillitis.

2. One attack of peritonsillar abscess.

Removal of the tonsils may be considered in the treatment of:

1. Tuberculous and non-tuberculous cervical adenitis.

2. Rheumatic fever, rheumatoid arthritis, or nephritis if the tonsils are thought to be a focus of infection.

3. Carriers of hæmolytic streptococci and diphtheria organisms.

Operation².—Tonsils can be removed by dissection or by enucleation with a guillotine. Pre-operative treatment with penicillin, and for several days

¹ Should a recently tonsillectomised patient contract poliomyelitis, he is liable to develop the more lethal bulbar type. Tonsillectomy is therefore contraindicated during an epidemic of this disease. The British Association of Otolaryngologists recommend inoculation against poliomyelitis before tonsillectomy is undertaken. The child should have had at least two inoculations before the operation.

² Pre-operative medication for children—in addition to atropine injection, a suspension of thiopentone may be given rectally fifteen minutes prior to the induction of anæsthesia in a dose of 10 to 20 mg./lb. body weight.

after operation, is a wise precaution, as 30 per cent. of patients develop bacteraemia and are in danger of developing infective lesions elsewhere in the body (e.g. bacterial endocarditis, pyelonephritis).

Dissection.—This method is more exact, and bleeding can be accurately stopped by ligating any bleeding vessels, arteries or veins, so that the patient does not leave the operating table until all bleeding has ceased. Either local or general anaesthesia can be employed. The mouth is kept open and the tongue depressed with a Davis's gag.

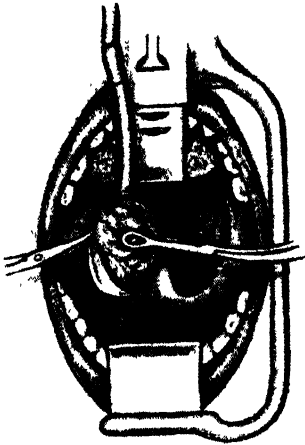


FIG. 807.—Removal of the tonsils by dissection. Davis's gag in place.

The tonsil is seized with vulsellum forceps. An incision is made through the mucous membrane (fig. 807), and the capsule of the tonsil is exposed. The tonsil is removed by dissection, starting at the upper (palatal) pole. When the pedicle is defined, it is severed by a wire snare.

By Enucleation with the Guillotine.—When the tonsils have been removed with the guillotine, considerable hæmorrhage occurs, and special care must be taken to bring this under control (ligatures and pressure). The patient is kept in the operating theatre until the bleeding has ceased and the air-way is clear.

After-treatment.—Until the patient has recovered consciousness he should be kept with his head low and well over to one side (fig. 808).

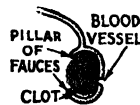


FIG. 808.—Position of patient after tonsillectomy.

On no account should he be permitted to lie on his back or be left unattended.

Hæmorrhage after Tonsillectomy.—The main disadvantage of the guillotine operation is that occasionally serious reactionary hæmorrhage occurs from the tonsillar bed. In such cases prompt measures are necessary. The most important of the immediate measures are : (1) removal of clot from the tonsillar bed (fig. 809), (2) the

FIG. 809.—Hæmorrhage from the tonsillar bed may be due to clot preventing the surrounding musculature from contracting. (*After Lee McGregor.*)



application of pressure and styptics by means of a swab on a holder. If bleeding persists in spite of these measures an intravenous infusion *must* be set up and blood taken for grouping and cross-matching (p. 73). The patient should be returned to the operating theatre, and under general anaesthesia the bleeding-point sought and ligated. When the bleeding-point cannot be found, coaptation of the pillars of the fauces with sutures will arrest the hæmorrhage¹.

ADENOIDECTOMY

Indications.—Hypertrophic adenoids associated with: (a) Recurrent otitis media. (b) Post-nasal obstruction. (c) Post-nasal discharge. (d) Recurrent sinusitis.

¹ Morphine must not be given to children suffering from post-operative hæmorrhage. It is a factor in tonsillectomy deaths.

The original Davis's gag was invented by Dr. Davis, of Boston, Mass. Henry Edmund Gaskin Boyle, 1875–1941, anaesthetist, St. Bartholomew's Hospital, London, improved it.

Removal of Adenoids.—Removal is undertaken either before dissection of the tonsils, or at the conclusion of guillotine tonsillectomy. Adenoids are removed with a guarded curette (fig.

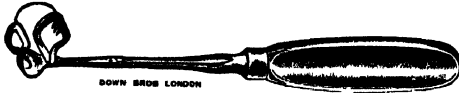


FIG. 810.—St. Clair Thomson's adenoid curette.



FIG. 811.—Curettage of adenoids.

810) pressed against the roof of the nasopharynx (fig. 811) and then carried backward and downward with a firm sweeping movement. The after-treatment is similar to that described above. Reactionary hæmorrhage can almost always be stopped by sitting the patient bolt upright. If this is not quickly successful, after removal of clots a succession of swabs dipped in hydrogen peroxide, applied on a holder, usually remedies matters unless a tag has been overlooked, in which case the tag must be removed with punch forceps. As a last resort, the nasopharynx may have to be packed. A rubber catheter is passed through one nostril and withdrawn through the mouth. A piece of folded sterile gauze is attached to the catheter by strong silk. The catheter with the pack is then drawn back until the pack rests in the nasopharynx, and bleeding is arrested by pressure.

MALIGNANT TUMOURS OF THE TONSIL

Both carcinoma and lymphosarcoma occur in the tonsil. The diagnosis in many instances is not easy. Any unilateral enlargement of the tonsil occurring in adult life should be regarded with suspicion. A biopsy is often required to confirm the diagnosis.

Carcinoma of the Tonsil (85 per cent.).—The patient is commonly an elderly man and pain is the leading symptom. The pain is severe and radiates to the ear, and, unlike that of tonsillitis, is unilateral. The breath is foul. Later, bleeding occurs, and as the ulcer deepens the loss of blood may be copious.

Treatment is by external irradiation of the primary growth and the side of the neck as far as the clavicle. Small residues can be treated with accessory local irradiation. The five years' survival rate after treatment is 25 per cent.

Lymphosarcoma of the tonsil (15 per cent.) has the reputation of being very malignant. While this is true if it is allowed to grow beyond the peritonsillar bed, the condition is by no means hopeless in its early stages. The patient is usually between fifty and sixty years of age, complains of a lump in the throat, which in the early stages is painless. Thick speech is a common symptom, and the tonsil appears large and pale. Later, the growth spreads, and often forms a swelling of the palate, which may be mistaken for a peritonsillar abscess, and incised. Once the barrier formed by the capsule of the tonsil has been breached, the growth extends rapidly into the neck, often forming a swelling behind the angle of the mandible. While the cervical lymph nodes soon become involved, a swelling in this position is likely to be an extension of the primary growth. Eventually bleeding, dysphagia, and dyspnoea foretell that the end is not far distant.

Treatment is by external irradiation of the growth and of the lymph nodes, and some apparently hopeless cases may respond dramatically.

PERITONSILLAR ABSCESS (*syn.* QUINSY)

As its name implies, a peritonsillar abscess is due to suppuration occurring outside the capsule of the tonsil, most usually in the tissues of the soft palate (fig. 812). The route of infection appears to be by way of the intra-tonsillar cleft, which is embryologically a remnant of the second pharyngeal pouch. As a rule the abscess is unilateral, but it is not uncommon for the contralateral side to become involved a few days later. The condition

s rare in children, the incidence being highest in adult males. The general symptoms are often severe. Extreme pain is experienced in the tonsillar region, radiating to the ear and to the side of the neck. Swallowing is so painful that saliva dribbles from the mouth; speech is thick and muffled. As the patient can open the mouth only to a slight extent, examination is often difficult. With good illumination, a diffuse swelling of the soft palate, mainly near the superior border of the affected tonsil, will be seen. The swelling displaces the œdematous uvula to the contralateral side.

Treatment, in the early stages, is the same as that for acute follicular tonsillitis (p. 618). In addition, penicillin should be administered intramuscularly. If suppuration occurs, evacuation of pus in the following manner should not be delayed.

A bistoury is modified by winding a strip of strapping around the blade so that only 1 cm. ($\frac{2}{8}$ inch) of the tip projects. Except in small children, no general anæsthetic is used¹. The patient sits upright. An incision is made in the position shown in fig. 812, which is usually described as midway between the base of the uvula and the third upper molar tooth. Dressing forceps are now pushed firmly *directly backwards*. As soon as pus is encountered, the forceps are opened widely and withdrawn. Some surgeons remove the affected tonsil and thereby drain the abscess effectively.

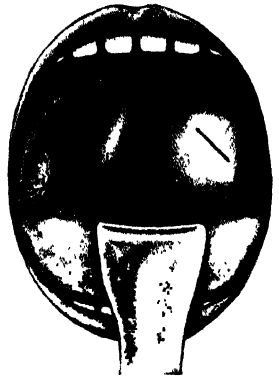


FIG. 812.—Peritonsillar abscess, showing site of incision.

Parapharyngeal abscess is similar to the above, but the maximum swelling is behind the posterior faucial pillar, and there is little or no œdema of the palate. The abscess is opened with a really blunt instrument, such as a tongue depressor. Often the gloved finger will suffice.

RETROPHARYNGEAL ABSCESS

Acute.—*Between* the prevertebral fascia and the pharynx².

Chronic.—*Behind* the prevertebral fascia.

Acute retropharyngeal abscess is seen most commonly in children under the age of four. It is the result of suppuration of the lymph nodes that occupy the space. The prevertebral lymph nodes are most luxuriant during the first year of life; this explains why fifty per cent. of cases occur under one year of age. The portal of entry is the tonsils, the nasopharynx, or the oropharynx.

In infants the condition is sometimes very acute, and accompanied by rigors, convulsions and vomiting. The neck is held rigidly, usually on one side, saliva dribbles from the child's mouth, and feeds are regurgitated. A croupy cough is common. The cry may resemble a 'squawk'. *Difficulty in breathing is the leading symptom* and this should always be the signal to examine a child's throat. The posterior wall of the pharynx is swollen.

¹ In adult patients surface anæsthesia (five per cent. cocaine on a pledget of wool), is the safest.

² The buccopharyngeal fascia divides this fascial compartment down its centre, hence an acute retropharyngeal abscess is always to one side of the midline.

This is sometimes visible only when the base of the tongue has been depressed firmly. On digital examination a localised soft cushion-like projection can be felt on the posterior pharyngeal wall. The only condition with which acute retropharyngeal abscess may be confused is laryngeal diphtheria. A less acute form is seen in older children as a complication of otitis media.

Treatment.—No anæsthetic is used for infants, who must be placed in the prone position, with the head low. A pair of dressing forceps guided by the finger is thrust into the abscess cavity, the contents of which are evacuated. Suction must be available. Suitable antibiotic therapy is prescribed.



FIG. 813. — Chronic retropharyngeal abscess secondary to tuberculous disease of the cervical vertebrae.

Chronic retropharyngeal abscess is sometimes due to an extension of tuberculosis of a cervical vertebra (fig. 813), and this possibility should always be confirmed or eliminated by suitable radiographs of the cervical spine. With the decline in the incidence of bone tuberculosis in this country, retropharyngeal abscess from this cause is now encountered less frequently than formerly. Cases of chronic retropharyngeal abscess due to tuberculous retropharyngeal lymph nodes are less uncommon. When the collection of pus is large, in addition to the retropharyngeal swelling, there is a fullness behind the sternomastoid on one side. A chronic retropharyngeal abscess must never be opened into the mouth, for such a procedure will lead to secondary infection. The pus should be evacuated by an incision in front of the sternomastoid, to lead into the plane between the carotid sheath and the thyroid gland. The dissection towards the retropharyngeal space is conducted carefully until the abscess is opened. The cavity is then mopped dry and the wound closed. Suitable treatment of the underlying tuberculous lesion must then be instituted (p. 23).

DIVERTICULUM OF THE PHARYNX

Congenital lateral diverticulum is really a blind internal branchial fistula (p. 519) opening into the fossa of Rosenmüller (the pharyngeal recess). Occasionally such a fistula becomes distended and food lodges within it.

Pharyngeal Pouch.—**Ætiology.**—The inferior constrictor muscle is made up of two portions—the upper oblique fibres (thyropharyngeus) of each side that interdigitate posteriorly, and the lower fibres (cricopharyngeus) that are arranged transversely, and function as a sphincter. Between these two sets of fibres there is a weak area in the middle line posteriorly, known as Killian's dehiscence which is marked by a dimple (fig. 814) in the overlying mucous membrane. The lower (sphincteric) portion of the cricopharyngeus constantly maintains enough tone to prevent the entry of air into the œsophagus during inspiration (Negus). If for some reason the cricopharyngeus fails to relax during swallowing, the pressure exerted by the bolus falls on Killian's dehiscence; so commences a pharyngeal pouch (fig. 815). As time goes



FIG. 814.—The pharyngeal dimple.

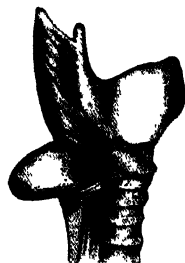


FIG. 815.—Pharyngeal pouch protruding between the thyropharyngeal and cricopharyngeal portions of the inferior constrictor of the pharynx. (After Sir Victor Negus.)

on, the sac becomes larger and fills with food at every meal. Unable to expand posteriorly because of the resistance of the vertebral column, the pouch turns outwards, usually to the left (fig. 816), and obtrudes itself into the side of the neck. The pouch is the *result*, not the cause of the dysphagia.

Clinical Features.—Patients suffering from this condition are usually elderly, and it is twice as common in men as in women.

There are three stages in the development of symptoms. Rarely does the patient present during the first stage.

Stage 1.—As shown by a lateral radiograph after a barium swallow, there is a small diverticulum directed towards the vertebral column, viz.→

Usually a small pharyngeal diverticulum is symptomless, and the finding of it is incidental during the course of a barium meal. Occasionally it gives rise to symptoms identical with those of a foreign body in the throat. At this stage the diverticulum can be ignored.

Stage 2.—The diverticulum is larger and more globular, but its mouth still lies in the vertical plane, viz.

Regurgitation of undigested food at an unpredictable time after a meal, during the swallowing of the next meal, or after turning from one side to the other at night, is the chief complaint. Sometimes the patient is awakened from sleep by a feeling of suffocation, followed by a violent fit of coughing. Infrequently an abscess of the lung results from food inspired from the pouch. Removal of the pouch is indicated.

Stage 3.—The pouch has become larger, and what is so important is that its mouth looks horizontally upwards, viz. →

The fundus of the pouch has become dependent, and consequently when the pouch is full it compresses the œsophagus. The symptoms of the second stage persist; in addition, there are gurgling noises in the neck, especially when the patient swallows. In about one-third of cases, the pouch is large enough to form a visible swelling in the neck: sometimes such a pouch can be seen to enlarge when the patient drinks. Nevertheless, when this stage has been reached the symptom that transcends all others is increasing dysphagia, and in a large number of instances it is this symptom alone that compels the patient to seek relief. However, even at this stage a few intelligent patients acquire a knack of overcoming their disability. It is recorded that Lord Jeffrey, a Scottish nobleman, was in the habit of emptying his pouch with a large silver spoon after every meal. Other sufferers have found that they can swallow their food better if the pouch is full, and accordingly take porridge as a first course. Notwithstanding these ingenious devices, eventually there is progressive loss of weight due to semi-starvation, and cachexia is sometimes extreme.

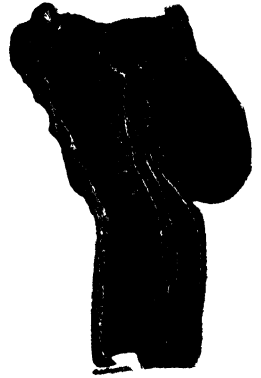


FIG. 816.—A pharyngeal pouch, showing compression of the adjacent œsophagus.

Radiography.—If a pharyngeal pouch is suspected, a very thin emulsion of barium should be used for the barium swallow; a thick mixture often requires much washing through a tube to remove the barium from the pouch. Quite often the fundus of the sac will be seen invading the superior mediastinum. Radiologically, the antero-posterior appearance of a barium-filled pouch can be simulated closely by a partial septum obstructing the commencement of the œsophagus. Therefore, if this mistake is to be avoided a semi-lateral view (fig. 817) also must be taken. In this view the overflow of barium emulsion into the œsophagus often can be seen to come from the top of the pouch—not from the bottom, as is the case with an œsophageal web.



FIG. 817.—Semi-lateral radiograph of a pouch after barium has been swallowed.

has perforated its fundus, which is thin and fragile, and mediastinitis has resulted.

Treatment.—When the pouch is of a considerable size, operation is strongly advised, because progressive symptoms are inevitable. When emaciation is extreme a preliminary temporary ‘feeding’ gastrostomy or jejunostomy may be required.

The operation is performed in one stage. To prevent mediastinitis, antibiotics should be given before and after operation.

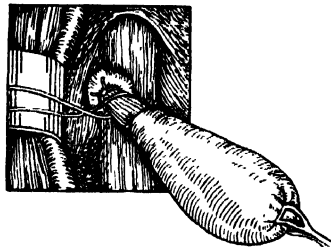


FIG. 818. — Double ligation of the mucous membrane of the neck of the sac. Note that a cuff of the superficial layers of the neck of the sac has been dissected back. This will be sutured over the stump. (After Frank Lahey.)

Operation.—The pouch is approached through either a transverse incision at the level of the cricoid cartilage, or, as many prefer, an oblique incision following the anterior border of the left sternomastoid. The first step is to mobilise the lateral lobe of the thyroid gland, therefore it is necessary to ligate and divide the middle thyroid veins, and sometimes the inferior thyroid artery. When this has been completed, the lateral lobe is retracted forwards, and the carotid sheath is retracted backwards. At this stage a large stomach tube is passed by the anæsthetist and it enters the pouch; the tube is guided by the surgeon from the pouch into the œsophagus, and there it remains during the operation as a *helpful guide in determining the limits of the neck of the sac during the later stages of the dissection.*

The walls of the sac vary in thickness; in some cases they are so thin that great care must be taken not to tear them. Having freed the pouch completely, a cuff of the outer layer of the pouch is dissected from the mucous membrane. This permits the closure of the neck of the sac, which must be performed very accurately in two layers (fig. 818); alternatively, if it is considered that the neck of the pouch is

too wide for ligation, it is closed in two layers in the same manner as a duodenal stump is closed after partial gastrectomy. It is very important not to narrow the upper end of the œsophagus at the point where the pouch is removed. In some cases it is useful to divide any hypertrophied circular muscle (crico-pharyngeus) (cf Heller's operation, p. 722). The wound is closed with drainage.

After-treatment.—The patient is fed through an indwelling transnasal gastric tube for three days. Fluids only are permitted for the next three days. After this, semi-solids are given, and the diet is then increased gradually.

Complications.—1. *Infection.*—Severe infection of the wound and the mediastinum is now infrequent. 2. *Pharyngeal Fistula.*—Usually the fistula closes spontaneously.

NEOPLASMS OF THE PHARYNX

Surgical Anatomy.—Concerning neoplasms of the pharynx, the clinico-anatomical division into various component parts (fig. 819) is of practical importance.

The nasopharynx (*syn.* post-nasal space, epipharynx) is that portion of the pharynx lying above the level of the soft palate which forms its incomplete floor. With the exception of this floor, the nasopharynx has rigid, immovable walls. Each Eustachian tube opens into the antero-lateral wall of the nasopharynx just behind the posterior end of the inferior turbinate. Above and behind this orifice is the supratonsillar fossa of Rosenmüller.

The oropharynx (*syn.* the mesopharynx) extends from the inferior border of the soft palate to the lingual surface of the epiglottis. In the sulcus between the back of the tongue and the anterior (lingual) surface of the epiglottis lie a median glosso-epiglottic and a right and left pharyngo-epiglottic fold. The corresponding depression on either side of the glosso-epiglottic fold is known as the vallecula (fig. 820).

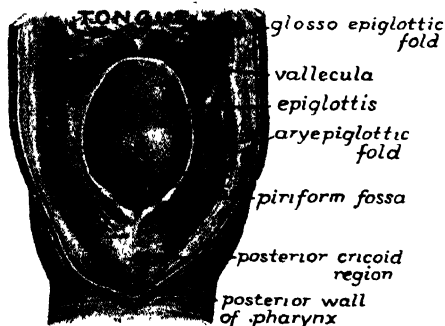


FIG. 820.—The epiglottis: the valleculæ are shown also. In, or on, all the structures labelled a carcinoma can arise.

of the hypopharynx, except during deglutition, is kept closed by tonic muscular contraction. In the antero-superior portion of the laryngopharynx lies the inlet of the larynx, and it is the immediate environs of this inlet that are known as the epilarynx (fig. 820). The inlet of the larynx faces almost backwards, and is bounded above by the epiglottis, laterally by the aryepiglottic folds, and below by the short interarytenoid fold. On the lateral side of each aryepiglottic fold is a recess—the pyriform fossa.

TUMOURS OF THE NASOPHARYNX

Benign:

Angio-fibroma¹.—Although its local behaviour is the antithesis of benignity, this tumour is not malignant, for it never metastasises, neither does it infiltrate tissues. However, on account of its ability to send tentacles into first one and then the other

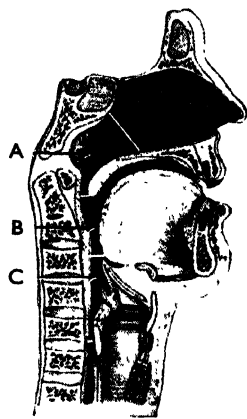


FIG. 819.—The component parts of the pharynx. (a) Nasopharynx; (b) Oropharynx; (c) Laryngopharynx, the antero-superior portion of which contains the inlet of the larynx, and is known as the epilarynx.

The laryngopharynx is the longest of the three divisions of the pharynx and it diminishes in width from above downwards. It extends from the tip of the epiglottis to end opposite the body of the sixth cervical vertebra, where it is continuous with the œsophagus. It is convenient to subdivide the laryngopharynx into the *epilarynx* above and the *hypopharynx* below, the dividing line being at the level of the upper border of the cricoid cartilage (Stanford Cade). As has been explained on p. 622, the lowest part

¹ Formerly called 'nasopharyngeal tumour', which is ambiguous.

² Sir Stanford Cade, *Contemporary*. Honorary Consulting Surgeon, Westminster Hospital, London.

nasal fossa, and thence into the accessory nasal sinuses, and above all because of its power to cause pressure necrosis of bone, it is a *highly destructive* tumour. As a result of these intrusions the tumour expands the nose, may fill the antra and in turn expand the cheek, and cause the palate to bulge, and at times invades the ethmoid and produces a 'frog face' appearance.

Nasopharyngeal angio-fibroma is a reddish, firm tumour covered with normal mucous membrane. Ulceration seldom occurs unless the tumour is traumatised. Histologically it is composed of immature fibroblasts and blood-vessels ; in the early stages cavernous blood-vessels predominate. In long-standing cases fibrous tissue is more plentiful.

Clinical Features.—This tumour is almost confined to juvenile male patients.

Appearing at the age of puberty, the tumour usually regresses in the early twenties, provided that no secondary complications occur. Although nasopharyngeal angio-fibroma is rare, when a boy presents with progressive nasal obstruction, recurrent epistaxis, a purulent nasal discharge and a firm mass in the nasopharynx, this clinical entity should spring to mind.

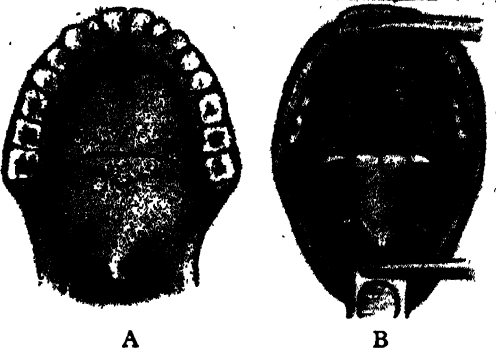


FIG. 821.—Transpalatine approach to the nasopharynx. A. Incision. B. Permanent fenestration of the hard palate. (After C. P. Wilson, F.R.C.S.)

Biopsy should be avoided unless there are compelling reasons for undertaking it, when matched blood must be in readiness and the surgeon must be prepared to deal with hæmorrhage.

Treatment.—Pre-operative external irradiation will shrink the tumour and render it less vascular. Removal of the tumour should follow some six weeks later.

Operation.—The transpalatal route (fig. 821A) is used. The tumour is excised with diathermy and the soft palate is replaced and

sutured to the mucoperiosteum of the hard palate. Alternatively, in order that early recurrences may be seen, and destroyed by diathermy, a permanent palatal fenestra can be made (fig. 821B). The fenestra is closed by an obturator attached to a denture. The patient can then talk and eat quite normally.

A choanal polypus may give rise to difficulty in differential diagnosis. Ætiologically it belongs to nasal polypi, originating either in the ethmoid region or in the maxillary antrum. If large, it may descend through the posterior nares into the nasopharynx and present behind the soft palate (fig. 822). A choanal polypus may be distinguished from angiofibroma by its mobility due to the long pedicle by which it is attached, avascularity and an elastic rather than firm consistency. It can be easily removed with a snare.

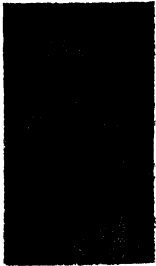


FIG. 822.—Large choanal polypus descending into the nasopharynx and presenting behind the soft palate.

Malignant :

In China, Japan, and Malaya malignant tumours are more common in the nasopharynx than in any other part of the body save the cervix uteri, a possible cause being the use of smoky kerosene lamps in these countries.

Type of growth

Carcinoma	67 per cent.
Reticulosarcoma	15 " "
Lympho-epithelioma ¹	11 " "
Other tumours, including mixed salivary tumour	7 " "

(James Ewing)

fifty per cent. of these growths arise in the lateral wall of the nasopharynx, mostly in the supratonsillar fossa of Rosenmüller (fig. 823); the remainder are divided equally between the roof and the posterior wall.

Clinical Features.—These depend mainly on whether the growth is obstructive (reticulosis), or infiltrating (carcinoma). Usually the *first* symptoms for which advice is sought fall into four groups:

1. *The Nasal Group.*—Slight, intermittent epistaxis and nasal speech are early nasal symptoms; other nasal symptoms, viz. a feeling of obstruction to the airway and a post-nasal discharge, usually are delayed.

2. *Aural Group.*—Unilateral deafness, with pain in the ear, is the usual complaint. Obstruction of the pharyngeal orifice of the Eustachian tube by a growth leads to a collection of sero-sanguinous fluid within the middle ear. The deafness that results is relieved by paracentesis of the tympanic membrane and suction, but a fresh accumulation soon occurs. Bleeding on Eustachian catheterisation is a sign of the utmost importance.

3. *Enlarged Cervical Lymph Nodes.*—By the time the diagnosis is established, 70 per cent. of patients with a malignant tumour of the nasopharynx have enlarged cervical lymph nodes, and 40 per cent. present on account of the cervical swelling. The swelling is in the upper jugular chain and the nodes are firm, rather than hard, and may be mistaken for tuberculous adenitis.

Unlike carcinoma of the tongue, metastases more distant than the neck occur eventually in about a quarter of cases.

4. *Cranial Nerve Involvement.*—In Far Eastern patients this is the most common presenting symptom; in British patients 30 per cent. have some cranial nerve involvement when first seen. All suffer implication by the growth either at their exit through their respective foramina at the base of the skull or, less frequently, by intracranial extension of the growth. Pain of trigeminal distribution is an important feature in this group. X-ray of the base of the skull may show destruction of bone by the tumour, but it is not unusual for patients to develop root pains without X-ray changes.

Trotter's Triad.—The three cardinal symptoms of a locally invasive tumour can be summarised as follows: (i) Conductive deafness. (ii) Elevation and immobility of the homolateral soft palate—due to direct infiltration. (iii) Pain in the side of the head—due to involvement of the fifth cranial nerve from infiltration via the foramen lacerum.

Biopsy is necessary to ascertain the histological characteristics of the tumour. Inspection of the nasopharynx is facilitated if a soft rubber catheter is passed through the nose, and withdrawn through the mouth. Gentle traction displaces and immobilises the soft palate, and thereby an uninterrupted view is obtained. If more detailed inspection is desirable, Wilson's



FIG. 823.—Papillary carcinoma of the fossa of Rosenmüller, as seen in a laryngeal mirror. (After W. L. Mattick.)

approach (fig. 821) through the junction of the hard and soft palate should be used.

Treatment.—Present practice favours supervoltage external irradiation, given to the primary tumour and to the lymph node fields on both sides of the neck at least as far down as the clavicles. Reticulosarcoma, undifferentiated carcinoma, and lympho-epithelioma are very radio-sensitive. If the primary responds satisfactorily, it is feasible to perform a cervical block dissection of the enlarged lymph nodes.

Prognosis.—Owing to their secluded position and consequent late diagnosis, and the inaccessibility of the nasopharynx, the prognosis is poor. The five-year survival rate is about 40 per cent. for the reticulosis group and under 20 per cent. in the case of carcinoma.

NEOPLASMS OF THE OROPHARYNX

Benign:

Diffuse cavernous angioma involving the pharynx, fauces, and often extending into the neck (where it forms a swelling) has been treated successfully by injecting into the bluish mass in the pharynx 0.5 ml. of 1 : 4 solution of ferrous chloride in sterile water. The injected area immediately turns bright red, remains swollen for a few days, and then cicatrizes. Further injections may be required. Later, if necessary, abnormal tissue is removed by diathermy, with practically no bleeding.

Malignant:

Usually carcinoma of the oropharynx is of the ulcerative type. The commonest site of origin is the tonsillo-lingual sulcus. There is discomfort at the back of the throat, foetor, and blood-stained sputum. Pain is absent until the growth is far advanced.

Treatment.—The management is identical to tumours of the nasopharynx of similar histological picture.

Operation.—Although the long-term results are far from good, when possible, extirpation of the growth and the regional lymph nodes holds out a better prospect of success than irradiation.

Lateral pharyngotomy with partial pharyngectomy is performed as follows. A tracheostomy is usually performed as the first step. Then a block dissection of the cervical and retropharyngeal lymph nodes is carried out. The lateral lobe of the thyroid is then mobilised and displaced forwards. The inferior constrictor muscle is detached from the thyroid and cricoid cartilages. The great cornu of the hyoid bone and the posterior two-thirds of the ala of the thyroid cartilage are removed without opening the mucous lining.

Should the growth be situated on the lateral wall and have invaded the tonsillar region, the cervical operation is halted at this stage, and through the widely open mouth an incision is made at least 1 inch (2.5 cm.) distant from the accessible margins of the neoplasm with a diathermy knife, the internal carotid artery being held aside in the neck (Raven). Returning to the neck, the pharynx is opened longitudinally and that portion of the pharyngeal wall containing the growth and a wide margin of healthy tissue is removed in continuity with its lymphatics.

When the loss of the pharyngeal wall is not great the pharynx is closed. A portion of the upper part of the posterior skin flap is anchored by stitches to the prevertebra fascia, thus providing free exit to infected matter should leakage occur. The remainder of the cervical wound is closed with drainage at its lower end.

After-treatment includes transnasal intragastric feeding and antibiotic therapy.

When it is known beforehand that the defect in the pharyngeal wall is likely to be a large one, the operation is performed through a rectangular skin flap, and after partial pharyngectomy has been performed, the first stage of the plastic procedure is carried out as shown in fig. 824. In the early post-operative period, the patient is fed by a tube passed into the stomach through the fistula.

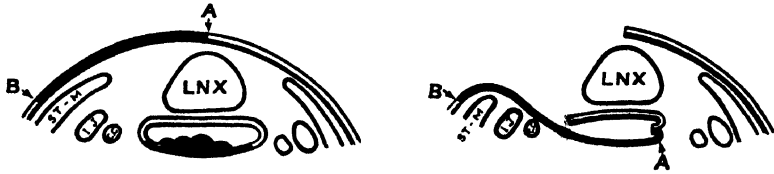


FIG. 824.—Trotter's method of reconstructing the pharynx from a flap of the skin (A B) of the neck after excision of an extensive pharyngeal carcinoma.

In both the procedures just described the resulting cervical fistula will require closure; this is undertaken after a short convalescence.

Growths situated in the vallecula, unless very advanced, must be treated by pharyngolaryngectomy with, if necessary, excision of a portion of the back of the tongue.

NEOPLASMS OF THE LARYNGOPHARYNX

In accordance with their site of origin, it is customary to subdivide malignant tumours of this part of the pharynx into four groups:

1. Epilaryngeal (20 per cent.).—Nearly always the patient is a man between fifty and sixty years of age. The lesion is situated on an aryepiglottic fold, extending to the epiglottis or the corresponding arytenoid cartilage, and is either of the ulcerative or the papillary type. It may be symptomless in the early stages. The earliest symptom is a slightly sore throat. A muffled voice and increasing dysphagia occur later. Real hoarseness indicates involvement of the interior of the larynx, i.e. the vocal cord. Later there are attacks of dyspnoea associated with blood-stained sputum. The diagnosis is made by indirect laryngoscopy.

2. Sinus Piriformis¹ (40 per cent.).—Again, this group occurs chiefly in men about fifty years of age, and the lesion is notoriously silent. Often its first intimation is an enlarged lymph node behind the angle of the jaw. Frequently this is not heeded in its early stages (fig. 825). Exceptionally, the patient presents himself at an earlier stage, because of slight difficulty in swallowing saliva, as opposed to food. Pain is absent until the growth has involved the superior laryngeal nerve. The pain is referred to the ear. Carcinoma of the piriform fossa is nearly always of the ulcerative type. The growth may be seen with a laryngeal mirror, but sometimes its extent cannot be determined without direct laryngoscopy.

3. Lateral Wall (12 per cent.).—Once again men

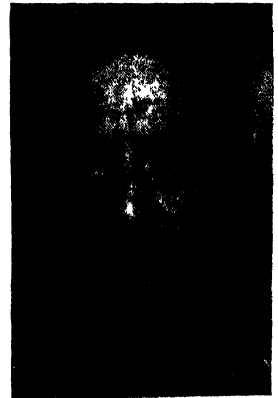


FIG. 825.—Malignant lymph nodes of the neck secondary to laryngopharyngeal carcinoma in the 'silent' area. The patient presented himself on account of the lump in the neck.

¹ Piriformis—Latin, *pirum* = a pear.

are attacked much more often than women. Contrary to the sinus piriformis, the growth is often papillary.

A lateral radiograph of the neck, with air inflation of the pharynx, is often more informative than a barium swallow in the demonstration of a neoplasm in this and nearby situations.

4. Post-cricoid (28 per cent.) occurs on the anterior wall of the hypopharynx at the level of the cricoid cartilage. The patient is nearly always a woman of about forty years of age, who gives a history of increasing dysphagia.

The higher incidence in women is possibly due to their ability to swallow hotter fluids and food than men can tolerate. Many of these neoplasms are secondary to the Plummer-Vinson syndrome (p. 720).¹ Indirect laryngoscopy seldom reveals the growth, which lies hidden beneath a pool of mucus. Radiographic examination after a barium swallow is often helpful in determining the site of the lesion. Direct pharyngoscopy is the most informative, and allows a portion of the growth to be removed for biopsy.

Treatment of Carcinoma of the Laryngopharynx.—The condition is notoriously difficult to treat successfully. Surgery is often the treatment of choice : it involves the major operation of laryngo-pharyngectomy, combined with block dissection of the neck, the establishment of a permanent tracheostomy, and the plastic reconstruction of the pharynx. About 20 per cent. of patients survive five years.

External irradiation is the alternative method of treatment and is advised for those patients who are considered unsuitable for such a major procedure, either because of their poor general condition, or because it is thought that they will not be able to adjust themselves to a life without a larynx.

Types of Operation.—Operations which combine excision and reconstruction of the gullet in one stage are preferable to staged procedures. The following is an example of a one-stage operation, in which the colon is used to replace the pharynx :

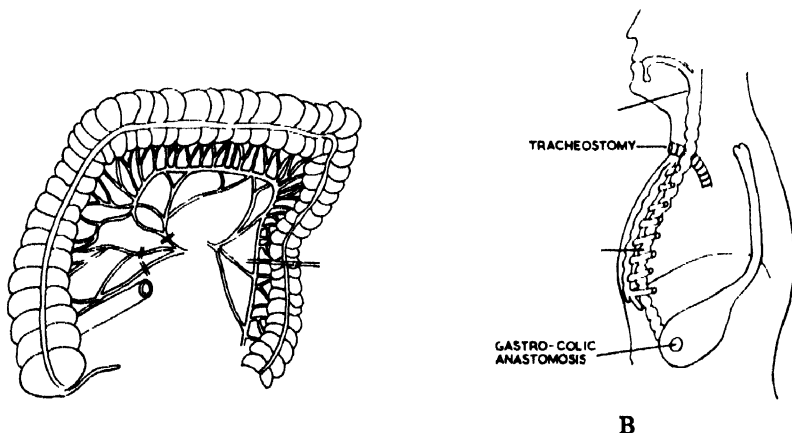


FIG. 826.—Reconstruction of the pharynx, using colon.

(A) Mobilisation of the colon. The middle and right colic vessels are divided. The blood supply of the colon now depends upon the left colic artery and the marginal arterial anastomoses. (B) The completed operation. The right and transverse colon have been passed retrosternally up into the neck. (J. T. John, F.R.C.S., Bath.)

Two teams are used. The first team performs the pharyngo-laryngectomy, the block dissection of the lymph nodes of the neck on the side of the tumour, and establishes the tracheostomy. The larynx and pharynx are excised from above the level of the hyoid bone down to the level of the third or fourth tracheal ring, and including the whole of the cervical œsophagus. The stump of the œsophagus is closed by inverting sutures and allowed to slide into the thorax. The thyroid gland is included in the excision. A block dissection of lymph nodes on the less involved side may be undertaken, but the sternomastoid muscle and the internal jugular vein are usually spared.

The second team, working in the abdomen, mobilises the colon so that it is attached only by the mesocolon and its blood supply. The pattern of the blood supply of the colon is such that a long enough length of bowel can be obtained to join the oro-pharynx to the stomach based on a single feeding artery, usually the upper left colic artery (fig. 826A). A tunnel is made in the anterior mediastinum by stripping the pericardium, pleura, and great vessels from their sternal attachments. The colon is drawn up through the tunnel and the upper end anastomosed to the divided oro-pharynx and the lower end to the front of the stomach (fig. 826B). Restoration of continuity of the abdominal colon is achieved by end-to-end anastomosis.

The patient is allowed to swallow fluids the following day and to eat normal foods within two weeks. Should recurrence of the neoplasm occur, radiotherapy can be given with little risk of damage to the artificial gullet and hence little interference with the patient's swallowing.

Alternatives. (i)—The colon may be drawn up through a presternal, subcutaneous route (fig. 827). (ii) Another method of reconstruction is to transect the œsophagus at the cardia closing its distal end, and mobilising the stomach to elevate it through the posterior mediastinum: then it is sutured to the pharyngeal remnant high in the neck.

CHEMOTHERAPY IN HEAD AND NECK CANCERS

Cytotoxic drugs in the treatment of head and neck cancers cannot at present compete with surgery or radiotherapy, but they do have a limited value as an adjunct to these forms of therapy in patients who have not responded, or who have recurrent growths. Various methods of administration are possible. Growths in this region are particularly suitable for treatment by *intra-arterial infusion*. The reasons are that the neoplasms are mainly within the supply of the external carotid artery and that they rarely metastasise systemically until very late. Tumours of the ear, nasopharynx, oropharynx, and tonsils, maxillary sinuses and laryngopharynx can be treated in this way.

The cytotoxic agents used are two types: (a) Biological alkylating agents, such as nitrogen mustards and epoxides (Ethoglucid). These are given by single injections directly into the exposed appropriate artery. Their activity only lasts a few minutes and further injections, say every other day, may be given. (b) Anti-metabolites, such as methotrexate; these are administered as a continuous intra-arterial infusion lasting two or three weeks.



FIG. 827.—Reconstruction of the pharynx with pre-sternal colon, showing patient (left) and his barium swallow (right).

Technique.—The external carotid artery is exposed through an oblique neck incision. Its branches are identified so that the arterial supply to the tumour area can be isolated. A catheter is threaded through an incision in the superior thyroid branch, passed in a retrograde manner to the external carotid and thence to the artery of supply. It is firmly tied in place. For tumours in the lower part of the neck such as hypopharynx, the superficial temporal artery is catheterised instead.

Other branches of the external carotid are either clipped off or ligated. A tracer dye (methylene blue or disulphine blue) injected into the cannula will discolour the area of infusion and ensure that it is limited only to the region of the tumour (fig. 828). The artery must also be isolated if the single injection method is being used. The neck incision is closed and the infusion fluid is injected using a small pump, calibrated to give the appropriate rate of flow.



FIG. 828.—Perfusion with cytotoxic drugs. The tracer dye (see text) shows that the intra-arterial catheter is correctly placed for perfusion of carcinoma of the ethmoid. (*Professor D. F. N. Harrison, F.R.C.S. London.*)

The infusion can be followed up with intravenous or oral cytotoxic drugs. To be effective, the white cell count should not rise above 2000 c.mm.

The toxic side-effects of cytotoxic drugs include: bone marrow depression, alopecia, cutaneous vesiculation, and œdema. They may be lessened by giving a systemic antidote at the same time as the infusion (e.g. protective doses of folic acid may be given intramuscularly with methotrexate).

The technique itself enables the smallest effective dose to be infused into the tumour area with minimal spill-over into the systemic circulation. A later development by Harrison is to use hypothermia, cooling the patient during the infusion. This gives added protection to the bone marrow and brain.

Before deciding to give chemotherapy, one must balance the prognosis of the disease against the expected relief and possible toxic side-effects. There is no doubt that, although it cannot claim a cure, it can afford marked relief of pain and be useful palliation. But its future use may well be in combination with surgery and radiotherapy rather than as a sequel to them.

CHAPTER 28

THE LARYNX

FOREIGN BODY IN THE LARYNX

IMPACTION of foreign bodies in the larynx is more likely to occur in children than in adults. The first symptoms are those of acute laryngeal obstruction; they are usually transient, and very rarely does death from asphyxia result. These symptoms are succeeded by those of irritation. There is pain, persistent cough, and often expectoration of blood-stained mucus. From time to time there are paroxysms of dyspnoea. Cardiac arrest and death may occur.

Treatment.—If the foreign body cannot be dislodged by hooking it out with a finger, or by inverting a child and slapping its back, immediate tracheostomy is necessary in urgent cases with obstructive symptoms. In less urgent cases radiography is indispensable when the foreign body is opaque to X-rays. If any difficulty arises, direct laryngoscopy should be performed, and by its aid the object can be seized and removed.

ACUTE ŒDEMA OF THE GLOTTIS

Pathology.—There is œdema of the entrance of the larynx, especially of the aryepiglottic folds and the epiglottis. Strictly speaking, the œdema is not of the glottis (the chink between the vocal cords) but of the tissues at a higher level, but this ancient and inaccurate term is difficult to displace.

Ætiology

1. Extension of acute inflammation, especially acute streptococcal laryngitis or tonsillitis, diphtheria, acute parenchymatous glossitis (p. 495), and Ludwig's angina.
2. Angioneurotic œdema.
3. Irradiation either with or without perichondritis.
4. Trauma (e.g. car accidents).
5. Indirect irritants such as ingested corrosives, scalds or inhaled noxious gases.
6. Extension of an adjacent carcinoma.
7. Local dropsy (renal or heart failure).

The patient's voice is reduced to a hoarse whisper and some dysphagia may be present. Increasing dyspnoea occurs and frequently the œdema is sufficient to cause urgent stridor. If laryngoscopic examination is possible, the entrance to the larynx can be seen resembling the appearance of the cervix uteri (fig. 829). In obscure and less urgent cases, the urine should be examined for albumin.

Treatment.—Inhalation of medicated steam and spraying with a dilute solution of cocaine and adrenaline afford relief in early and mild cases. Systemic antihistamines (e.g. Phenergan) or cortisone are valuable¹. When dyspnoea is urgent, intubation (p. 637) or tracheostomy must be performed forthwith.



FIG. 829.—
Acute oedema
of the glottis—
it resembles the
cervix uteri.

TRACHEOSTOMY

The indications are as follows :

(a) *To relieve obstruction of the upper air passages*

1. Acute infections such as: acute laryngo-tracheo-bronchitis of children, acute epiglottitis of influenzal (*hæmophilus influenzae*) or virus origin. Laryngeal diphtheria.

2. Oedema of the glottis.

3. Bilateral abductor paralysis of the vocal cords following injury to the recurrent laryngeal nerves during thyroidectomy.

4. Tumours, particularly carcinoma of the larynx.

5. Chronic stenosis following tuberculosis or scalding.

6. Congenital webs or atresias.

7. Cut throat.

(b) *To improve respiratory function*, by reducing the anatomical dead space, and also enabling effective aspiration of bronchial secretions to be done.

1. Fulminating bronchopneumonia.

2. Chronic bronchitis with severe emphysema.

3. Stove-in chest injuries.

(c) *Respiratory Paralysis.*—It allows assisted or positive pressure respiration to be performed. Also secretions or inhaled foreign material (e.g. vomitus) can be aspirated.

1. Unconsciousness associated with head or facio-maxillary injuries.

2. Coma from other causes persisting for more than a few hours where there is difficulty in maintaining a free airway, e.g. barbiturate poisoning.

3. Bulbar type of poliomyelitis.

4. Tetanus. Many of these patients are, of necessity, heavily sedated, have trismus, and are in mortal danger because of inability to expectorate.

(d) *As a Preliminary to certain operations, particularly extirpation of the larynx.*

Operation.—In cases of dire emergency the operation has been performed successfully with nothing available except a penknife, or it may be possible to introduce a gum-elastic coude catheter through the glottic chink. If at all possible, intubation even with a small intratracheal tube, should be attempted. If successful, general anaesthetic can be administered and a hurried procedure transformed into a deliberate, calm operation. Intubation brings the abnormally low intrapleural tension to normal and prevents the occurrence of surgical emphysema and possibly pneumothorax, especially in children (see Mediastinal emphysema, p. 636). Insertion of an intra-

¹ In cases of laryngeal obstruction, never give morphine. A patient under the influence of morphine stops fighting for breath, seems peaceful, and not infrequently the nurse returns to find him dead (Chevalier Jackson).

tracheal tube also permits aspiration of secretions which have accumulated below the laryngeal obstruction. Intubation will also prevent spasm of the glottis.

If the services of a skilled anæsthetist are not available, local anæsthesia is employed, but in desperate cases none is required. The patient may be pinned in a blanket so that a sudden movement of the arms may not embarrass the surgeon. When preparations are complete, a rolled towel or a small sandbag is inserted beneath the shoulders, and an assistant keeps the head extended strictly in the midline (fig. 830).

The surgeon, standing at the right side of the patient, places his left index finger on the upper border of the cricoid cartilage, with the thumb and the second finger on either side of the trachea, and makes an incision vertically downwards for 1 to 1½ inches (2.5 to 3.75 cm.), dividing skin, fascia, platysma, pretracheal fascia, and passing between the infrahyoid muscles (fig. 831). (When circumstances permit, a horizontal skin incision mid-way between the cricoid and the suprasternal notch should be used, as subsequent healing is far more satisfactory.)



FIG. 830.—The position for tracheostomy.



FIG. 831.—Exposure of the trachea. The first ring of the trachea is always to be avoided, for its division is so likely to be followed by tracheal stenosis

the dilator is removed, and the surgeon keeps his finger on the tube while the assistant ties the attached tapes around the patient's neck. The inner tube¹ is then fixed in position, and one or two nylon or silk stitches are introduced if necessary. In cases of diphtheria, anti-diphtheritic serum is given, and, on account of myocarditis, the patient is nursed lying flat, with one pillow.

In the case of a less urgent tracheostomy all bleeding is stopped before the trachea is opened. The injection of a few drops of 2 per cent. cocaine before the trachea is incised prevents the bout of coughing that follows the insertion of the tube. When

If seen, the isthmus of the thyroid gland is divided between hæmostats. In an emergency, hæmorrhage is ignored. A cricoid hook (fig. 832) is then inserted under the cricoid cartilage and grasped in the left hand. The hook steadies the trachea and brings it to the surface of the wound. The trachea is incised with a scalpel, the second, third, and often the fourth rings being divided: the lower the tracheostomy, the less will be the liability to laryngeal stenosis. A tracheal dilator is inserted through the tracheostoma, the cricoid hook removed, and the edges of the tracheal wound are separated gently. In the case of diphtheria the surgeon places a swab over the wound so that the violent expiratory efforts which follow do not spray membrane, infected mucus, and blood over himself and his assistants. When respiratory efforts have become less violent, a tracheostomy tube on a pilot (fig. 832) is inserted into the trachea,

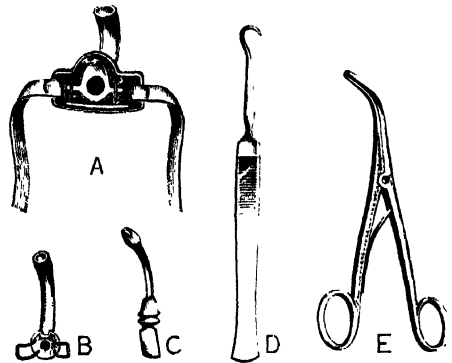


FIG. 832.—Instruments for tracheostomy (excluding scalpel and hæmostats).

A. Outer tube with tapes attached. B. Inner tube¹. C. Pilot. D. Cricoid hook. E. Tracheal dilator.

¹ The inner tube is longer than the outer tube, so that the latter cannot remain obstructed when the inner tube is removed for cleaning. To carry out suction effectively, the inner tube must be of such a diameter that it will not be occluded by the passage of a No. 3 or 4 rubber catheter; tracheostomy tubes between the sizes of 28 and 32 French catheter gauge fulfil these requirements.

the operation is performed on an adolescent or an adult, the isthmus of the thyroid gland is divided. The tracheostomy opening should be circular in shape (not just a vertical slit), by excising the edges of the incision with a scalpel strong enough to cut cartilage. A circular stoma facilitates the introduction and later changing of the tracheostomy tube, and heals well after eventual removal of the tube.

If a patient is unable to breath unaided, an inflatable rubber or polyethylene cuff-tube is introduced through the tracheostomy opening in order to seal off the air passage. The airway must be kept clear by frequent aspiration, assisted by postural drainage.

After-treatment.—Beside the bed is placed a trolley containing a tracheal dilator, duplicate cannulæ and introducer, retractors, a fine rubber catheter with a well-fitting syringe attached (to remove secretions from the trachea) and dressings. Oxygen is at hand. For the first few days a special nurse must be in constant attendance. A mechanical humidifier is essential in order to render the secretion less viscid. A sucker with a catheter attached should be at hand to keep the tracheo-bronchial tree free from secretions. The catheter must be kept sterile on a special tray covered by a sterile towel. The introduction of the catheter must be carried out under aseptic precautions by all concerned (nurses and physiotherapists). Unless these precautions are observed, secondary broncho-pneumonia infection is inevitable. When mucus is very tenacious, and consequently difficult to aspirate, normal saline or a detergent such as Alevaire (Bayer Products Ltd.) is administered through the tracheostomy by a fine nebuliser.

The inner tube is removed and washed in sodium bicarbonate solution every four hours, or more often if necessary, and in four to seven days the tracheostomy tube itself can usually be dispensed with, but before this is attempted the patient should be able to sleep with the tube occluded.

Complications:

1. *Crusting in the trachea* or main bronchi can seriously embarrass the airway, it can be cleaned by the aid of a bronchoscope passed through the tracheostome.
2. *Surgical emphysema* in the neck is a complication and may occur if the skin is too tightly sutured around the tube, or if the tube slips into the tissues of the neck (fig. 833).

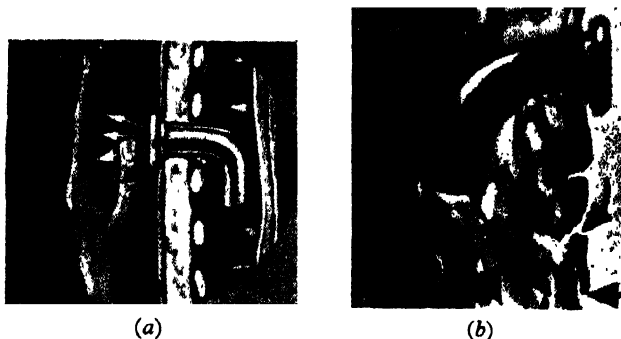


FIG. 833.—(a) Tracheostomy tube in the correct position. Testing for a free airway. (b) The tube has slipped, or has been incorrectly positioned, so causing surgical emphysema.

3. *Mediastinal Emphysema.*—The cause is an abnormally low intrapleural tension—air is sucked into the tissue planes during the operation before the trachea is opened. In severe examples the air in the mediastinum causes the mediastinal pleura to rupture, and a pneumothorax results. Should dyspnoea and cyanosis occur *after* tracheostomy has been performed, and the airway is free, one should think of the possibility of mediastinal emphysema, the diagnosis of which can be confirmed by radiography. Apart from oxygen therapy in high concentration, there is no specific treatment. The extravasated air is slowly absorbed.

Endotracheal catheterisation is a substitute for tracheostomy (mainly cases in group (c), p. 634), but it is usually less desirable, for two reasons:

(a) Repeated bronchial tree toilet is performed more easily by a nurse through a tracheostomy. (b) Intubation granuloma of the vocal cords (p. 639) and subsequent stricture may occur.

LARYNGEAL PARALYSIS

The muscles of the larynx are innervated by the recurrent laryngeal nerves, with the exception of the crico-thyroid muscle which is supplied by the superior laryngeal nerve. Lesions of the recurrent laryngeal nerve cause the vocal cord on the affected side to lie in the paramedian position; this is due to the unopposed tensing and adducting action of the crico-thyroid. Lesions of the vagus nerve above the origin of the superior laryngeal nerve will cause complete vocal cord paralysis on the affected side. The cord will be flaccid and lie midway between abduction and adduction, which is the state and position of the vocal cords soon after death.

Etiology.—The lesion may be central, cervical, or mediastinal. Of over-riding importance is the relation of a goitre, and especially of thyroidectomy, to recurrent laryngeal paralysis. Routine laryngoscopic examination must be made before thyroidectomy, as 3 to 5 per cent. of patients are found to have paresis or paralysis of one vocal cord, possibly due to neuritis following exanthemata during childhood, although no symptoms point to such a lesion. Pre-operative laryngoscopy is especially necessary when operating on a case of recurrent goitre. If pre-operative paralysis of a vocal cord is found, this fact *must* be recorded in the patient's notes in order to protect the surgeon from possible litigation. Pre-operative paralysis of a vocal cord with symptoms, e.g. a recent husky voice, is highly suggestive that the goitre is carcinomatous. Other causes of recurrent laryngeal paralysis are a central lesion (e.g. tabes), carcinoma of the upper œsophagus, carcinoma of the bronchus, malignant disease of the mediastinal lymph nodes, aneurysm of the arch of the aorta (always left-sided), and peripheral neuritis. Thirty per cent of cases are idiopathic.

Clinical Features.—**Unilateral recurrent laryngeal palsy** of sudden onset produces a whispering voice and occasionally some slight difficulty in



FIG. 834.—Normal larynx on inspiration. Indirect laryngoscopy.



FIG. 835.—Left recurrent nerve laryngeal palsy.



FIG. 836.—Bilateral recurrent nerve laryngeal palsy.

swallowing fluids, due to paralysis of the crico-pharyngeus on the affected side. These symptoms are short-lived and the voice may return to normal within a few weeks as the muscles in the opposite cord compensate and move

it across the midline to meet the paralysed cord. Owing to this efficient compensation, in slowly progressive lesions, the patient may only experience slight weakness of the voice towards the end of the day.

Bilateral recurrent laryngeal palsy is an occasional and very serious complication of thyroidectomy. Acute dyspnoea occurs due to the paramedian position of the cords which tend to get sucked together on inspiration. Unless tracheostomy or intubation is carried out forthwith, death from asphyxia is probable.



FIG. 837.—Bilateral flaccid paralysis of the vocal cords. The cadaveric position.

Unilateral complete laryngeal paralysis causes a hoarse voice in which compensation does not readily occur owing to the flaccid state of the vocal cord and its lateral position. The healthy vocal cord has difficulty in meeting it.

Bilateral complete laryngeal palsy is an uncommon condition which occurs in lesions of the brain stem. It is usually associated with other cranial nerve lesions.

Treatment.—Tracheostomy should be performed in all cases of bilateral lesions, even when the paralysis is flaccid, for it is far better to provide the patient with a free airway than to permit him to suffer from chronic dyspnoea and its attendant evils, chief of which is the risk of asphyxia. The use of a tracheostomy tube with a recessed valve allows the patient to speak: the delicate valve opens on inspiration and closes on expiration. The expired air passes through the vocal cords so that the patient has an audible voice. With an ordinary tracheostomy tube the inspired and expired air by-passes the larynx and the patient's voice amounts only to a whisper. In cases following thyroidectomy where the patient is otherwise in good health, the next step is to wait up to a year in the hope that one or both of the nerves will recover.

Unilateral extralaryngeal arytenoidectomy (de Graaf Woodman's operation) gives permanent relief of stridor at the expense of a good voice. As the abducted cord remains immobile, there is inevitable air-waste and the voice is not strong. The vocal cord is approached through an incision along the anterior border of the sternomastoid. The perichondrium and the underlying thyroid cartilage is incised along its posterior border. After the arytenoid cartilage has been displayed an unabsorbable suture is passed through the submucosa to include the vocal process of the arytenoid; with the exception of this process, the whole of the arytenoid cartilage is excised with nibbling forceps. The long ends of the suture are carried around the inferior cornu of the thyroid cartilage and tied. Thus the vocal cord on that side is maintained in an abducted position. The wound is closed with drainage. A tracheostomy tube is introduced, which, in favourable cases, can be removed three weeks later, and the wound is closed with drainage.

TUBERCULOSIS OF THE LARYNX

Rarely is the larynx the seat of primary tuberculosis. Occasionally the first symptoms are laryngeal, but a chest X-ray and the presence of tubercle bacilli in the sputum demonstrate that the primary focus is in the lungs, and in every case the laryngeal mucosa becomes infected by the sputum. Since the introduction of antibiotics the incidence of this disease has greatly diminished.

Stage 1.—The earliest symptoms are attacks of aphonia and hoarseness, and when accompanied by periods of dysphagia, especially for fluids, for which there is no pharyngeal cause, the probability of tuberculosis of the larynx is strong. The earliest

aryngoscopic sign is that the mucosa is pallid from œdema ; interarytenoid swelling, due to inflammation of the network of submucosal lymphatics, is very characteristic.

Stage 2.—Ulceration of one or both vocal cords and the epiglottis occur, giving a 'mouse-nibbled' appearance. Pain on swallowing fluids is increased, and it is often referred to the ears. Cough becomes troublesome, and painful.

Stage 3 is characterised by perichondritis and necrosis of the arytenoid cartilages, the epiglottis and, rarely, of other laryngeal cartilages. The voice becomes weak, coughing is almost incessant, and pain is a distressing feature.

Prognosis.—The prognosis of this once almost fatal disease has much improved since the introduction of treatment with streptomycin and tuberculostatic drugs (p. 23). Recovery is dependent largely on the state of the lungs.

Treatment.—Beside the usual treatment for the pulmonary tuberculosis, the complete cure of laryngeal tuberculosis depends upon *resting the voice*. The patient should have a pencil and writing-pad always to hand. As the first effect of successful treatment is disappearance of pain on swallowing, the patient's nutritional state usually improves rapidly. Œdema of the glottis may call for tracheostomy.

LARYNGEAL SYPHILIS

Secondary syphilitic manifestations are sometimes noted in the larynx. The mucous membrane becomes congested and mucous patches form. The voice is husky. Syphilitic laryngitis clears up under anti-syphilitic treatment.

Tertiary Syphilis.—A gummatous infiltration attacks the epiglottis and may implicate all the structures of the larynx. Necrosis of cartilage is liable to follow. Usually the only symptom is hoarseness, the voice during the remissions being particularly raucous. In contradistinction to tuberculosis, pain is singularly absent. The process is arrested by anti-syphilitic treatment, but subsequent scarring may cause such a degree of laryngeal stenosis as to require a permanent tracheostomy.

SIMPLE SWELLINGS OF THE LARYNX

1. **Vocal Cord Polypus.**—This is the most common of the simple swellings in the larynx and must be distinguished from true benign neoplasms. It originates in the sub-epithelial space within the vocal cord. Initial congestion is followed by localised areas of œdema and hyalinisation ; a soft, pearly grey often pedunculated mass is formed. It is easily removed endoscopically with cupped forceps.

2. **Intubation granuloma** may arise as a rare complication following the use of endotracheal anæsthetic tubes. As a result of static pressure of the tube, particularly during long operations, small non-specific granulomas appear in the region of the vocal processes of the arytenoids. Treatment is by prolonged voice rest initially, but in persistent cases careful endoscopic removal is recommended.

3. **Laryngocele.**—A laryngocele is a unilateral (occasionally bilateral) narrow-necked, air-containing diverticulum resulting from a herniation of laryngeal mucosa. It originates in the laryngeal sacculus, a vertical blind pouch situated in the anterior third of the laryngeal ventricle, and ascending between the false cord and the ala of the thyroid cartilage, and it herniates through the gap in the thyrohyoid membrane, which gives entry to the superior laryngeal vessels. Once the sac has found its way through this foramen, it enlarges comparatively rapidly, and when distended forms a visible, often resonant, swelling in the neck (fig. 838). Cervical air-pouches are present in many mammals, and can be inflated voluntarily; they are exceptionally well developed in South American monkeys that utilise them for howling (howling pouches). The condition, therefore, can be looked upon as partly atavistic but mainly acquired, for it occurs more frequently in professional trumpet-players, glass-blowers, and in persons with a chronic cough, than in others. The symptoms, due to a recrudescence of infection, come in attacks when



FIG. 838. — Laryngocele during an 'attack'.

the swelling, which often appears when the patient blows his nose, does not abate completely for hours or days; the explanation being that the neck of the sac becomes obstructed by mucopus. The voice is hoarse during an attack, which often terminates with a gurgling noise and discharge of mucus into the pharynx. Shortness of breath accompanies the attack, and, rarely, rapid distension of a laryngocele is a cause of sudden death from asphyxia.

Treatment.—For reasons given, the sac should be excised from its fundus to its neck, which is crushed, ligated, divided with a diathermy knife, and invaginated like the stump of a vermiform appendix. The wound is closed with drainage.

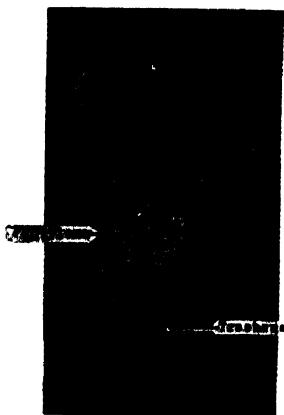


FIG. 839.—Papilloma of the vocal cords of a child.

NEOPLASMS OF THE LARYNX

Benign.—**Papilloma** is the commonest benign tumour of the larynx.

In an adult a papilloma is usually single, and its pedicle is attached to one of the true or false vocal cords. The symptoms to which it gives rise are similar to those of carcinoma of the larynx, from which it must be distinguished. The diagnosis is made by laryngoscopic examination. Rarely, a papilloma becomes malignant; therefore the papilloma should be removed and submitted to microscopical examination.

In a child the growth is relatively common: it is usually more vascular and softer than a papilloma appearing during adult life. Moreover, implantation growths soon appear in the vicinity and may obstruct the glottis (fig. 839). There is a marked tendency to recurrence after removal, and the papillomas may spread to almost any site in the larynx, pharynx, or trachea.

Treatment.—Chevalier Jackson's warning was not to be too radical in the treatment of multiple papillomas for fear of damaging the vocal cords. "Laryngeal papilloma," he said, "is a self-limiting disease and disappears spontaneously in early adult life provided the patient can be carried through until that time." Endoscopic removal with cupped forceps is the usual method of treatment. Painting the papillomas with podophyllin (as for the treatment of warts and condylomas) is employed by some, while others use stilbæstrol paint to speed up keratinisation, as normally occurs in the vagina at puberty.

Angiofibroma is always single, and is distinguished from a papilloma by its smooth contour (fig. 840). Except that occasionally it gives rise to hæmoptysis, it resembles a papilloma in symptomatology. In appropriate cases the condition must be distinguished from *singer's nodules*, which are nearly always bilateral. The latter condition, which produces a pearly-white nodule on the free edge of the vocal cord, is not a neoplasm, but an epithelial hypertrophy, and should, if possible, always be treated by prolonged rest (which is sometimes successful) before resorting to operation. On the other hand, an angiofibroma should be removed endoscopically with cupped forceps. Great precision is necessary, because if normal tissue is removed the speaking voice will be impaired and the singing voice ruined.



FIG. 840.—Angiofibroma of the left vocal cord. It seldom becomes much larger than depicted here.

Malignant.—Carcinoma is more common than an innocent tumour of the larynx. It usually occurs between forty and sixty years of age, and men are ten times more often attacked than women.

There are three varieties of laryngeal carcinoma:

1. *Glottic* (70 per cent. of growths), arising from the true vocal cord, which is relatively common and the most favourable type.
2. *Subglottic* (10 per cent.), below the vocal cords, with a worse prognosis.
3. *Supraglottic* (20 per cent.), originating from the ventricular bands (false vocal cords), laryngeal ventricles or the root of the epiglottis. This group has the worst prognosis.

1. **Carcinoma of a vocal cord** usually arises from the anterior half of one of the true vocal cords. Most frequently it is of the papillary variety (fig. 841), occasionally it is flattened, rarely it is ulcerative. Due to the paucity or absence of lymphatic vessels of the vocal cords, this type of carcinoma of the larynx remains locally malignant for a long period.

The first symptom is huskiness of the voice. The huskiness is progressive, and the patient can speak only in a low whisper, which finally gives place to aphonia. About this time the growth breaks through its confines, and metastases occur in the cervical lymph nodes and elsewhere.

The diagnosis is made by laryngoscopic examination and biopsy, and every patient with hoarseness persisting for more than three weeks should be submitted to this form of examination. According to the length of time the growth has been present, *four stages* of the disease are recognised:

1. The growth is confined to a *still mobile* vocal cord.
2. Infiltration impairing mobility of the cord. Extension to other cord.
3. Fixation of the cord. The growth has entered adjoining part of larynx. Isolated involved lymph nodes.
4. Extension to the pharynx or skin. Lymph node metastases with fixation.

2. **Subglottic carcinoma** is a less common variety that occurs beneath the vocal cords. In this site the neoplasm grows steadily and silently, until dyspnoea develops. Unlike carcinoma of the vocal cord, metastasis is not long delayed. The first lymph node to be involved is that on the crico-thyroid membrane, the lymph node of Poirier. Spread then continues to the paratracheal and lower deep cervical nodes. The thyroid gland is often involved by direct extension of the growth.

3. **Supraglottic Carcinoma.**—The initial symptom is often a sense of discomfort in the larynx. Pain and hoarseness came relatively late. In 60 per cent. of cases there is cervical lymph node involvement at the time of presentation.

Tomograms are very helpful to determine the extent of the growth (fig. 842).

Treatment of Carcinoma of the Larynx :

The most important anatomical fact on which treatment and prognosis of laryngeal carcinoma depends is the uneven distribution of laryngeal lymph vessels.



FIG. 841.—Carcinoma of a true vocal cord.

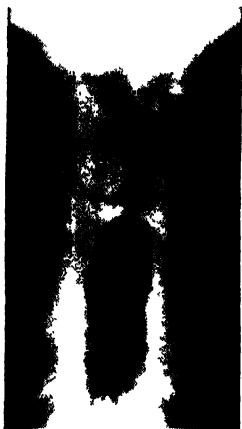


FIG. 842.—Tomogram of the larynx showing tumour of the right false vocal cord encroaching on the laryngeal ventricle. The vocal cords are not affected.

Whilst the true vocal cords have relatively few lymph vessels, the lymphatic drainage of the laryngeal aditus and the subglottic region is abundant. Because of this rich lymphatic drainage supraglottic tumours metastasise early into the cervical lymph nodes, whereas tumours of the true vocal cords remain locally malignant for many months.

For early (stage 1 and 2) carcinoma of the true vocal cord, the results of irradiation are as good as those of surgery and the voice is better. Therefore, where adequate facilities exist, irradiation (super-voltage X-ray therapy, or telecobalt) is preferable to surgery. Although in the stage 1 growths there is an 80 per cent. five years' survival rate, every effort should be made to have a careful follow-up. If recurrences occur, or where the cervical lymph nodes are involved by metastatic deposits of squamous-celled carcinoma, laryngectomy, with block dissection of the lymph nodes in continuity, gives results superior to irradiation.

Total laryngectomy is performed for carcinoma of the vocal cord which is already fixed by infiltration, when radiotherapy fails to destroy the growth or where the growth recurs after radiotherapy.

Anæsthesia.—General anæsthesia is given through an endotracheal tube inserted via the nose. This is changed by the surgeon during the later stages of the operation after delivery of the larynx (fig. 844B).

Incision.—A variety of incisions are used. A satisfactory exposure is shown in fig. 843. This may be modified if a block dissection is necessary.

Dissection.—The laryngeal cartilages and trachea are exposed by division of the thyroid isthmus. The strap muscles are divided and the inferior constrictor is carefully dissected off the thyroid cartilage. The hyoid is freed. The pharynx is opened by incisions through the thyrohyoid membrane (fig. 844A). After changing the anæsthetic tube, the hyoid is drawn forward and the mucous membrane incised down each pyriform fossa, to

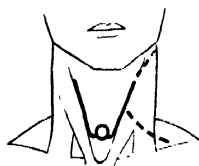
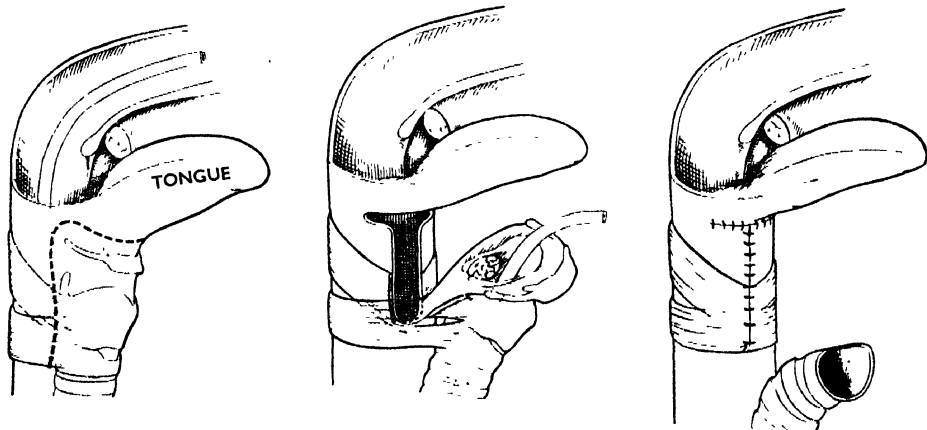


FIG. 843.



B

FIG. 844.—Total laryngectomy (see text).

meet along the back of the arytenoids (fig. 844B). A nasal feeding tube is passed into the oesophagus and the defect in the pharynx is closed by interrupted sutures, and reinforced by suturing the inferior constrictors together anteriorly (fig. 844C).

The trachea is divided between the second and third rings. The tracheostomy is fashioned by suturing the trachea to the cut edges of the wound in the suprasternal notch.

For supraglottic carcinoma, subglottic tumours, and in cases where cervical lymph nodes are obviously the sites of secondary deposits, block dissection of the lymph nodes must be combined with the laryngectomy. If the carcinoma extends into a lobe of the thyroid gland, this lobe is also removed.

Speech after Laryngectomy.—Many younger patients learn oesophageal speech, belching swallowed air for this purpose. Others require a mechanical larynx which can be fitted to the tracheostomy tube. Another type of electrically operated *oral vibrator* attached to the patient's upper denture or an artificial palate has been invented recently.

Fenestration with interstitial radium may be employed where no super-voltage therapy exists, with a high proportion of favourable results. The vocal cord must be still mobile.

The larynx is exposed by a J-shaped incision commencing above the level of the hyoid bone, and ending below the lower border of the cricoid cartilage. The flap is raised. The infrahyoid muscles are split, and a large window is cut in the ala of the thyroid cartilage, without damaging the perichondrium or opening the underlying mucosa. The object of removing the cartilage is to allow radium needles to come in closer proximity to the growth, and minimise radium necrosis of the cartilage—a great disadvantage of radium. The radium needles are left in place seven to nine days (fig. 846). Subsequently the voice is but little impaired.

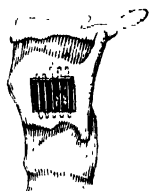


FIG. 846.—Alar fenestration and insertion of radium needles.

Laryngo-fissure and Excision of the Growth.—The vocal cord must be still mobile. Under local infiltration, a preliminary tracheostomy is performed. The thyroid cartilage is bisected in the middle line and the crico-thyroid membrane opened. The perichondrium in the region of the affected vocal cord is raised by blunt dissection, and the whole vocal cord, with a margin of healthy tissue, is removed.

The larynx is repaired, and the tracheostomy tube is removed after a few days. As the vocal cord becomes replaced by fibrous tissue, which will be immobile and may not form exactly on the same plane as the remaining vocal cord, the patient will have a gruff but useful voice.



FIG. 845.—Patient after complete laryngectomy. A transverse skin incision was used in this case. (Jackson and Babcock.)

CHAPTER 29

THE THORAX

WILLIAM P. CLELAND

ANATOMY OF THE BRONCHIAL TREE

A THOROUGH knowledge of the anatomy of the bronchial tree is essential, not only for the diagnosis of many chest conditions, but for the planning and execution of the majority of pulmonary operations.

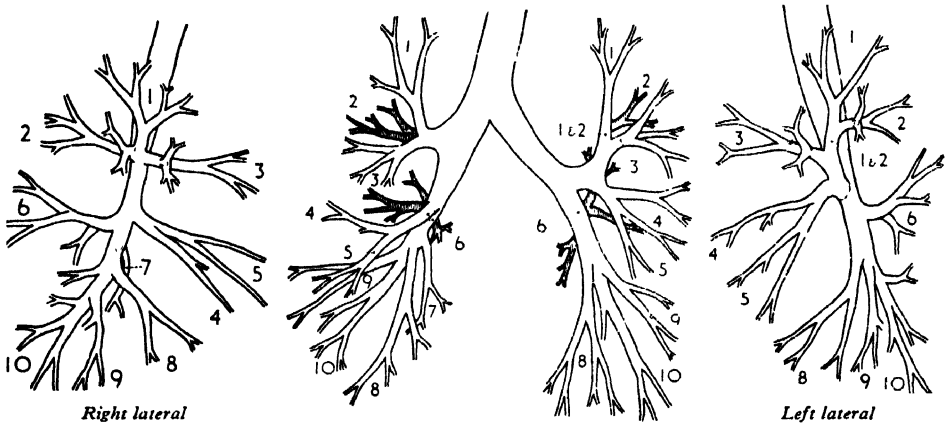


FIG. 847.—The bronchial tree with internationally adopted nomenclature.

RIGHT LUNG

- | | |
|---------------------|--------------|
| 1. Apical | |
| 2. Posterior | Upper Lobe. |
| 3. Anterior | |
| 4. Lateral | Middle Lobe. |
| 5. Medial | |
| 6. Apical | |
| 7. Medial (Cardiac) | |
| 8. Anterior basal | Lower Lobe. |
| 9. Lateral basal | |
| 10. Posterior basal | |

LEFT LUNG

- | | |
|----------------------|-------------|
| 1. Apical | |
| 2. Posterior | |
| 3. Anterior | Upper Lobe. |
| 4. Superior lingular | |
| 5. Inferior lingular | |
| 6. Apical | |
| 8. Anterior basal | Lower Lobe. |
| 9. Lateral basal | |
| 10. Posterior basal | |

Fig. 847 illustrates the common pattern of the bronchial tree. Each bronchus is accompanied by a branch of the pulmonary artery and together they form a broncho-vascular pedicle supplying conical segments of lung tissue. The pulmonary veins lie between the segments with tributaries issuing from adjacent segments. Each segment is largely an independent unit which can be removed (segmental resection) without interfering with adjacent segments. The veins indicate the true position of the intersegmental plane, which must be followed accurately during the operation. Two or more bronchial arteries on each side enter the lung in the areolar tissue surrounding the bronchi. They arise from the descending thoracic aorta and supply the broncho-pulmonary tissues with arterialised blood. They develop links with the smaller pulmonary vessels which, in certain diseases, may enlarge considerably.

The trachea and main bronchi with their lobar and first segmental divisions can all be examined visually by bronchoscopy. This investigation will demonstrate abnormalities in size (stenosis, obstruction or dilatation), movement (immobility or spasm), shape or position. The examination will also reveal the presence of a tumour or an intrabronchial foreign body (fig. 848). Material for biopsy and secretions for cytological or bacteriological examinations can be obtained. Only the proximal parts of the bronchial tree can be examined, even with the aid of the telescope, so that the examination is of limited value in the investigation of peripherally situated lesions.



FIG. 848.—Chondroma:
Right main bronchus.
Bronchoscopic photograph.
(Dr. P. Stradling, London.)

A more detailed anatomical study can be made by bronchography, which consists of the introduction of radio-opaque material (iodised oil) into the bronchial tree. An attempt is made to fill all the branches of the bronchial tree on one side, and radiographs are taken in two or more planes. Bronchography provides information about the size and distribution of the bronchi, and will also demonstrate bronchial occlusion or narrowing.

Developmental Anomalies

The trachea develops as a bud from the primitive fore-gut which soon divides into two main branches—the right and left main bronchus. Each bronchus continues to develop by branching so that all generations are present by the sixteenth week of intrauterine life. The fully developed segment has approximately 20 to 25 generations of branches. Failure of a main bronchus to develop results in *unilateral agenesis*, whilst failure of a lobar bronchus to develop produces *lobar agenesis*. Subsequent aberrations of development result in variations of the segmental pattern. Each developing bronchial bud becomes closely associated with the future pulmonary artery which covers its surface. Failure to link up with the pulmonary artery system occurs occasionally. In this case, ectopic bronchial buds develop anomalously, producing a mass of bronchial elements which often lose their connection with the parent bronchi. Vascular supply is derived direct from the descending aorta. In adult life the condition presents as a bizarre cystic mass, usually lying in the posterior aspect of the lower lobe with large systemic arteries the size of the radial artery or even larger entering the mass through the pulmonary ligament—*intralobar sequestration*. Infection of the cystic spaces is common and is the usual cause of symptoms. Removal of the lower lobe with the offending mass is recommended.

INJURIES TO THE CHEST

Apart from road accidents chest injuries are not common in civil practice; in war, however, they constitute nearly 10 per cent. of all wounds, whilst of those killed in battle, 25 per cent. have chest injuries. They are often associated with injuries elsewhere, particularly the head and abdomen. Two main varieties are encountered—crush injuries and wounds—although in any particular patient both may be present.

Crush Injuries

These are common both in war and in civilian practice. The injury may be produced by a localised blow or a more extensive crushing force. Road accidents frequently result in severe crush injuries. Similar injuries can also

be produced by concussion waves from explosions conveyed either through air or water (blast injuries).

The injuries may produce contusion of the chest wall, rib fractures, a stove-in chest, a flail chest, lung contusion or laceration.

The effects of a crush injury depend on its severity and the extent of involvement of chest wall, pleura and lung. The more severe forms interfere seriously with ventilation and coughing either from pain or from instability or deformity of the chest wall. The presence of blood or secretions in the bronchial tree makes ventilation still less effective. Respiration may, in addition, be depressed further by an associated head injury.

The serious interference with ventilation results in rapid and shallow respiration with cyanosis, tachycardia and hypotension. Respiration may be noisy from tracheo-bronchial secretions. Milder degrees of anoxia produce cerebral confusion and restlessness, and this is followed by unconsciousness in severe cases.

Management in the early stages depends on a careful assessment of the damage. The chest wall is examined for paradoxical movement (multiple fractured ribs) or depressions (stove-in chest). The posterior aspect of the chest should be examined as the patient is gently rolled on one side. The position of the mediastinum (trachea and apex beat) provides useful information concerning the presence of a pneumothorax, a hæmothorax or massive collapse of the lung. An early chest X-ray is imperative for accurate diagnosis of the intrathoracic damage.

Treatment is directed at improving ventilation by the following means where indicated:

(1) Relief of pain with morphine or other analgesics, or intercostal nerve block.

(2) Stabilisation of the chest wall: (a) strapping can be used in mild cases and in emergencies; (b) internal fixation of fractured ribs by wire or nails for more severe cases; (c) musculo-fascial chest wall traction with a Steinmann pin is an effective and simple way of elevating a stove-in chest or stabilising a flail chest (fig. 849).

(3) Removal of pleural blood or air should they be large enough to compress the lung. Simple aspiration may be sufficient, but rapid reaccumulation may demand an indwelling tube or thoracotomy.

(4) Removal of tracheo-bronchial secretions may be effected by natural means after relief of pain. Bronchoscopic aspiration may be used in an emergency but if secretions persist a tracheostomy is advisable.

(5) *Tracheostomy* is the most important measure in all serious cases (see also p. 634). It permits effective and repeated aspiration of the tracheo-bronchial tree. By eliminating the dead space of the oropharynx it reduces the ventilatory demands and the work of respiration. It reduces or eliminates paradoxical chest wall movements.

Tracheostomy alone will often transform the condition of a seriously ill patient. If ventilation is seriously depressed, artificial respiration can be applied through the tracheostomy tube by mechanical respirators.

Tracheostomy and artificial respiration are of particular value in serious multiple injuries, especially those involving the head, in addition to the chest.

(6) Oxygen is usually necessary and is best administered by a tent or mask.

(7) Blood transfusion, fluid replacement and hypertensive agents may be required to maintain the circulation.

Fractured Ribs.—Isolated rib fractures due to direct violence are a common complication of many varieties of chest trauma. The condition is painful but rarely disturbing. Localised tenderness and crepitus are often elicited, but the fractures may be difficult to demonstrate radiologically. Local support and analgesics are often all that is required for treatment.

Multiple rib fractures (indirect or burst) are produced by crushing. The ribs fracture at the site of maximum curvature and stress, at the anterior and posterior angles. The stability of the chest wall is often greatly disturbed and in addition to pain, paradoxical movement occurs with difficulty in breathing and coughing. Minor degrees of paradox can be controlled by strapping, but the more extensive varieties are best treated by chest wall traction (fig. 849)

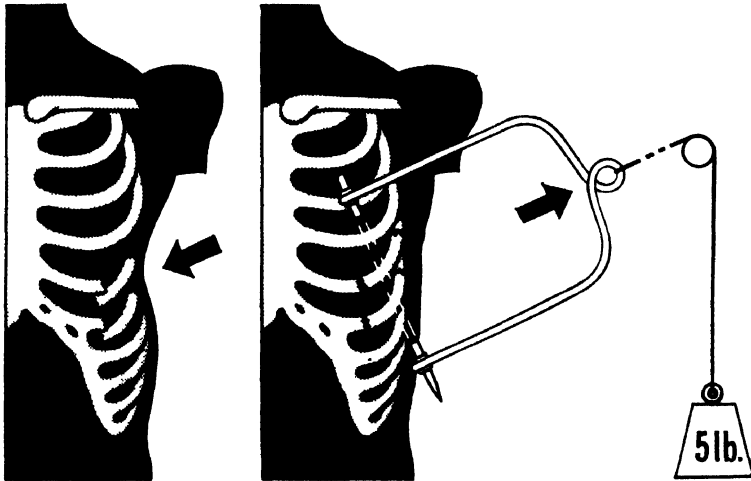


FIG. 849.—Chest wall traction with Steinmann pin. The pin is passed over the outer surface of the fractured ribs deep to the pectoralis major and serratus magnus muscles. (After P. Jewsbury.)

or by exposure of the fractured ribs and fixation with wire or nails (see flail chest). Pain unrelieved by analgesics should be controlled by intercostal nerve block.

Stove-in Chest.—This severe injury is produced by an extensive but localised crushing force which produces multiple rib fractures and a permanent indentation of the chest wall. The affected hemithorax may be considerably reduced in size and the function of the underlying lung markedly reduced. Some paradoxical movement is usually present. The compression of the underlying lung and its relative immobility results in the accumulation of broncho-pulmonary secretions.

The minor degrees of stove-in chest may require little specific treatment additional to that already outlined for rib fracture, but the more severe forms

require surgical correction. Under anæsthesia it is possible to elevate the depressed chest wall with towel clips placed around the ribs. If the deformity tends to recur, the ribs must be fixed to an external 'cage' with heavy stainless-steel wire loops passed around several ribs. Fixation will be required for several days.

Crushed chests associated with a fractured clavicle are particularly serious as the deformity will increase unless the clavicle is controlled with a plate or pin.

Flail Chest.—This type of injury is seen particularly after automobile accidents of the more severe type, and is often accompanied by other serious injuries.

Multiple rib fractures result in a flaccid unstable chest wall exhibiting paradoxical movement in which the flail area is sucked inwards during inspiration and pushed out in expiration. With quiet respiration paradoxical movement may not be apparent, but if the respirations are difficult or if the patient should cough the paradox becomes more obvious. The paradoxical movement produces imperfect ventilation with anoxia whilst defective coughing results in the retention of secretions. Any attempt on the part of the patient to overcome these defects by over-breathing or coughing only aggravates the condition.

Tracheostomy is urgently required and will often result in dramatic improvement. The dead space of the mouth and pharynx is eliminated with a consequent reduction in the ventilatory requirements. The presence of a tracheostomy will permit ready and repeated removal of broncho-pulmonary secretions by a catheter, and a clear airway will similarly result in a quieter respiration. Paradoxical movement is often 'cured' by tracheostomy, but if it persists the affected ribs should be exposed and the ends joined by stainless-steel wire or nails, or chest wall traction employed (fig. 849).

With the acute ventilatory crisis adequately controlled by the tracheostomy, attention must be given to the prevention and control of the broncho-pulmonary infection by breathing exercises and antibiotics.

Traumatic Pneumothorax.—Air in the pleural cavity appears commonly after many forms of trauma. In the majority it is associated with blood as well (*hæmo-pneumothorax*). Air may reach the pleural cavity through a wound in the chest wall (*sucking wound*). More commonly air leaks from damaged lung; if the leak is valvular a considerable quantity of air will accumulate producing total collapse of the lung and displacement of the mediastinum towards the opposite side (*tension pneumothorax*).

Sucking wounds should be sealed with an occlusive pad until definitive surgical repair can be arranged. A tension pneumothorax can be relieved in an emergency by plunging an unmounted aspirating needle into the chest which allows the air under tension to escape. Subsequently, a small soft catheter should be introduced through a cannula and connected to a water seal or suction motor.

Traumatic Hæmothorax (see also p. 653).—Blood collects in the pleural cavity in the vast majority of chest injuries. The chest wall, lung or

heart and great vessels may be the source of the bleeding. Bleeding is usually limited and should be dealt with by repeated daily aspirations after an initial interval of twenty-four hours to allow the bleeding vessel to seal. If the accumulation is large, earlier aspiration is necessary but, if progressive, a thoracotomy will be required to control the bleeding.

Pulmonary Contusion and Laceration.—The lung is injured in the majority of cases of moderate and severe trauma. Contusion results in areas of consolidation which will usually resolve spontaneously. Laceration permits the leakage of blood and air into the pleural cavity. Minor lacerations heal spontaneously, but more severe degrees produce persistent collapse of the lung and leakage of air and should be explored by thoracotomy. Secondary infection of the damaged lung is an important complication which can often be prevented by chemotherapy, bronchial aspirations and physiotherapy.

Fracture of Bronchus.—Severe crushing injuries occasionally result in a transverse fracture of a bronchus. The commonest site is at the junction of the relatively fixed mediastinal bronchus and the more mobile hilar bronchus. Total atelectasis and mediastinal surgical emphysema result. The condition can be confirmed at bronchoscopy. Early repair should result in a functioning lung but delay will lead to irreversible infection and fibrosis.

Wounds of the Chest

These may be simple, or complicated by serious underlying visceral damage. In either case the wound may be penetrating or perforating (through and through wound). In all cases, however simple, it is necessary to exclude serious damage to either thoracic or abdominal viscera.

Simple wounds from stabs or bullets may give rise to little trouble—the external wound is small and usually clean. A hæmothorax is usual and will require aspirations until the pleural cavity is dry.

Complicated wounds are common in injuries with large missiles such as shell and bomb fragments. The chest wall damage may be considerable and fragments of clothing, etc., may be carried into the chest. Lung contusion and laceration is common and the mediastinal structures, the diaphragm and abdominal viscera may be involved.

Operative treatment will be required in the majority of cases, and should be carried out as soon as practicable after resuscitation. The only indication for emergency thoracotomy is uncontrollable internal or external bleeding.

Endotracheal anaesthesia is essential in order to ensure adequate ventilation and permit the removal of bronchial secretions during the operation. The chest wound is excised, rib fragments removed, and intercostal vessels ligated, if necessary. If the wound is conveniently situated, the pleural cavity can be explored through it, but if not, a separate thoracotomy is required. All blood, clot, and debris should be removed from the pleural cavity in addition to any retained foreign bodies. Lung lacerations should be sutured if small, or a formal resection carried out if more extensive. The pleural cavity should be drained through a separate stab incision. After operation it is essential that the lung should expand rapidly and completely; this is encouraged by suction drainage, breathing exercises and early ambulation.

Thoraco-abdominal Wounds.—These are always potentially serious owing to the involvement of both the abdomen and the chest; their importance depends on whether or not one of the hollow abdominal viscera is dam-

aged. The predicted course of the missile should be considered in order to anticipate the visceral damage. Apart from serious involvement of the chest, as outlined above, the main indications for exploration are signs of persistent bleeding or evidence of involvement of a hollow viscus. The thoracic approach has the advantage of permitting correction of the thoracic injuries, whilst giving adequate exposure of the upper abdomen through the diaphragm.

DISEASES OF THE CHEST WALL

Kypho-scoliosis.—Deformities secondary to pleural and pulmonary disease are frequent unless preventive measures are taken; they can cause a considerable loss of pulmonary function, and may lead to repeated respiratory infections and cardiovascular disturbances. Vigorous breathing exercises can do much to prevent serious deformity provided they are initiated sufficiently early, but even in established cases some improvement can be expected. Primary kypho-scoliosis is considered on p. 322.



FIG. 850.—Funnel chest deformity.

Tietze's disease is a not uncommon painful and non-suppurative swelling of the second or third costal cartilages. Examination of the spine may reveal an underlying (and ? causative) scoliosis. X-ray is negative. Reassurance and not exploration is required.

Funnel Chest (Pectus Excavatum).—This deformity consists of a depression of the body of the sternum and the xiphoid process, combined with an inward curving of the costal cartilages and adjacent ribs. The deformity is usually minimal at birth, but becomes progressively more obvious during childhood. It predisposes to repeated respiratory infections and to cardiovascular disturbances, whilst the cosmetic appearances are frequently embarrassing (fig. 850).

Correction of the deformity is carried out through a midline or transverse incision: the sternum and costal cartilages are exposed and the deformed costal cartilages are resected subperichondrially. The attachments of the diaphragm to the xiphoid process are detached and the sternum separated from the mediastinal tissues. A wedge osteotomy of the anterior table of the sternum is performed at the upper limit of the deformity, and the mobilised sternal body can then be easily elevated. The sternum should be maintained in an elevated position by a stainless steel strut which is placed beneath the sternum with the ends resting on the ribs laterally. The strut is removed after several months. The use of internal splinting has greatly improved the long-term cosmetic results.

Cold Abscess

The majority of cold abscesses of the chest wall are secondary to tuberculous intercostal lymphadenitis. A minority are associated with Pott's disease of the spine and tuberculosis of the ribs or sternum. The intercostal lymph nodes are situated posteriorly near the neck of the rib, or anteriorly in association with the internal mammary vessels. Tuberculous pus, forming in these sites, may track a considerable distance in the intercostal space before becoming subcutaneous. They usually reach the superficial tissues by following the lateral or anterior branches of the intercostal vessels, and are thus most commonly found in the anterior axillary line or the parasternal region. The abscess has all the characteristics of a tuberculous cold abscess. Involvement of the skin and rupture is common in neglected cases, resulting in a persistent discharging sinus surrounded by typical

tuberculous granulation tissue. A cold abscess must be distinguished from a lipoma or an empyema necessitatis; in the latter, the swelling is often tender and may exhibit an impulse on coughing, and is associated with the physical and radiological signs of an empyema.

The abscess should be treated by repeated aspirations through healthy skin with the instillation of streptomycin. If the abscess fails to respond to repeated aspirations, surgery will be required. The extent of the ramifications of the abscess should first be determined by the injection of iodised oil into the abscess. Ideally, the abscess and its ramifications should be completely excised, together with any secondary involved bone; but if this is not practicable, the abscess should be evacuated, granulation tissue removed and the wound closed. Chronically discharging sinuses can sometimes be cured by regular instillations of streptomycin; alternatively, excision of the track is advisable. All patients should have a long course of chemotherapy.

Empyema Necessitatis

This is caused by an empyema perforating the chest wall and presenting with a subcutaneous collection of pus communicating, often by a tortuous channel, with the main pleural collection. The condition is seen either with a neglected or an undiagnosed empyema, or following aspirations of thin, highly infective pus from an empyema. In the latter case, the superficial tissues are infected by the seepage of pus through a needle track. The site of the subcutaneous abscess does not always correspond to the site of pleural perforation, as the abscess may track along the intercostal spaces before becoming superficial. The signs are those of a diffuse, fluctuant, tender swelling which may exhibit an impulse on coughing, and is associated with the clinical and radiological signs of an empyema. Treatment is primarily aimed at the empyema, which should be aspirated and drained. The superficial abscess may disappear as a result of aspirating the empyema, or may itself require separate aspirations or drainage.

Tumours of the Rib

Rib tumours may be benign or malignant, and the latter either primary or secondary.



FIG. 851.—Chondroma of rib.

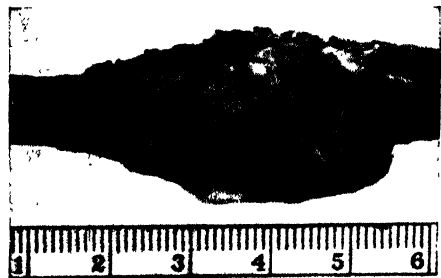


FIG. 852.—Chondroma of rib (same case as fig. 851).

Benign tumours are frequently discovered by Mass Miniature Radiography. Many are symptomless and produce no external evidence of their presence. A few produce pain or an external swelling. The commonest is the chondroma (figs. 851 and 852), whilst osteochondroma, multiple exostoses,

fibrous dysplasia, lipoid granulomas and multiple myelomas are less common. Benign tumours usually produce expansion of the rib and are less dense than normal rib, whilst pathological fracture is rare. Malignant change is not uncommon, particularly with the chondromas, and removal is advisable.

Primary malignant tumours are rare, the commonest being chondrosarcoma; they should be widely excised. *Secondary deposits* in rib are particularly common in carcinoma of the lung and with breast cancer. The deposits are almost always painful and tender. They result in destruction of bone, and pathological fracture is common. X-ray therapy will usually relieve the pain.

Neurogenic Tumours (see also p. 685).

These are of two main types—neurofibroma (figs. 853 and 854) arising from the intercostal nerves and ganglioneuroma from the sympathetic chain. The former appear close to the neck of the rib, whilst the latter lie slightly more medially applied to the vertebral bodies. The majority are found on routine radiography. They are usually symptomless and single. Their growth is slow and the adjacent ribs and vertebral bodies are often distorted and eroded. A small percentage extend through the intervertebral foramen into the vertebral canal, where compression of the spinal

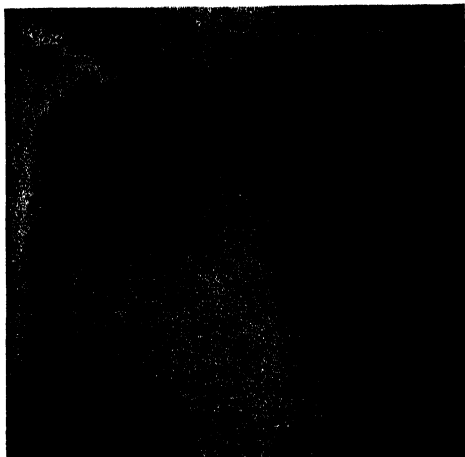


FIG. 853.—A large neurofibroma of the chest wall. The tumour is lying posteriorly.



FIG. 854.—Tumour removed from case illustrated in fig. 853.

cord may result in paraplegia (dumb-bell tumour). Removal of the tumour is safe and simple and is recommended owing to the risks of malignant changes. Dumb-bell tumours, and particularly those with spinal-cord involvement, require urgent treatment. A small intraspinal extension can easily be removed from the chest, but extensive intraspinal tumours will require laminectomy for their removal; the latter can usually be carried out at the same time as the chest operation, but if very extensive, separate operations are advisable.

DISEASES OF THE PLEURA

Pneumothorax—A pneumothorax is produced by the presence of air between the layers of the pleura. The air may be present alone or associated with serous fluid (*hydro-pneumothorax*), pus (*pyo-pneumothorax*), or blood (*hæmo-pneumothorax*). Air may be deliberately introduced into the pleura, in the treatment of pulmonary tuberculosis (*artificial pneumothorax*), or be associated with trauma (*traumatic pneumothorax*), or may appear without any obvious exciting cause (*spontaneous pneumothorax*). The physical signs of pneumothorax are a hyper-resonant percussion note with absent breath

sounds. Radiographs reveal translucency on the affected side with absence of lung markings; the edge of the collapsed lung is usually visible.

Spontaneous Pneumothorax.—Causes :

- | | |
|----------------------|-------------------------------|
| (a) Tuberculous. | (1) Emphysematous bullæ. |
| (b) Non-tuberculous. | (2) Solitary lung cysts. |
| | (3) Honeycomb or cystic lung. |
| | (4) Idiopathic. |

A tuberculous spontaneous pneumothorax is usually associated with obvious clinical or radiological tuberculosis. The condition is produced by the rupture of a small subpleural tubercle or cavity, and is usually associated with infection or irritation of the pleura resulting in the appearance of fluid in the pleural cavity and a febrile reaction. Treatment depends on the underlying pulmonary lesion.

A non-tuberculous spontaneous pneumothorax by contrast is rarely associated with fluid formation or fever. In a minority, an obvious underlying pulmonary lesion, such as an emphysematous bulla, a lung cyst, or a honeycomb lung, can be demonstrated. Absorption may occur spontaneously and rapidly, or may be considerably delayed (*chronic spontaneous pneumothorax*). Attacks may be single or repeated (*recurrent spontaneous pneumothorax*). With a first attack, in the absence of an underlying cause, air can be allowed to absorb spontaneously, or expansion can be encouraged by occasional aspirations. Should tension occur, however, air should be removed with a pneumothorax apparatus, but if reaccumulation is rapid, a fine catheter should be introduced into the pleural cavity and attached to an under-water seal or suction motor. A soft catheter is preferable to a rigid needle or cannula as it will not lacerate the lung as it expands.

Failure of absorption (*chronic spontaneous pneumothorax*), or repeated attacks (*recurrent spontaneous pneumothorax*), demand full investigations to determine the cause. The most useful investigations are tomography and thoracoscopy (inspection of the lung). These investigations may reveal :

- (a) A localised lesion, such as a cyst.
- (b) Generalised lesions, such as emphysema or honeycomb lung.
- (c) Nothing abnormal.

A localised condition is best treated by thoracotomy and excision, but the latter two groups should be treated by artificial obliteration of the space (pleurodesis). This is achieved by the use of chemical or other irritants (5 per cent. silver nitrate, iodised talc, 0.5 per cent. camphor in oil) introduced either through a needle or applied to the lung surface at thoracoscopy. These irritants set up a diffuse pleuritis which will result in the fusion of the visceral to the parietal pleura if the two surfaces can be maintained in apposition. It is thus important to ensure that the lung expands rapidly and completely after pleurodesis by using catheter drainage with suction. Thoracotomy is preferred by some for all cases. The lung leak is oversewn, cysts if present are excised and the parietal pleura at the apex is removed in order to ensure fusion of the lung to the chest wall and prevent recurrence.

Hæmothorax.—Blood in the pleural cavity may occur in a variety of conditions and is often associated with air as well. The respiratory and cardiac movements defibrinate the blood as it reaches the pleural cavity so that the collection remains fluid. Massive clotting only occasionally occurs.

Blood is a pleural irritant and its presence produces pain and shock in the early stages and later excites the formation of a considerable effusion. It is also an excellent culture medium and infection is relatively common.

Causes.—(1) Trauma (fig. 855).

(2) Post-operatively, following pulmonary, cardiac or œsophageal operations, thoracoscopy for division of adhesions, cervical sympathectomy and after refills of an artificial pneumothorax.

(3) Associated with new-growths of lung, mediastinum or pleura.

(4) Leaking aneurysms.

(5) Spontaneous.

The signs are those of a collection of fluid in the pleura, and the diagnosis is confirmed by exploring with a needle.

Treatment.—The initial treatment should be aimed at relieving pain, shock and blood loss. Aspirations of blood for

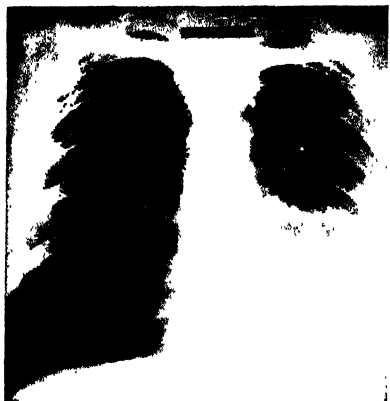


FIG. 855.—A small left hæmothorax with two retained metallic foreign bodies.

therapeutic purposes is only indicated in the early stages if respiration is embarrassed. If signs of persistent bleeding are present, thoracotomy is advised. In all other cases the aim should be to remove the blood by aspiration as early and as completely as possible. Aspirations can be safely started twenty-four hours after the onset and should be repeated daily until no more fluid is obtained and the X-rays appear clear. Air replacement should be avoided. Early and vigorous breathing exercises are advised.

A *clotted hæmothorax* is diagnosed when the aspirating needle fails to remove a collection of blood. Liquefaction of the clot is often possible with streptokinase or Trypsin ferments (Trypur). An initial dose of 200,000 units of streptokinase or 1 ml. of Trypur is injected into the clot and aspirations performed after twenty-four hours. Further doses can be given if the initial injection is not effective. In many instances however, the result is unsatisfactory, and thoracotomy with evacuation of clot and decortication of the lung is required.

An *infected hæmothorax* should be treated initially with repeated aspirations and the instillation of suitable antibiotics. Early drainage or decortication will probably be required, owing to the difficulty of sterilising and aspirating the hæmothorax if clotting has occurred.

Pleural Effusion.—The pleural cavity is constantly bathed in fluid which exudes from the visceral pleura and is absorbed by the parietal pleura. Any disturbance of the finely adjusted balance may result in the accumulation of fluid in the pleural cavity. Disturbances in osmotic or hydrostatic pressures produce a transudate (protein less than 3 gm. per cent.) ; inflammatory lesions produce an exudate (protein more than 3 gm. per cent.) and neoplasms produce variable changes.

Pleural fluid when first formed gravitates to the most dependent part of the chest (usually posterior costo-phrenic angle), but later adhesions may lead to encystment.

Pleural fluid produces stony dullness with absent breath sounds and voca-

remitus with displacement of the mediastinum to the opposite side. Diagnosis can only be confirmed with the aspirating needle.

In all cases the ætiology must be determined by (a) bacteriological, biochemical and cytological analysis of fluid, (b) needle biopsy of pleura (Abrams), fig. 856, (c) thoracoscopic examination and biopsy of pleura, or (d) open thoracotomy.

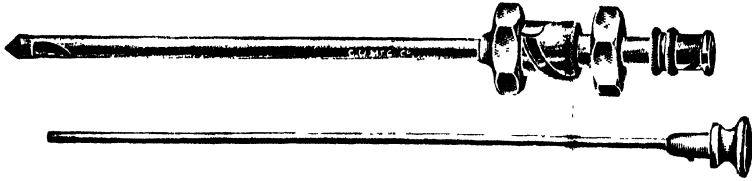


FIG. 856.—Abrams' Pleural Biopsy Punch.

In addition to these local measures a careful assessment of the lung and the heart is required and a search should be made for distant neoplasms (ovaries, etc.).

Chylothorax.—Chyle may leak into the pleural cavity from the thoracic duct or its tributaries as a result of trauma (stab wounds, crush injuries, surgical injury) or obstruction by tumour. It is most commonly mistaken for pus.

Chyle contains fat and the diagnosis depends on its demonstration by naked eye or microscope. Confirmation is obtained by giving 100 gm. of butter mixed with 1 gm. of confectioner's green dye (D and C green No. 6) by mouth. The effusion is aspirated twelve to twenty-four hours later when a chylous effusion will be uniformly green.

Pseudochylous effusions have a milky appearance not due to fat (cholesterol and calcium phosphate crystals, filarial parasites).

Chyliform effusions contain fat, but this has been derived from the breakdown of cells in encysted effusions. The dye test will be negative.

Management will depend on the cause. The majority of surgically produced chylous effusions and some traumatic ones resolve with conservative measures (low fat diet and chest aspiration). If accumulation continues after a trial period of two weeks, thoracotomy is indicated to locate and suture the severed duct.

Empyema.—An empyema is a pleural abscess and consists of a collection of pus in the pleural cavity. The term is often wrongly used in a much wider sense to include all phases of pleural infection from an infected turbid effusion to a mature abscess containing thick pus. In the management of an empyema, it is just as important to consider the degree of localisation and pus formation as it is in other forms of septic cellulitis.

Ætiology.—An empyema is never primary. The majority are secondary to pulmonary infection, particularly pneumococcal pneumonia and bronchopneumonia, but any infective process, such as tuberculosis, lung abscess and bronchiectasis may be complicated by an empyema. Any inflammatory condition in the vicinity of the pleura may give rise to an empyema, namely :

(a) Chest wall (wounds, osteomyelitis of rib).

(b) Lung (pneumonia, abscess, bronchiectasis, tuberculosis, newgrowth).

- (c) Post-operatively (thoracotomy).
- (d) From the œsophagus (perforations, carcinoma).
- (e) From below the diaphragm (subphrenic abscess).

Pathology.—In the common post-pneumonic empyema actual pleural infection is preceded by the development of a serous effusion. The pleura is subsequently invaded by organisms from the lung with associated inflammatory changes and further exudation of fluid. Fibrin is deposited on the surfaces of the pleura, whilst intrapleural clotting is common in certain types which form a protein-rich exudate (pneumococcal empyema). The natural defences of the body are aimed at encircling the septic area by a barrier of fibrous tissue: this is initially achieved by the fusion of the lung to the chest wall at the periphery of the collection of fluid. Subsequently the fibrin deposits on the pleura are invaded by blood-vessels from the adjacent lung or chest wall with the formation of granulation tissue, and later of fibrous tissue. This process is progressive with an ever-increasing thickness of the wall of the empyema. Left to her own devices, nature will try to obliterate the empyema by converting it into a plaque of fibrous tissue. As the empyema becomes walled off by adhesions, the fluid thickens so that the presence of thick pus is a good indication that the empyema is localised and is unlikely to spread further. A mature empyema consists of visceral and parietal layers of fibrous tissue on the lung- and chest-wall surfaces respectively, with pus and debris between them. There is usually a good plane of cleavage between the wall of the empyema and the visceral pleura which is important in the operation of decortication. Secondary changes in the surrounding structures appear as the fibrous tissue contracts. The ribs are drawn together and lose their mobility. The diaphragm is elevated and fixed, and the mediastinum drawn towards the affected side. The lung is encased in a rigid covering of fibrous tissue and is immobile and functionless. The ultimate picture of a neglected chronic empyema is a rigid contracted chest with relatively functionless lung underneath (frozen chest).

Clinical Features.—Empyemas, for convenience, can be divided into three groups: (1) Acute. (2) Subacute. (3) Chronic.

The commonest organisms found are pneumococci and streptococci, but a wide variety of organisms, either alone or in combination, can be encountered.

Acute Empyema.—The acute fulminating toxic empyema is now rare except when it follows perforation of the œsophagus or rupture of a lung abscess. There is profound toxæmia and shock with pleural pain and rapid, shallow respiration. The signs are those of pleural fluid, which should be confirmed by needle exploration. Early thoracotomy is required if the condition follows rupture or perforation of the œsophagus. Other cases should be treated by repeated aspirations with systemic and intrapleural antibiotics in an attempt to control the infection. If these measures fail to control the toxæmia, drainage should be carried out with an intercostal catheter inserted through a cannula and connected to an under-water seal.

even though the pus is thin and the empyema not walled off. Subsequent management will be that of the subacute empyema.

Subacute Empyema.—The majority of empyemas present in a less severe form largely owing to the general use and efficiency of the antibiotics administered for the primary condition. As a result, an empyema may develop insidiously and its presence be completely overlooked. It should always be considered in cases of delayed resolution, slow convalescence or persistent fever following a pneumonic illness. Likewise, it should be suspected where the physical or radiological signs of resolution are incomplete. Clinical signs are those of fluid with stony dullness, absent breath-sounds, diminished chest movements, and displacement of viscera.

Chronic Empyema.—Many chronic empyemas are the result of mismanagement of the acute or subacute stages, some are due to failure to diagnose the original condition, and the remainder to some underlying pathology in the lung (bronchiectasis, lung abscess, tumour), pleura (foreign bodies, actinomycosis), or chest wall (rib sequestrum). Toxic absorption from the empyema is slight but symptoms of vague ill-health, febrile bouts, anæmia etc., may occur. A chronic empyema may present in one of several ways, namely :

- (1) A closed collection of pus completely walled off from its surroundings (localised empyema).
- (2) An empyema which is discharging either continuously or intermittently into a bronchus (bronchopleural fistula).
- (3) An empyema which is discharging either continuously or intermittently through a sinus in the chest wall.

Diagnosis.—An empyema is diagnosed by finding pus with an exploring needle. Failure to find pus may be due to :

- (1) Use of too narrow a needle or the employment of an inefficient syringe.
- (2) Selection of the wrong site for aspiration.
- (3) The presence of clot or fibrin tags which block the aspirating needle.

The commonest cause of failure is selection of the wrong site, but this should be avoided by a careful clinical and radiological examination beforehand. If pus is found, it is an advantage to keep two specimens—one for bacteriological studies, and the other for comparison with subsequent specimens.

Iodised oil can be injected into the empyema so that in subsequent radiographs the lowest limits of the space will be outlined (fig. 857).

Having confirmed the diagnosis, it is important to determine the underlying cause ; if the empyema is secondary to a carcinoma, lung abscess or a subphrenic abscess, etc., the primary lesion requires appropriate treatment.

Management.—The principles of management of an empyema are basically the same for all varieties though the emphasis tends to vary in the different types. Following diagnosis and the determination of the cause, management resolves itself into controlling infection and eliminating the dead space.

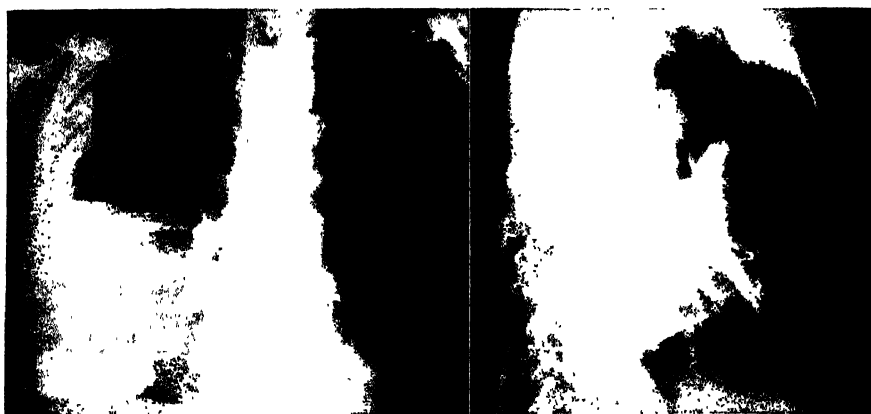


FIG. 857.—Postero-anterior and lateral radiograph of an empyema after injection of iodised oil to delineate the lower limits of the space prior to drainage.

Every case requires vigorous breathing exercises if residual deformity and limitation of movement are to be minimised or avoided.

Control of Infection.—Initially all empyemas should be treated by aspirations repeated on alternate days, with the removal of as much pus as possible and the introduction of antibiotics. Sterilisation is usual within seven to ten days. Only occasionally do aspirations fail to control infection, in which case drainage should be carried out.

Elimination of Dead Space.—(a) *Aspirations.*—Repeated aspirations without replacement of air will often result in full expansion of the lung and cure of the empyema. This is often the case with children and is more likely after the use of streptokinase. Aspirations should be continued until fluid is no longer obtained and the X-rays show no residual opacity.

(b) *Drainage.*—Simple tube drainage after rib resection is indicated when aspirations fail to produce expansion of the lung.

The operation is carried out under local anaesthesia. The site selected for drainage lies immediately above the lowest limit of the empyema posteriorly. The lower posterior site is preferred because this is the most dependent part of the empyema with the patient sitting up in bed. A vertical incision is made over the selected rib and the muscles divided in the line of incision. The periosteum of a 3-inch (7.5-cm.) segment of rib is elevated and the segment removed. The intercostal bundle should be securely ligated. The empyema is entered through the posterior periosteal bed and the hole enlarged to permit complete evacuation of the empyema and a thorough inspection and biopsy of the pleura. Drainage should at first be closed, using an under-water seal, but when the discharge is reduced to 2 ounces (60 ml.) daily open drainage should be instituted. Drainage must be maintained until the empyema cavity is completely obliterated, which may take as long as six to eight weeks. Premature removal of the tube is a frequent cause of chronicity. Control is effected by carrying out serial pleurograms with opaque oil injected into the empyema cavity at intervals of three weeks. The tube should project 1 inch to 2 inches (2.5 to 5 cm.) into the empyema cavity and will require little alteration until the final stages, when it should be adjusted to keep it $1\frac{1}{4}$ inches (4 cm.) shorter than the empyema track.

(c) *Decortication (Excision of Empyema).*—This operation, as an alternative to drainage, has become popular since the last war, when experience showed that satisfactory results could be obtained. The operation aims at a

complete removal of the empyema with its fibrous-tissue walls, leaving the lung and chest wall free to expand. It gives excellent functional results with minimal pleural thickening. Convalescence is rapid without the necessity for repeated dressings, and return to work is possible within a few weeks. The operation is, however, a major one requiring skilled anæsthesia and blood transfusion. It is indicated particularly for large and chronic empyemas, and for those secondary to bronchiectasis, lung abscess, or carcinoma where the underlying lung condition can be dealt with at the same time. It is contraindicated in the frail and elderly, or where complete resolution of a pneumonic process has not yet occurred.

(d) *Operations on the chest wall*, such as a thoracoplasty, the deroofting procedure of Schede, Roberts' flap operation, and the use of muscle grafts have been almost entirely replaced by the operation of decortication.

Tuberculous Empyema.—May be either a simple tuberculous infection or one complicated by secondary infection or a bronchopleural fistula. The majority are complications of an artificial pneumothorax or surgical treatment. They are much less common than they were, partly due to the widespread use of streptomycin, but also to the decreased popularity of artificial pneumothorax. The treatment is fundamentally the same as for any empyema, namely control of infection and obliteration of the pleural space, but is coloured by the presence of active tuberculous disease of the lung or a bronchopleural fistula. Control of infection is often possible with regular aspirations and the use of systemic and intrapleural streptomycin, P.A.S., or I.N.A.H. The method adopted to obliterate the empyema depends on the condition of the underlying lung. If active pulmonary disease is present, either a thoracoplasty or a pleuropneumonectomy will be required. If the lung disease is controlled and inactive, obliteration may be possible by repeated aspirations over a prolonged period, or alternatively, by excision of the empyema. The operations of decortication and pleuropneumonectomy have dramatically altered the outlook in these cases.

Interlobar Empyema.—An interlobar empyema is a rarity, but one which is wrongly diagnosed with remarkable frequency. It can only occur when the interlobar space is completely shut off from the general pleural cavity. Occasionally a collection of pus remains localised in the fissure in cases of generalised pleural infection when the main collection has been adequately dealt with by aspiration. The diagnosis is largely a radiological one. The collection of fluid is oval or fusiform in shape with the long axis lying in the plane of one of the fissures. The appearances are easily confused with those of segmental atelectasis; if suspected, pus should be sought with an aspirating needle. Treatment consists of repeated aspirations with chemotherapy, or drainage if aspirations fail.

DISEASES OF THE BRONCHI

Intrabronchial Foreign Bodies

Inhalation of foreign bodies is not a rare occurrence, particularly in children. In many instances the individual assumes that the foreign body has been expelled as the initial choking may soon pass. The commonest foreign bodies are teeth, mutton and rabbit bones, peanuts, pins, screws and nuts. The changes produced depend upon the size and nature of the foreign body. Small, smooth metallic foreign bodies produce little reaction, whilst larger foreign bodies may lead to partial or complete obstruction of one of the bronchi. Organic foreign bodies, particularly peanuts, produce marked inflammatory changes in the bronchial wall.

The clinical features may be :

(1) Wheezing, irritating cough and signs of unilateral obstructive emphysema (figs. 858 and 859).



FIG. 858. — Obstructive emphysema of the left lung due to an inhaled peanut.

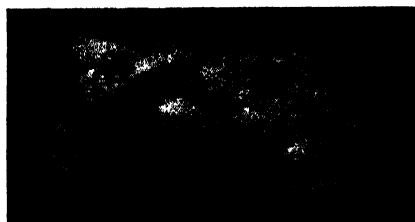


FIG. 859. — Peanut removed bronchoscopically from case illustrated in fig. 858.

(2) Symptoms due to atelectasis or pulmonary suppuration ; cough, sputum, fever, etc.

(3) The patient may be symptomless.

Radiography is an essential investigation in every suspected case. If the foreign body is radio-opaque, it should be visible either in the postero-anterior or lateral film, but in many instances the foreign body is either not opaque or is obscured by secondary inflammatory changes.

Treatment.—Bronchoscopy must be performed in all suspected cases. Removal is easier before secondary swelling and inflammation of the bronchial wall have occurred. Many can be removed without special equipment. Prolonged attempts at removal should not be made owing to the risks of oedema of the glottis or of damaging the bronchial tree further. Operative removal is preferable in such cases. Antibiotics should be given beforehand to minimise the inflammatory reaction. Removal is often possible by bronchotomy, but if suppurative changes have already occurred in the lung resection will be necessary.

Bronchiectasis

The term bronchiectasis strictly describes any abnormally dilated bronchus. Bronchial dilatation may follow many inflammatory conditions of the lung, especially those associated with obstruction of the bronchus. These changes, which are often patchy and affect only small peripheral bronchi may follow pulmonary tuberculosis, a lung abscess or pulmonary suppuration. Only rarely do they give rise to symptoms. Clinical 'bronchiectasis', however, is usually applied to the variety where involvement of the bronchi is more extensive and usually follows an anatomical pattern, e.g. a segment, a lobe or a lung. It is infection in these abnormally dilated bronchi which produces cough, sputum and hæmoptyses; these together form the pattern of the clinical condition of bronchiectasis.

Ætiology.—Many cases are considered to be congenital in origin, though there is no good evidence to support such a thesis. The majority are the result of compression of the small soft bronchi in children by enlargement of the hilar lymph nodes associated with whooping-cough, measles or primary tuberculosis; the obstruction is often made

complete by viscid intrabronchial secretions. Bronchial obstruction produces atelectasis. (The majority of cases of pneumonia complicating whooping-cough are really atelectases.) The subsequent course depends on whether secondary suppurative changes occur in the atelectatic lobe, or not. If no infection occurs and the obstruction is relieved, the lobe will expand and return to normal. If suppuration occurs, the bronchial walls are severely damaged and distended by purulent secretions. They lose their elasticity and mobility, and when obstruction is eventually relieved they remain as inert, dilated tubes.

Pathology.—The bronchi appear thickened and irregular, whilst histologically there is marked destruction of all the normal elements and extensive infiltration of the remnants with inflammatory cells. The columnar epithelium is replaced by granulation tissue or by squamous or cuboidal epithelium. The lung supplied by the affected bronchi may be fibrotic and airless with loss of the alveolar pattern and infiltrated with inflammatory cells (bronchiectasis with an atelectatic lobe) (fig. 86o). In other cases the abnormal bronchi are surrounded by aerated lung tissue but the latter is usually emphysematous and relatively functionless.

Clinical Features.—The majority of cases date their symptoms from an attack of whooping-cough, measles or pneumonia in childhood. The symptoms may persist from that time or the attack may be followed by a latent period of some duration. They may subsequently be intermittent and are often regarded as attacks of bronchitis. Frequently, especially in severe cases, they are persistent throughout the year, but with exacerbations from time to time.



FIG. 86o.—Bronchogram showing bronchiectasis and atelectasis of right middle and lower lobes.

Characteristically, the patients have a loose cough productive of purulent sputum; the latter may be offensive and is often blood-stained, whilst frank hæmoptyses are not infrequent. Acute exacerbations with fever and increase of cough and sputum are frequent during the winter months. In children, both physical and mental retardation may occur. Finger clubbing is frequent and persistent râles and rhonchi are audible over the affected areas. Dullness and bronchial breathing with mediastinal shift occurs with an atelectatic lobe.

Diagnosis.—The diagnosis is usually possible on clinical grounds from a prolonged history of cough and sputum associated with finger clubbing and physical signs over a localised area of the lung. Bronchography is essential, not only to demonstrate the presence of bronchiectasis but also to determine its extent (fig. 86o). Bronchoscopy is advisable in order to exclude the presence of a foreign body or tumour.

Treatment.—(a) *Prophylactic Treatment.*—Much can be done to prevent the development of bronchiectasis by treating the respiratory complica-

tion of measles and whooping cough with antibiotics and breathing exercises, and carrying out bronchoscopy at an early date should atelectasis occur.

(b) *Conservative Treatment*.—Considerable reduction of infection can be achieved by postural drainage, physiotherapy and chemotherapy. Such medical treatment, however, cannot cure the condition as the essential abnormality still remains. Relapse is frequent even though treatment is maintained for long periods. Postural drainage is carried out in a position designed to place the affected lobe above the draining bronchi so that pus will gravitate from the lobe into the bronchus from whence it can be expectorated. Periods of half to one hour two or three times daily are advisable. Chemotherapy is reserved for acute attacks or as a preparation for surgery.

(c) *Surgical Treatment*.—Extirpation of the diseased area is the only available method of curing the condition, but is not applicable to every case. It is essential in all cases to carry out complete bronchograms on both sides. Resection is only practicable if less than half of the total lung tissue is involved. Ideally, all abnormal areas should be removed (radical treatment) but in certain circumstances it is justifiable to remove a grossly diseased lobe and leave behind less severely damaged tissue (palliative resection). Children will tolerate the removal of more lung tissue than adults, and bilateral resections are rarely advisable after the age of thirty-five years. Removal should be as conservative as possible, particularly in the more extensive cases. Prior to operation, the patient should have intensive treatment with postural drainage and chemotherapy in order to reduce sputum to a minimum, whilst after operation, rapid and complete expansion of the remaining lung is encouraged by breathing exercises, posture and suction drainage to reduce the risks of empyema and bronchopleural fistulæ. The results of surgical treatment are best in cases with limited bronchiectasis in an atelectatic lobe. Disappointing results are encountered in patients with generalised bronchitis or asthma in addition to bronchiectasis.

Lobectomy.—The operation is carried out through a postero-lateral thoracotomy. The passage of pus from the bronchiectatic lobe into normal lung is prevented by intrabronchial balloons or by posture (face down position). The hilum is approached either from its posterior or interlobar aspect. The relevant bronchus, artery and vein are isolated by blunt dissection using long curved artery forceps. The bronchus is divided between clamps and the vessels are securely ligated. The bronchus is closed by interrupted sutures of silk, thread, nylon or steel wire. The lobe is then freed from its attachments to the parietes and mediastinum. The chest should be temporarily drained by a basal tube, with an additional apical tube if leakage of air is at all prominent.

TUMOURS OF THE LUNG AND BRONCHI

With one or two rare exceptions, all pulmonary tumours arise from some part of the bronchial tree¹.

¹ Mesothelioma is another type of tumour but it originates from the pleural membrane. It is believed to occur more commonly in those who work with blue asbestos fibre.

Classification**Benign.—Adenoma.**

Hamartoma (fibroma, chondroma, lipoma, angioma).

Malignant

Primary.—Carcinoma (common)¹.

Alveolar-cell carcinoma (rare).

Sarcoma (rare).

Secondary.—Sarcoma.

Teratoma of testis.

Carcinoma (bowel, breast, thyroid, kidney).

Symptomatology.—The symptoms produced by a tumour may be due to:

- (1) The presence of an irritating lesion in the bronchial tree producing cough, sputum, wheezing and hæmoptysis.
- (2) The occurrence of partial or complete bronchial obstruction giving rise to obstructive emphysema or atelectasis respectively.
- (3) Secondary inflammatory changes in the lung producing consolidation, suppuration or abscess formation.
- (4) Symptoms due specifically to the presence of a primary tumour or of its secondary deposits—malaise, anorexia and loss of weight.
- (5) Pressure on the œsophagus (dysphagia), trachea (stridor), recurrent laryngeal nerve (hoarseness) or superior vena cava.
- (6) Systemic disturbances due possibly to active secretion by the tumour cells—(carcinoid syndrome, Cushing's syndrome, osteo-arthritis, neuropathy).

Benign Tumours

These represent about 2 per cent. of all pulmonary tumours. They are slightly more common in women and more frequent in the younger individual.

Pathology.—The tumours fall into two main anatomical groups, those arising from the larger bronchi and those situated in the periphery of the lung. The former are usually visible through the bronchoscope. The intra-bronchial portion of the tumour, however, usually only represents a portion of the whole as there is frequently a large extra-bronchial portion (iceberg tumours). The peripheral tumours are not visible through the bronchoscope.

Histologically there are two main types—adenoma and hamartoma. The adenoma is the more common; the cells are regular, well formed and consistent in appearance and tend to be arranged in solid acini or cylinders. Mitoses are infrequent. The hamartoma is a composite tumour composed of two or more tissue elements. They represent an abnormal mixing or development of the normal components of the organ in which they occur. Thus, in the lung, cartilage, fat, glandular or vascular tissue and respiratory epithelium may be found. The hamartomas are usually described according to the preponderant tissue, e.g. chondromatous, hæmangiomatous hamartoma, etc. (p. 57).

Clinical Features.—(1) Many are symptomless and found on routine radiography; this particularly applies to the peripheral types.

(2) Recurrent hæmoptysis. The adenoma is one of the causes of repeated large hæmoptyses occurring over a period of years. There is frequently complete freedom from symptoms between attacks, though occasionally wheeziness and an irritating cough are present.

(3) Symptoms due to bronchial obstruction and lung infection. Simple uninfected atelectasis or pulmonary suppuration with abscess formation and bronchiectasis both occur commonly (fig. 861). Cases sometimes present with a lung abscess or an empyema with the underlying adenoma completely unsuspected until bronchoscopy is performed.

Diagnosis.—The diagnosis is often suggested by the history, but can only be confirmed by bronchoscopy. The adenoma appears as a round, smooth or slightly lobulated raspberry-like tumour which is neither ulcerated nor necrotic. Tumours containing cartilage appear pale and hard and biopsy may be difficult. Bronchography is necessary to determine the presence and extent of any secondary lung changes which will influence subsequent treatment.



FIG. 861.—Adenoma of bronchus (arrow) producing secondary bronchiectasis of the left lower lobe.

Treatment.—Surgical resection is the treatment of choice. This can be planned conservatively as the risk of recurrence is negligible. Treatment by radon implantation, curettage or irradiation is ineffective and cannot be recommended. One or other of the following methods may be applicable to the individual case:

(1) *Bronchoscopic Removal.*—This is only practicable for the rare, strictly intra-bronchial and pedunculated tumours. Bronchoscopy is advisable at regular intervals after removal in order to detect a recurrence.

(2) *Bronchotomy.*—Local removal of the tumour with part of the bronchial wall is practicable for tumours of localised extent without extra-bronchial extension and without secondary lung damage.

(3) *Lung Resection.*—A conservative resection, either segmental, lobar, or total, is required in those cases associated with permanent lung damage. The extent of the resection is determined by the bronchographic findings.

Carcinoma of the Bronchus

Ætiology.—Carcinoma of the bronchus has shown a fifteen-fold increase during the last thirty years. It is ten times more common in men than women, and most frequently occurs during the fifth and sixth decades, but is not unknown in children and young adults. Statistical surveys show that heavy cigarette smoking over many years predisposes to the development of lung cancer. The exposure to other irritants, such as arsenic and radioactive substances (Schneeberg¹ cancer), exhaust fumes, sulphurous smoke and fog, and tarry particles from the roads may possibly be implicated. Workers in the chromate industry show a high incidence of lung cancer.

Pathology.—Three main macroscopic types are described :

(1) *Main Bronchus Tumours.*—These arise in the main bronchus or one of its primary or secondary divisions. They produce bronchial irritation and ulceration at an early stage and frequently give rise to bronchial obstruction. They are usually visible through the bronchoscope (fig. 862).

(2) *Peripheral Tumours.*—These arise from the smaller bronchi beyond the range of bronchoscopic vision and rarely



FIG. 862.—Carcinoma of the left upper lobe bronchus. The upper lobe is atelectatic and its bronchi are dilated and filled with mucus. The lower lobe is normal.

¹ The name of a district in Germany where these substances are mined.

produce secondary lung changes. Some are discovered at routine radiography (figs. 863 and 864).

(3) *Pancoast Tumours*.—These are essentially peripheral lung carcinomas arising at the apex of the lung. The tumour extends early along pre-existing apical adhesions to invade the brachial plexus, sympathetic chain, upper ribs and adjacent vertebræ producing the Pancoast syndrome (lower brachial plexus lesion, Horner's syndrome, an apical shadow, and rib erosion). The lesion is particularly distressing owing to the early production of intractable pain which is difficult to relieve, combined with a relatively slow growth.



FIG. 863.—Peripheral carcinoma of the lung showing thick-walled irregular cavity.



FIG. 864.—Peripheral type of lung carcinoma, showing typical lobulated outline and absence of secondary changes in the lung.

Histology.—Three main histological types are recognised. (1) Squamous-cell carcinoma = 60 per cent. (2) Anaplastic, oat-cell or round-cell carcinoma = 30 per cent. (3) Adenocarcinoma or columnar-cell carcinoma = 10 per cent. The anaplastic carcinomas are poorly differentiated tumours exhibiting rapid growth with early metastases. They are more common in young individuals.

Metastases.—(1) Direct extension into the mediastinum, the pleura, the chest wall or the pericardium

(2) The lymphatic system to the hilar lymph nodes and thence to the subcarinal nodes or the paratracheal chain. The upper nodes of the chain are continuous with the inferior deep cervical group and may be detected by digital palpation deep to the insertion of the sternomastoid muscle, or by scalene node biopsy.

(3) The blood-stream to the liver, brain, and bone (ribs, vertebræ, pelvis particularly). Other tissues and organs may be involved.

Clinical Features.—Symptoms produced by a carcinoma of the bronchus are many, but the most important are cough, sputum, hæmoptysis, dyspnoea, pain and wheezing. It is important to recognise the early manifestations as it is at this stage that treatment is likely to be most effective. Any of the above symptoms appearing for the first time in a middle-aged individual and tending to become more severe with time, demand early investigation. Only some 10 per cent. of cases present initially with an hæmoptysis; more frequently, the first symptoms may occur suddenly with an influenzal-like illness which fails to resolve satisfactorily. A number start insidiously with

cough and dyspnoea, whilst in the case of the chronic bronchitic there may be an alteration in the typical pattern of his symptoms. Occasionally, first symptoms are due to the presence of secondary deposits (especially in the brain) or invasion of the mediastinum. A diagnosis of unresolved or virus pneumonia should always be made with reservations, and if recovery is not complete, carcinoma should be suspected. Finger clubbing is a frequent and important sign. A small group present with neurological (carcinomatous neuropathy) or arthritic (osteo-arthropathy) symptoms without chest symptoms. The true nature of the condition is revealed by chest X-ray.

Investigations.—The suspicion of a lung cancer demands a thorough investigation. A *chest X-ray* is the most important single investigation and it is usual for some abnormality to be revealed. The appearances vary from that of a hilar or peripheral shadow due to the presence of the tumour to those of obstructive emphysema, atelectasis or consolidation due to secondary lung changes.

Bronchoscopy.—This is not only an essential diagnostic procedure but produces important information regarding operability. The examination will reveal the presence of the tumour in those lesions which are centrally placed. Paralysis of a vocal cord, compression of the trachea, widening of the main carina, and involvement of the main bronchus within 1.5 cm. of the carina are regarded as evidence of inoperability. It should be emphasised that bronchoscopy may not reveal the presence of some tumours, particularly those arising in the segmental divisions of the upper lobes, or the peripherally placed tumours.

Cytological examination of sputum may reveal malignant cells in cases where biopsy material is not available during bronchoscopy.

Barium Swallow.—This may provide evidence of extension of the growth into the mediastinum or the enlargement of mediastinal lymph nodes. If oesophageal distortion is considerable, adequate removal is improbable.

Tomography is useful in demonstrating enlarged mediastinal nodes.

Bronchography.—This may provide valuable information in cases of doubtful diagnosis but has little place in the investigations of the proven case.

Scalene Node Biopsy and Mediastinoscopy.—Nodes from the paratracheal and inferior deep cervical groups may be obtained by these means. If invaded radical surgery is contra-indicated.

Diagnosis.—A positive histological diagnosis is available in about 60 per cent. of cases, whilst malignant cells in the sputum or pleural fluid provide positive evidence in some of the remainder. In about one-third of cases, however, only a presumptive diagnosis can be made.

In the unproven case, a carcinoma is most likely to be confused with chronic suppurative pneumonia, a lung abscess or solid tuberculous lesions, and occasionally with a benign tumour. Careful consideration of the history and response to antibiotics will often provide strong circumstantial evidence against a carcinoma, whilst bronchography and tomography may be helpful in doubtful cases.

Treatment.—Surgical removal of the tumour offers the best prospects for cure, although in selected cases the results of radical radiotherapy are equally good.

The aim of operative treatment must be the adequate removal of the primary tumour and the regional lymph nodes, but with the preservation of as much normal lung tissue as possible.

The extent of the lung resection will thus depend on the site of the tumour. For lesions confined to one lobe *lobectomy* will suffice, but those involving the main bronchus *pneumectomy* will be required (fig. 865). An inter-

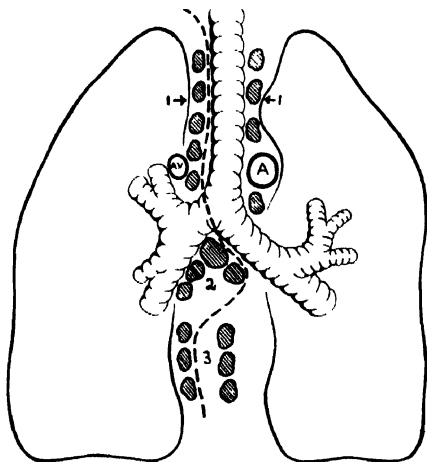


FIG. 865.—Right radical pneumectomy. The dotted lines indicate the plane of dissection. A.V. = azygos vein. A. = aorta. 1 = paratracheal nodes. 2 = subcarinal nodes. 3 = para-aortic nodes.

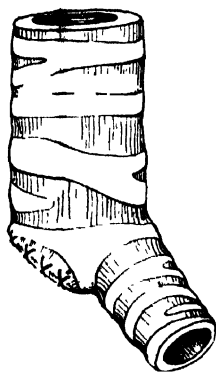


FIG. 866. — Sutured bronchus after right pneumectomy. Note oblique division, absence of stump, and figure-eight stitches.

mediate group can be adequately treated by removing the involved lobe and a cylinder of the main bronchus—*sleeve resection*—thus preserving some normal lung tissue. In all cases the paratracheal, subcarinal and para-aortic lymph nodes are removed. Limited resection of the chest wall, pericardium or diaphragm can be carried out should these structures be invaded.

Radical Pneumectomy

One lung anaesthesia is advisable. The feasibility of lung resection should be determined with as little lung disturbance as possible. At an early stage the pulmonary veins from the affected lobe should be tied to reduce the risks of tumour embolism. The remaining vessels are then isolated and divided after securing with double ligatures or a transfixed ligature. The bronchus is divided close to its origin after applying special bronchus clamps. The bronchus is closed with sutures of wire, nylon, or thread, using interrupted figure of eight stitches and occluding a rim of bronchus 3 to 4 mm. wide (figs. 865 and 866).

The paratracheal and subcarinal and para-aortic lymph nodes should be removed with surrounding tissues.

Only 20 per cent. of lung cancers are considered suitable for exploration and of these one in five is found to be too advanced for resection on exploration. The mortality of radical resection is 10 per cent. Of those surviving resection, 25 per cent. are alive in five years' time.

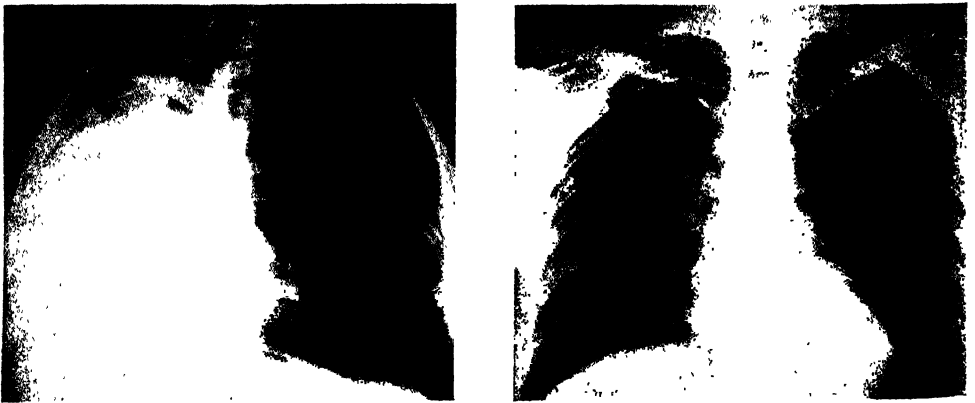
The following are contraindications to radical resection :

- (1) A growth which involves the trachea or the first 1·5 cm. of the main bronchus.
- (2) Evidence of spread into the mediastinum with involvement of the œsophagus, recurrent laryngeal or phrenic nerves, vena cava or pericardium.
- (3) Extensive invasion of the chest wall (fixed chest pain).
- (4) Evidence of distant metastases.
- (5) Patients over the age of sixty-five unless remarkably fit.
- (6) Patients with poor respiratory reserve. (This can usually be assessed on clinical grounds with simple exercise tests).
- (7) Symptoms due to cardiovascular disease, such as angina, auricular fibrillation, etc.
- (8) Chronic bronchitis with persistent signs in the normal lung where the patient is likely to be severely crippled by dyspnoea and bronchitis after operation (respiratory cripple).

Palliative Resection.—At thoracotomy it is advisable to remove the lung with the primary tumour whenever possible even though the growth cannot be completely removed. Patients are better off without the primary tumour and die more peacefully from secondary deposits. Occasionally a deliberate palliative resection is justified in cases with repeated hæmoptyses or with much pulmonary suppuration.

Interstitial Irradiation.—This is carried out by the insertion of radioactive grains of gold ^{198}Au . It is a useful method of palliation in those tumours found to be irremovable at thoracotomy and after palliative or incomplete resections. Gold grains can also be inserted into intrabronchial tumours through the bronchoscope.

X-ray Therapy.—Radical X-ray therapy, particularly with super-voltage machines, is employed in cases which are considered operable from the point of view of the tumour, but inoperable on other grounds. Survival rates of some years have been recorded but no extensive series is available (figs. 867 and 868). Palliative X-ray therapy is designed to relieve superior



FIGS. 867 and 868.—Main bronchus carcinoma, showing total atelectasis of right lung before irradiation, and aeration of the lung after treatment.

vena caval obstruction, bronchial obstruction and the pain of bone involvement. Considerable relief is obtained by such treatment, but survival is not appreciably lengthened.

Pre-operative X-ray therapy has given disappointing results largely due to an increased incidence of broncho-pleural fistulæ following resection.

Post-operative treatment is used occasionally where small areas of growth have not been removed (e.g. on the chest wall). Only fit patients should be treated if serious debilitating reactions are to be avoided.

Chemotherapy.—The use of cytotoxic drugs alone has been disappointing. Some benefit may follow their administration combined with surgery.

DISEASES OF THE LUNG

Lung Abscess

A lung abscess is a collection of pus in the lung substance. All cases are secondary to some pre-existing condition, such as foreign bodies, tumours, or bronchostenosis, or to the inhalation of infective material. There is no such condition as primary or idiopathic lung abscess, and in all cases the cause should be elucidated.

Classification.—*A. Due to specific pneumonias.*—(1) Staphylococcal, (2) Actinomycotic, (3) Friedländer's pneumonia, (4) Amœbic.

B. Due to bronchial occlusion.—(1) Carcinoma, (2) Benign tumours, (3) Intrabronchial foreign bodies, (4) Sputum plugs (post-operative atelectasis).

C. Due to vascular embolism.—(1) Infarct, (2) Pyæmic.

D. Traumatic.—(1) Infected hæmatoma of lung, (2) Foreign body in lung.

E. Aspiration (inhalation) pneumonia.—Non-specific suppurative pneumonia.

The most important group is that due to an aspiration pneumonia. In these cases infected material, usually from the upper respiratory passages, is inhaled into the lung, where various grades of infection ensue from a relatively benign bronchopneumonia to more severe states with necrosis, suppuration and abscess formation.

The most important factors in the production of a lung abscess are :

(1) A source of infected material (dental sepsis, sinus infection, vomit, operations on the upper respiratory tract).

(2) A disturbance of the natural defence mechanisms of the body by depression of the cough reflex (with drugs, in coma, or after anæsthesia or alcohol).

(3) A disturbance of the normal mucous production and ciliary action of the bronchial mucosa (Negus).

Pathology.—Infected material is inhaled into the bronchial tree and obstructs one of the smaller bronchi. The resulting atelectatic segment is invaded by pathogenic organisms producing pneumonic consolidation. The commonest organisms are *Hæmophilus influenzae* and Pneumococci but invasion by mouth organisms (anaerobic streptococci and spirochaetes), coliform organisms and cross infection with drug-resistant staphylococci sometimes occur and may be detected by microscopy and culture of the sputum. Suppuration and necrosis develop in varying degrees within the involved segment, first with the production of a suppurative pneumonitis which

later matures into an abscess. As pus accumulates, tension rises and eventually the abscess ruptures into the bronchus. This may be followed by complete expectoration of all sloughs and pus, and the inflammation may then subside. More commonly, however, a state of chronic infection with persistence of the abscess occurs (fig. 869). Progressive involvement of adjacent lung tissue or spread to other parts of the lung is possible.

Clinical Features.—The onset is often acute with influenza-like symptoms and variable toxæmia, but usually without any localising symptoms. After some days of ill-defined illness, pleural pain and a dry irritating cough may appear; the latter becomes worse and the patient may notice an offensive odour in his breath. About the tenth day, rupture of the abscess results in the sudden expectoration of considerable quantities of offensive blood-stained pus. With successful treatment, resolution occurs slowly with reduction of toxicity and diminution of cough and sputum, but healing takes several weeks and can only be regarded as complete when cough and



FIG. 869.—Chronic lung abscess showing irregular cavity filled with inspissated pus and sloughs and surrounded by an area of chronic pneumonia.

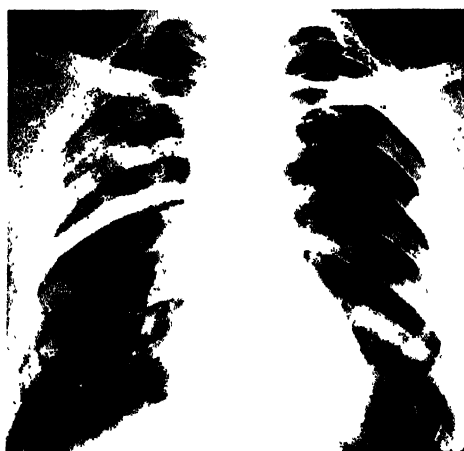


FIG. 870.—Lung abscess in the right upper lobe. There is an ill-defined area of consolidation surrounding the cavity which shows a fluid level.

sputum have disappeared and radiographs are clear. Chronicity is suggested by the persistence of cough with the expectoration of purulent sputum and the presence of a cavity on X-ray (fig. 870).

Complications.—(1) Spread of the disease to other parts of the lung by direct extension or by bronchial embolism: this is suggested by an exacerbation of symptoms and fresh radiographic changes.

(2) *Empyema*.—This may occur spontaneously or following surgical drainage but is less common following the introduction of antibiotics. Persistent fever, pleural pain, and signs of a pleural effusion call for exploration of the pleura with a needle.

(3) *Cerebral Abscess*.—Pyæmic emboli reach the brain through the paravertebral system of veins (Collis). The resulting abscesses are usually multiple but occasionally single. The onset is usually accompanied by headaches and fever. A fit is occasionally the first symptom. The incidence of cerebral abscess has fallen markedly since the introduction of antibiotics.

(4) *Secondary Hæmorrhage*.—Hæmorrhage may occur from a drained or an undrained abscess. It is often no more than blood-staining of sputum or discharge, but occasionally severe or fatal hæmorrhage occurs.

Diagnosis.—The diagnosis is suggested by a history of a sudden, acute influenza-like illness, followed later by coughing with purulent sputum and the demonstration of an area of lung consolidation, with or without cavitation. All cases must be investigated to determine the cause by:

- (1) Bronchoscopy to exclude foreign bodies or neoplasm.
- (2) Complete bacteriological examination of the sputum.
- (3) Examination of the teeth and upper respiratory tract.

Treatment.—Eighty-five per cent. of lung abscesses resolve with conservative measures, and surgery is only required for the minority which do not respond. Medical treatment consists of:

(1) *Chemotherapy*.—Penicillin is the antibiotic of choice as it can be given in very large doses (2 to 8 million units daily) for long periods (four to six weeks) with little risk of side-effects. Other antibiotics should be used if the organisms are resistant to penicillin. The response to antibiotics is often slow and treatment must be continued for some weeks until there is no further clinical or radiological improvement.

(2) *Postural Drainage*.—This should be carried out for periods of up to one hour three or four times a day with the patient placed in such a position that the abscess lies above the appropriate draining bronchus.

(3) At the same time, percussion over the site of the abscess should be carried out to encourage drainage.

Conservative treatment is continued whilst there is clinical and radiological evidence of improvement, or until symptoms have disappeared and the X-ray is clear.

Surgical treatment is indicated in those cases where resolution does not occur with the above regime. In the majority of cases a formal resection (lobectomy) is possible. External drainage of the abscess—once the standard operation—is now only occasionally required in patients who show no response to chemotherapy and who remain too ill for resection.

Lung Cysts

These may be divided into four groups :

(1) *Epithelial Cysts*.—These are developmental in origin and may be solitary and large or multiple and small. They are lined by respiratory epithelium and may have traces of cartilage, muscle or glands in their wall.

(2) *Emphysematous Cysts*.—These include a wide variety of conditions where the normal alveolar framework is destroyed or disrupted, resulting in large air spaces which may become further distended with air.

(3) *Parasitic cysts*, of which the commonest is the hydatid.

(4) *Pseudo-cysts*.—Certain inflammatory conditions of the lung result in cavity formation, and in many instances the cavities may closely resemble epithelial cysts. The cavities, when chronic, are often lined by squamous epithelium. These pseudo-cysts may occur in association with staphy-

lococcal pneumonia, pulmonary tuberculosis, or following a lung abscess. Their true nature is often suggested by their history, course, and radiological features, but the distinction is often difficult.



FIG. 871.—Simple congenital epithelial lung cyst containing fluid and air in patient with a congenital pulmonary stenosis. (Note the prominent left pulmonary artery.)

Epithelial Cysts.— These are often associated with other congenital abnormalities (cervical rib, pulmonary stenosis, patent ductus) (fig. 871). They appear as spherical shadows in the X-ray with a thin, sharply defined wall. They may contain air, fluid, or both air and fluid. Symptoms may be produced by:

(1) The size of the cyst which compresses the lung and produces dyspnoea, tightness in the chest, etc. This is particularly so in infants and children where the thorax is

small and the cyst often relatively large.

(2) Infection is common with the production of fever, cough and sputum. Radiography will demonstrate a fluid level in the cyst. Infection is usually readily controlled with antibiotics, but recurrence is frequent and removal indicated.

(3) Hæmorrhage from cysts is usually associated with infection.

Spontaneous pneumothorax is uncommon with epithelial cysts (cf. emphysematous cysts.)

Treatment.—Solitary cysts producing symptoms should be excised. Symptomless cysts can be kept under observation. Infection of multiple cysts should be controlled by antibiotics if excision is not practicable.

Emphysematous Cysts.— Although many cases are acquired and due to degenerative changes in the lung, others cannot be explained on this basis. The latter may be due to a congenital deficiency of the elastic tissue of the lung, or to absence of cartilage in the walls of the bronchus. Either of these conditions may lead to abnormal distension of the lung with rupture of the alveolar walls. Progressive coalescence of alveolar spaces combined with distension with air may result in the development of an enormous cyst (figs. 872 and 873). These cysts have no epithelial lining, and



FIG. 872.—Giant emphysematous cyst of right lung.

when large the remnants of the more resistant bronchi and blood-vessels are seen stretching across the space. The changes may be limited to a small area of the lung, such as a segment or a lobe, or the condition may be generalised and bilateral.

Clinical Features.—The most important symptom is dyspnoea, which is due to a combination of compression of normal lung tissue by the cyst, increase in the dead space, and a poor gaseous exchange. Emphysematous cysts are frequently associated with chronic bronchitis. In such cases, persistent coughing tends to distend the cysts still further, whilst the presence of bronchial secretions impair respiratory function still more.

The presence of bronchitis is a serious complication which renders radical treatment more hazardous.

Spontaneous pneumothorax is a common and serious complication and may be the first presenting feature. Infection and hæmorrhage are unusual, (cf. epithelial cysts).

Treatment.—Emphysematous cysts which are not associated with generalised emphysematous changes should be excised, but if changes are widespread, excision is impracticable. Obliteration by plication with multiple sutures has given good results; it is applicable to multiple cysts and does not remove potentially functioning lung tissue. Removal of the pulmonary autonomic nerves can be combined with the above procedures; its chief value lies in the reduction of bronchial spasm. In cases complicated by a spontaneous pneumothorax, pleurodesis should be carried out to prevent further attacks of this serious complication.

Hydatid Disease of the Lung.—Hydatid disease is endemic in Australia, Iceland, and the Middle East, but sporadic cases occur throughout the world. In Britain the majority of cases come from Wales (see p. 802).

Pulmonary hydatid cysts occur in about 15 per cent. of all cases; the cysts are usually solitary, but multiple and bilateral cysts are not uncommon. The disease is more common in children and young adults.

The cases may present in any of the following ways:

- (1) On routine clinical or radiological examination and without symptoms.
- (2) Dyspnoea, pain or tightness in chest due to the presence of a large cyst.
- (3) Hæmoptysis due to ulceration into a bronchus.
- (4) Expectoration of watery fluid and 'grape skins' due to rupture into the bronchial tree. This event may produce severe distress with suffocating symptoms or even death if the leak is a large one. Anaphylactic shock and urticaria may occur.
- (5) Symptoms due to secondary infection of the cyst, viz. cough, purulent sputum and fever.

Radiology is the most helpful investigation:

- (1) An uncomplicated cyst appears as an almost spherical, sharply defined dense homogeneous opacity.
- (2) Communication with the bronchial tree may result in a crescentic cap of air overlying the cyst (perivesicular pneumo-cyst).
- (3) Rupture of the cyst permits air to enter the cyst and the laminated membrane



FIG. 873.—A giant emphysematous cyst which presented through the wound on opening the chest.

collapses. This floats on any fluid which may be present, producing an irregular projection above the fluid level ('water-lily' appearance) (fig. 874).

(4) Infection results in disintegration of the laminated membrane with appearances simulating a lung abscess.

(5) Varying degrees of pneumonitis, atelectasis, or pleural effusion may accompany any of the above and modify the radiological appearance.



FIG. 874. — Hydatid cyst. 'Water-lily' appearance.

Diagnosis.—The most important point is to remember the condition. A history of residence in an endemic area may support the diagnosis. The intradermal test of Casoni will give a positive result in 75 per cent. of uncomplicated cysts provided fresh hydatid fluid is used. An eosinophilia of over 4 per cent. is suggestive of the condition.

Treatment.—Removal of the cyst is indicated in all cases owing to the considerable risk of serious or even fatal complications. The earlier operation of marsupialisation has been almost completely replaced by thoracotomy with enucleation of the cyst or lobectomy. Enucleation is employed for uncomplicated cysts without significant associated lung damage. Excessive care and utmost gentleness is required to remove the laminated membrane intact without spilling the contents with the risk of infecting the pleura or wound. The adventitia is carefully incised and the laminated membrane will usually slowly extrude itself. It should never be grasped with forceps as it is friable and is sure to tear. Extrusion can be expedited by gentle positive pressure by the anaesthetist. After removal, any bleeding-points or open bronchi are secured and the residual space can be obliterated by mattress sutures if desired. The pleura is drained as some leakage of air is inevitable.

Complicated cysts are best treated by a more formal resection (lobectomy), particularly if there is any surrounding lung damage.

Secondary Pleural Hydatid Disease.—This is an occasional but serious complication of rupture of a cyst into the pleura or of ill-executed surgery. Multiple cysts grow widely throughout the pleura and may invade the chest wall, diaphragm, or mediastinum. Radical pleuropneumonectomy offers the only hope of eradicating the disease.

Hepato-Bronchial Fistula.—This is caused by the rupture of an infected hepatic cyst into the lung. The infected material traverses the subphrenic and pleural spaces (both being obliterated by adhesions) before discharging into the lung. It results in the production of bile stained sputum. Treatment demands a thoracotomy with resection of the affected lobe; the track through the diaphragm must be enlarged to expose the underlying residual cyst which is drained through an independent opening.

PULMONARY OEDEMA

Blood in the lung capillaries is separated from air in the alveolar spaces merely by the thin capillary wall and the alveolar membrane. Gaseous diffusion occurs readily across this membrane and any disturbance of the physico-chemical balance in the blood may permit the transudation of fluid from the capillaries into the alveolar spaces.

Initially, transudation occurs into the interstitial tissues of the lung, producing dyspnoea and cyanosis; later, when fluid reaches the alveoli, thin frothy blood-stained fluid is expectorated, often in considerable quantities. Coarse râles are audible throughout both lungs and radiographs show consolidated areas spreading from each hilum towards the periphery ('bat's wing' appearance).

Causes.—(1) Obstruction to the flow of blood through the left side of the heart as in left ventricular failure and mitral stenosis.

(2) Increased volume of blood reaching the lungs as may occur after excessive transfusion of blood or saline.

(3) Disturbances of osmotic pressure in the blood.

(4) Traumatic, such as follows the inhalation of certain noxious gases (phosgene, mustard gas), hot air or steam.

(5) Inflammatory, as in influenzal pneumonia.

(6) Associated with certain brain injuries and lesions of the brain stem.

Treatment is urgently required along the following lines:

(1) Correction of the precipitating cause.

(2) Support of the heart and circulation with aminophylline (250 mg. intravenously), digitalis (1.0 mg. digoxin intravenously) and oxygen.

(3) Morphine, which should be given intravenously, is particularly effective in left ventricular failure.

(4) Venesection and mersalyl (2 ml. intramuscularly) for hydræmic states.

(5) Positive Pressure Respiration is the most effective method of controlling pulmonary oedema and should not be delayed when the above measures are not immediately effective. Initially it can be employed with a cuffed endotracheal tube, but tracheostomy will be necessary if ventilation required for more than twelve hours.

SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

In a book of this nature it is not possible to give more than an outline of the principles involved in the surgical treatment of pulmonary tuberculosis. Such treatment is usually an incident in the long-term management of the case, and as such can only be satisfactorily determined by consultation and discussion between physician and surgeon.

Principles of Treatment.—(1) *Rest.*—This can be either local or general, and is an essential part of all forms of treatment. General rest is obtained in bed, whilst local rest is achieved by the various methods of collapse therapy, phrenic paralysis, and pneumoperitoneum.

(2) *Chemotherapy.*—The introduction of streptomycin, para-aminosalicylic acid (P.A.S.), and iso-nicotinic acid hydrazide (I.N.A.H.) have revolutionised the treatment of tuberculosis (p. 23). They are employed in the vast majority of cases as a definitive treatment, or as an adjunct to surgical methods of treatment. The drugs should always be given in combination to prevent the emergence of resistant strains of bacteria. Cycloserine, ethionamide and pyrazinamide are more recent antibiotics which can be used for patients with resistant organisms. Side-effects are more common than with the standard drugs and they can usually only be used for relatively short periods. They are particularly valuable for covering surgical procedures in such cases.

(3) *Relaxation.*—Tuberculosis heals by the production of fibrous tissue resulting in the contraction of the diseased area and the collapse and obliteration of the destroyed areas and cavities. Complete contraction and healing is often prevented by anatomical structures (bony chest wall), so that

artificial methods of relaxing the diseased lung are required in many instances. These are divided into the minor and major methods. The former include artificial pneumothorax, phrenic paralysis, and pneumoperitoneum, whilst the latter include thoracoplasty, extrapleural pneumothorax, and plombage.

(4) *Excision of Diseased Lung*.—Prior to the introduction of streptomycin, excision of tuberculous lungs usually met with disaster. Antibiotics used before, and as a cover for operation, have transformed the situation and resection has become the surgical therapy of choice. Resection has the advantage over collapse therapy of removing the major foci of disease, leaving the body with the less formidable task of dealing with the residual disease.

(5) *Drainage of Pus*.—Apart from the drainage of an empyema, this method has little application now, though in the past tuberculous lung cavities were treated by catheter drainage (Monaldi), or open external drainage (cavernostomy).

Management.—All cases should be treated with bed rest and chemotherapy for several months before any active measures are undertaken. In many instances prolonged treatment will result in complete arrest of the disease; in others a tuberculous cavity, a positive sputum, or bronchial damage requires more active treatment. Surgical measures are almost entirely employed for dealing with residual lesions which persist after adequate conservative treatment. Resection is the treatment of choice in nearly 90 per cent. of all cases requiring surgical aid. Thoracoplasty is employed only when the risks of resection are unduly high or the disease too widespread for safe removal.

Apart from the failures of medical treatment and the persistence of unstable foci, resection is indicated in patients with destroyed lobes, in tuberculous bronchiectasis and in large solid lesions (tuberculoma). Where there is an empyema as well, surgical treatment will probably be required.

Thoracoplasty.—This is the standard method of treatment employing major collapse therapy. The operation is now performed infrequently owing to the greater efficiency and safety of lung resection. It is mainly used for cavities in the upper lobe associated with a persistently positive sputum and resistant organisms (figs. 875 and 876), especially where extensive disease makes resection too hazardous.

The aim of the operation is to relax the diseased area from all sides so that it will retract concentrically towards the hilum. Lateral relaxation is produced by removal of the upper ribs whilst apical relaxation is achieved by separating the lung from the apex and the mediastinum. The original operation was introduced by Sauerbruch and consisted of removal of ribs only (lateral thoracoplasty), but this operation has been replaced by Semb's modification which includes apicolysis in addition to rib removal.

The operation is carried out in stages under local or general anaesthesia. A 'J'-shaped incision is used with the vertical limb placed parallel with the spine and 2 inches (5 cm.) from it, extending from the first thoracic spine downwards and curving forwards beneath the angle of the scapula. The trapezius, rhomboids, latissimus dorsi, and serratus anterior are divided in the line of the incision. This permits the elevation of the scapula with the exposure of the upper ribs. Portion

of the third, second, and the whole of the first rib are resected subperiosteally in that order. The apex of the lung is then freed by a combination of blunt and sharp dissection from the apical and mediastinal structures. The intercostal bundles and



FIG. 875.—Chronic tuberculous cavity at the right apex.



FIG. 876.—Same case as in fig. 875 after thoracoplasty.

muscles are divided. The wound is closed without drainage. At subsequent stages further ribs are resected in order to relax the lung to a point immediately below the lowermost limit of the obvious disease. Usually two or three stages carried out at intervals of two weeks are necessary.

Lung Resection.—Since the introduction of streptomycin, resection of tuberculous lesions has been widely practised and has given good results. The operation has the advantage of removing the greater part of the diseased tissue, in leaving no external deformity, and disturbing respiratory function very little. But it cannot be safely employed in those with extensive disease. Before embarking on resection it is necessary to determine by tomography and bronchography whether the lung and bronchi to be left behind are free from disease. Pre-operative bronchoscopy is advisable, and if active tuberculous bronchitis is discovered, resection should be delayed until it is controlled by further chemotherapy. At operation

it is advisable to remove all the obvious tuberculous disease, although small healed nodules may be safely left behind (fig. 877). The remaining lobes must be prevented from over-expanding either by temporary phrenic paralysis or

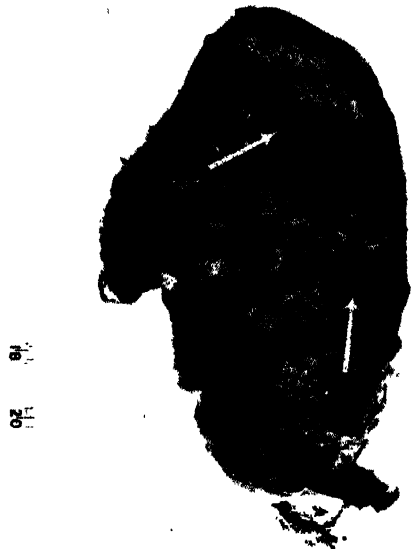


FIG. 877.—Upper lobe of left lung (surgical specimen) showing large cavity at apex (upper arrow) smaller cavity below and tuberculous pneumonia.

by performing a small apical thoracoplasty. Over-distention of the lobe is likely to cause reactivation of dormant foci. It is unwise to carry out a resection if the organisms are resistant to streptomycin.

Other Measures.—Minor collapse procedures, such as artificial pneumothorax, phrenic paralysis, and pneumoperitoneum and variations of the operation of thoracoplasty, such as extra-pleural pneumothorax and plombage, have been rendered obsolete by the continued efficiency of chemotherapeutic agents.

POST-OPERATIVE PULMONARY COMPLICATIONS

The pulmonary complications of general surgery are the cause of serious morbidity and an appreciable mortality, and yet they can be largely prevented by adequate pre-operative and post-operative care.

Predisposing Factors.—(1) *Type of Operation.*—Operations on the upper abdomen and on the upper respiratory tract are more commonly implicated than others, but no operation is exempt. Operations for septic conditions are more prone to be followed by complications.

(2) *Sex.*—Males are more commonly affected than females.

(3) *Age.*—Complications are particularly frequent in the very young and the elderly.

(4) *Chronic Bronchitis.*—Chronic bronchitis is an important predisposing condition.

(5) *Smoking.*—Probably through the production of chronic bronchitis, heavy smokers are more prone to complications than non-smokers. Morton records 60 per cent. of complications occurring in heavy smokers and less than 10 per cent. in non-smokers.

(6) *Anæsthetic.*—The anæsthetic plays some part though this is less than is usually thought. Trauma to the tracheo-bronchial tree should be avoided, and prolonged post-operative unconsciousness is undesirable.

(7) *Pain.*—Post-operative pain is an important factor as it restricts coughing and deep breathing.

(8) Lack of mobility in bed and dehydration predisposes to venous stasis and thrombosis.

Excessive Bronchial Secretions

Abnormal bronchial secretions account for the majority of the chest complications of surgery. Many patients are a little 'chesty' following even the most trivial operation. This 'normal' post-operative bronchitis, however, may lead to very serious and perhaps fatal sequelæ unless adequately controlled.

Preventative Measures.—Adequate time should be devoted to preparing patients for operations of election. This can usefully be done in a regular pre-operative clinic where breathing exercises and coughing instruction is given, dental sepsis is attended to and bronchitis treated by antibiotics. A routine chest X-ray may reveal unsuspected tuberculous or other lesions. Smoking should be forbidden for several days before operation.

The following complications may be encountered, but it should be realised that one may merge into another, or neglect of one frequently leads to the production of more serious conditions.

(1) *Bronchitis.*—This is the commonest complication and is important in that it is often the precursor of more serious lesions. It may arise *de novo* or represent an exacerbation of a pre-existing bronchitis. The attack may vary

considerably from a simple post-operative cough with mucopurulent sputum to severe suppurative bronchitis. Signs in the lungs are generalised, and there are no radiographic changes to be seen.

(2) *Bronchopneumonia* (Aspiration Pneumonia).—This is usually a sequel to the above. Patchy consolidation occurs with more profound systemic changes. The commonest organisms are *Hæmophilus influenzae* and pneumococci but cross-infection with ward organisms such as drug-resistant staphylococci and *P. pyocyaneus* may occur. Their presence should be sought by microscopy and culture of the sputum. Signs are more localised and bronchial breathing may be heard. Radiographs will reveal patchy mottling.

(3) *Atelectasis*.—This is produced by occlusion of a bronchus by viscid secretions of mucus or pus and is usually a sequel to bronchitis. The obstruction leads to absorption atelectasis of the involved lobe. Secondary invasion of the lobe by pathogenic organisms results in particularly serious changes. Depression of the cough reflex by pain or sedation and poor ventilation are important predisposing factors. Atelectasis usually occurs after the second post-operative night, frequently following heavy sedation. Secretions accumulate during sleep, and by morning have blocked the bronchus. The patient does not feel quite so well and there may be slight fever, tachycardia and breathlessness. Cough and sputum may not be obvious as the latter cannot be expectorated. Signs consist of restricted movement of the affected side of the chest with diminished breath-sounds and impaired percussion note over the affected lobe. Radiography will reveal the dense opacity of the atelectatic lobe.

(4) *Lung Abscess*.—This usually follows bronchopneumonia or atelectasis, and is often the result of ineffective early treatment. It is a particularly serious complication often resulting in spreading suppuration throughout the lung by organisms which are by then often resistant to all drugs.

(5) *Empyema*.—This is an occasional complication of any of the above; its management is no different from the average case.

Treatment.—*Prophylactic*.—At the conclusion of an operation the tracheo-bronchial tree should be aspirated if any secretions are suspected. Early return of consciousness and the cough reflex are desirable. Subsequently coughing and breathing exercises combined with regular rolling of the patient from side to side are desirable. Pain should be controlled by repeated small doses of analgesics of which pethidine (50–100 mg.) is the most useful, as it does not depress respiration.

The Established Case.—It is important to encourage the expectoration of sputum by all possible means. Much can be done by the physiotherapist and the nurse, whilst steam inhalations and saline expectorants help to loosen the sputum. When the secretions cannot be adequately removed naturally, they must be removed by catheter suction. If the simpler measures are not soon effective *bronchoscopy* should be carried out. This can readily be performed in the ward if the patient is too ill to move. It should be performed with minimal local anæsthesia so that the cough reflex is

preserved. A careful and unhurried clearance of the whole bronchial tree should be the aim. Bronchoscopy usually results in the aeration of an atelectatic lobe, but the condition may recur and further bronchoscopies be required. In such cases it is often preferable to perform a *tracheostomy*, particularly when secretions are profuse or the patient dyspnoic. The operation is performed under local anæsthesia and can be carried out in the ward in an emergency. Tracheostomy by reducing the 'dead space' greatly improves the efficiency of respiration; it also provides easy access for repeated and atraumatic aspiration of the tracheo-bronchial tree. In addition to post-operative complications it has an important place in the management of severe chest injuries, head injuries, bronchopneumonia and poliomyelitis.

Subphrenic Abscess (p. 873).

Broncho-pleural Fistula

This complication is one peculiar to thoracic operations involving resection of lung tissue. Several factors may be involved in its production.

(1) Poor surgical technique in closing the bronchus (sutures too tight or imperfectly placed).

(2) Infection of the bronchial stump (pyogenic or tuberculous).

(3) Indifferent healing (elderly and debilitated patients, and after deep X-ray therapy).

Symptoms usually appear towards the end of the first week after operation; fever and blood-stained sputum are the first signs. Subsequent developments depend on the size of the fistula.

A small leak allows air to enter the pleural cavity producing a pneumothorax and collapse of the lung whilst pleural fluid enters the bronchial tree giving rise to a persistent cough with much thin blood-stained sputum.

A larger fistula may give rise to serious flooding of the bronchial tree and the development of a tension pneumothorax.

Treatment.—The first essential when a fistula is suspected is to anticipate and prevent flooding of the bronchial tree by early aspiration of fluid from the pleural cavity and by nursing the patient with the affected side down.

A small leak may be controlled by repeated aspirations or by temporary tube drainage until the fistula heals.

Larger fistulæ should be treated at an early stage by re-opening the chest and resuturing the bronchus. The suture line should be reinforced by a pedicled intercostal muscle graft. If the pleural cavity is already infected, however, it is better to drain the pleura and carry out bronchial repair at a later date.

Pulmonary Embolism

Between 2 and 3 per cent. of all hospital deaths are due wholly, or in part, to pulmonary embolism. The condition is by no means limited to surgical wards. The clot most commonly originates in the veins of the calves, thighs or pelvis. Two types of venous lesions occur :

(1) *Thrombophlebitis*.—The thrombosis is secondary to infection or trauma. There is a marked inflammatory reaction and the resulting clot is firmly adherent to the vessel wall and is rarely dislodged.

(2) *Phlebothrombosis*.—Clotting is secondary to venous stasis and there is no inflammatory reaction in the veins. The clot is soft and insecurely attached to the vessel wall. Detachment of the clot is easy, particularly as the condition is often unsuspected and movements uninhibited (see p. 142).

Clinical Features.—Pulmonary embolism is the most dangerous complication of phlebothrombosis. Much depends on the size of the embolus. *Small emboli* may be silent but the cumulative effect of many such episodes leads to a reduction in the pulmonary vascular bed resulting in pulmonary hypertension. Patients require permanent anticoagulants. *Medium emboli* result in pulmonary infarction without any hæmodynamic disturbances. Classically the onset is abrupt with pain in the chest, dyspnoea, and hæmoptysis. Chest radiographs reveal a triangular infarcted segment. Signs of venous thrombosis may or may not be present. Anticoagulants should be administered without delay and the patient immobilised for one week. *Large emboli* may be impacted in the main pulmonary artery or one of its major branches. It produces serious hæmodynamic disturbances and may be rapidly fatal. Confusion with coronary thrombosis is easy but an E.C.G. is often of diagnostic value, showing right ventricular strain in pulmonary infarction. A massive pulmonary embolus may produce immediate death from ventricular fibrillation or complete obstruction of the circulation. Such cases often have an urgent desire for a bed-pan but are dead before one is produced. Those who survive complain of severe præcordial pain, tightness in the chest, and marked dyspnoea. Shock is marked and the patient becomes grey and cold with a rapid, feeble pulse, low blood pressure, and raised venous pressure. Death may occur at any moment, but the longer the patient survives the better are his prospects of recovery.

Treatment.—Every effort is made to support the circulation. The patient is nursed flat and given oxygen; aminophyllin (250 mg.) and coramine (2 to 5 ml.) are given intravenously. Eupavarine (1 mg. intravenously) is given to encourage dilatation of the pulmonary vessels. Anticoagulants are urgently indicated to limit the formation of secondary clot (p. 131). Cardiac arrest should be treated by closed chest cardiac massage. This often serves to dislodge the clot which is then carried deeper into the lung and permits the establishment of some circulation through the lung.

Pulmonary embolectomy is indicated in all severe cases. A supportive cardiopulmonary by-pass is established from femoral vein to femoral artery whilst preparations are made for embolectomy or transfer made to a centre where the operation can be performed. The embolectomy is carried out through a midline sternum-splitting incision which gives rapid and excellent exposure of the pulmonary artery. The latter is opened (with inflow or caval occlusion) and clot removed with sponge-holding forceps and suction. Steps must be taken to remove all secondary clot from the pulmonary arterial tree. Complete or partial occlusion of the inferior vena cava is advocated by some in order to prevent further serious embolism but the operation is still *sub judice* (p. 143).

THE DIAPHRAGM

Herniation of an abdominal viscus through the diaphragm into the chest may occur through a congenital or acquired defect; the former occur at

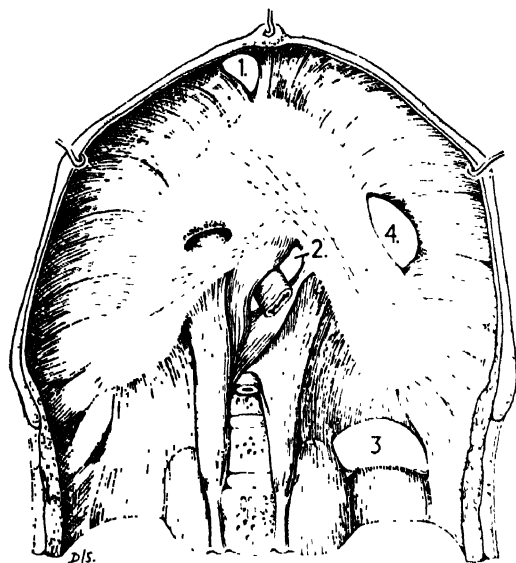


FIG. 878.—The usual sites of congenital diaphragmatic herniæ. 1. Foramen of Morgagni. 2. Oesophageal hiatus. 3. Foramen of Bochdalek (pleuro-peritoneal hernia). 4. Dome. (After Gray.)

certain well-recognised points (fig. 878) and are due to a failure of fusion of the various elements which make up the diaphragm. Such herniæ usually have a well-defined sac and adhesions are uncommon. Other congenital malformations may be present.

The acquired group are either traumatic or post-operative. They may occur anywhere in the diaphragm and extensive adherence to surrounding structures is usual. Stomach, small or large bowel, omentum or spleen are commonly found occupying the hernia. Liver and kidney are less frequently seen.

Symptoms may only occur when either obstruction or strangulation supervene. Many

patients, however, complain of vague dyspeptic symptoms but if the hernia is large, especially in children, respiratory embarrassment may occur. In infants a diaphragmatic hernia is one cause of acute respiratory embarrassment demanding urgent surgical correction. The lung on the affected side is often hypoplastic.

Surgical repair is recommended in all cases in order to avoid serious complications. Access is best obtained through a low thoracotomy incision. The sac is excised and the defect is repaired by a double row of thread or silk sutures. Occasionally reinforcement with fascia lata is required. In infants and small children with large hernias there may be difficulty in accommodating the abdominal viscera in the abdomen.

(1) **Eventration.**—This condition is due to defective development of the whole or part of the muscle of the diaphragm; the latter consists of a sheet of fibrous tissue covered with pleura and peritoneum. The thin, flaccid diaphragm is raised and immobile. Symptoms are uncommon and the condition is usually discovered on routine X-ray. It is important to distinguish eventration from true herniation.

(2) **Oesophageal Hiatus Hernia.**—This is the commonest of congenital abnormalities but may also occur as an acquired condition in the middle-aged or elderly. It is considered on p. 715.

(3) **Hernia through the Foramen of Morgagni.**—The defect lies

between the sternal and costal attachments of the diaphragm; it is more common on the right side (fig. 879).

(4) **Hernia through the Foramen of Bochdalek** (Pleuro-peritoneal Canal).—The canal may remain open with free communication between the

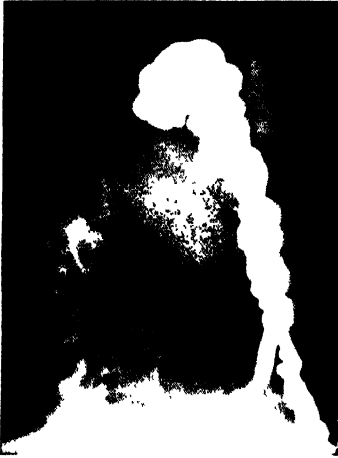


FIG. 879.—Colon occupying a Morgagni hernia. (Dr. Oliver Smith, Birmingham.)



FIG. 880.—A large traumatic diaphragmatic hernia due to penetrating thoraco-abdominal wound with a retained foreign body.

abdominal and pleural cavities. The hernia in such instances has no sac and the abdominal organs move freely in the chest, sometimes nearly filling the hemithorax and displacing the heart to the opposite side. More commonly the canal is covered by pleural or peritoneal membranes but muscle is absent, and in these cases the hernia has a sac and is usually smaller.

(5) **Hernia through the Dome.**—These are usually left-sided and may occur anywhere in the diaphragm. They are commonest, however, at the junction of the muscular and tendinous portions.

(6) **Traumatic Diaphragmatic Hernia.**—The diaphragm may be torn directly by a missile (thoraco-abdominal wound) (fig. 880) or ruptured by severe lower thoracic or abdominal compression (crush injury) without an external wound.

The defect may occur anywhere in the diaphragm in the former instance, but with crush injuries the dome is usually torn. Herniation may occur immediately or be delayed for some considerable time. A peritoneal sac is not present and adhesions between the lung and the herniated viscera always occur. Dyspeptic and obstructive symptoms are common and strangulation occasionally occurs.

Repair of the hernia should always be undertaken.

(7) **Post-operative Diaphragmatic Hernia.**—Occasionally herniation follows thoraco-abdominal operations, particularly those involving an intra-thoracic oesophagogastric anastomosis. Herniation may occur around the new hiatus or through the sutured diaphragm. Great care should be taken

in suturing the diaphragm to the stomach or jejunum in these cases, whilst the diaphragm is best closed with a reasonable overlap of the edges and with two rows of sutures.

Diagnosis may be extremely difficult as there are many other causes of vomiting, pain, etc., in the post-operative period. Radiography is often of little use as the suspected area is obscured by effusion, etc.

An early exploration is advisable if the condition is suspected.

MEDIASTINAL TUMOURS

The term 'tumour' covers a wide variety of conditions which are often difficult to distinguish, but precise diagnosis is not of great practical importance as the majority of innocent 'tumours' require removal, and those which are not amenable to surgery, e.g. secondary carcinoma, lymphosarcoma, lymphadenoma and reticulosis, are usually readily recognised. Innocent 'tumours' should be removed owing to the risks of malignant degeneration or compression or involvement of the mediastinal structures. The most important feature in differential diagnosis is the position of the tumour in the mediastinum, and a useful practical classification is as follows :

(1) Anterior Mediastinal Tumours

Retrosternal goitre.
Thymic tumours and cysts.
Persistent or enlarged thymus.
Teratoma.
Pleuro-pericardial cyst.

(2) Posterior Mediastinal Tumours

Neurogenic tumours (neurofibroma, ganglioneuroma).
Foregut cysts (paratracheal, para-oesophageal cyst, gastric cyst of mediastinum).
Oesophageal hiatus hernia with thoracic stomach.

(3) Tumours arising anywhere in the Mediastinum

Lymphadenopathies (these are often multifocal and the lesions are characteristically lobulated). Secondary carcinoma, tuberculous adenitis, lymphadenoma, reticulosis, and sarcoids are the commonest varieties.

Lymphogenous cysts.
Lipoma.
Fibroma.

Diagnosis in the difficult lymphadenopathies can often be made by a scalene node biopsy or limited mediastinal exploration.

The majority of the solitary lesions require operative removal if serious complications are to be avoided.

Retrosternal Goitre (p. 557).—This produces an anterior mediastinal tumour, often of characteristic shape and distribution. It is frequently bilateral though usually larger on one side. The tumour has a broad base superiorly which merges and cannot be separated from the tissues of the neck on radiological examination. Calcification is not uncommon as the tumour has probably been present for many years. This is one of the few tumours which compress the trachea (scabbard trachea).

Removal through a cervical incision is usually possible, and only occasionally is a sternal-splitting approach required.

Thymic Tumours (p. 574).—Persistence or enlargement of an otherwise normal thymus is not infrequently discovered in young children on radiological examination. Only very occasionally is the thymic enlargement sufficient to produce mediastinal obstruction and stridor (thymic asthma). In such cases deep X-ray therapy should be tried, and if this does not cause a diminution in size, operative removal may be necessary.

A variety of tumours arising in the thymus have been recorded, e.g. lymphosarcoma, lymphadenoma, perithelioma and epithelioma. Of these, only the latter has been found associated with myasthenia gravis, and 10 per cent. of myasthenics are found to have tumours. The epitheliomas are slowly invasive malignant tumours; they are often calcified. Excision is often possible, but recurrence is likely. Relief of myasthenia in cases where this co-exists is less dramatic than in the non-tumour group. Pre-operative irradiation is recommended, and the tumour is removed through the sternal-splitting incision (Keynes) (p. 574).

Teratoma.—The commonest 'inclusion' tumour is the teratoma; true dermoid cysts are only occasionally encountered. The tumours are almost always situated anteriorly with a pedicle attached deeply beneath the aortic arch (fig. 881). They are often slightly lobulated and may project into either side of the chest. Irregular areas of calcification, bone or tooth formation are sometimes seen.

Rapid increase in size without malignant change is sometimes associated with pregnancy. Occasionally a positive Aschheim-Zondek test is obtained in non-pregnant patients. Infection and malignant transformation are not uncommon complications. Removal is indicated in all cases, and is best carried out through a wide lateral thoracotomy.



FIG. 881.—Teratoma of the mediastinum. The tumour lies anteriorly.

Mediastinal Cysts.—These are usually developmental in origin and may be associated with the trachea, bronchi, or œsophagus (foregut cyst), pericardium (pleuro-pericardial, spring-water cyst), or lymph nodes (lymphogenous cysts).

They are characteristically low-tension cysts and so may alter their shape with posture, respiration or after the induction of an artificial pneumothorax.

Foregut cysts are likely to become infected and may rupture into the œsophagus, lung or bronchus. Removal is desirable.

Neurogenic Tumours.—These have already been considered under chest-wall lesions (p. 652). They consist of neurofibromas arising from the intercostal nerves and ganglioneuromas from the sympathetic chain. They lie posteriorly in the costovertebral gutter. Occasionally they extend through the intervertebral foramen into the spinal canal (dumb-bell tumours). Malignant change may occur.

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CHAPTER 30

THE HEART AND PERICARDIUM

WILLIAM P. CLELAND

SPECIAL METHODS OF INVESTIGATION

MODERN cardiac surgery demands precise methods of investigation to provide accurate anatomical and physiological details of the various cardiac abnormalities. It is only when such information is available that a precise diagnosis can be made and surgical treatment planned. The most important available methods are :

(1) **Radiology** (standard techniques and fluoroscopy). Such examinations provide details of the cardiac contour and variations in size of the chambers; it also provides information about the pulmonary circulation.

(2) **Angiocardiography** (figs. 882 and 883).—By the injection of 85 per cent. Hypaque the chambers of the heart and great vessels can be outlined



FIG. 882.—Normal angiogram showing the right side of the heart and pulmonary arteries.

and films taken at frequent intervals during the passage of the opaque material through the heart. Important anatomical information concerning the size and shape of the heart chambers, the presence of a shunt or of obstruction or incompetence of a valve may be provided. The aorta and pulmonary arteries and their branches will also be outlined. Recent progress with ciné angiocardiography holds promise of providing more information about the dynamics of the cardiac circulation than is obtained by the routine methods.

(3) **Cardiac Catheterisation**.—A fine catheter is introduced through a vein into the heart where its passage can be followed by fluoroscopy and

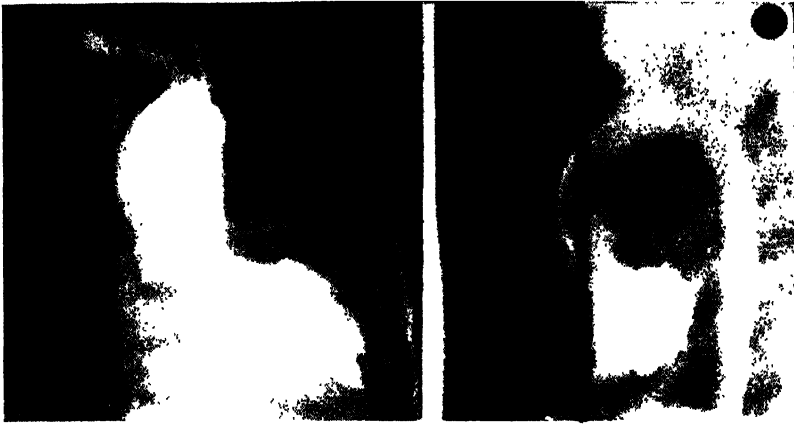


FIG. 883.—Normal angiogram showing the left side of the heart and aorta.

blood samples and pressure tracings recorded. This investigation supplies functional as well as anatomical data (fig. 884).

(4) **Dye Dilution Studies.**—Injection of tracer dyes (Evans' Blue, Coomassie Blue) can be made into the chambers of the heart during cardiac catheterisation and the appearance of the dye in the systemic circulation recorded by an ear oximeter. The time of appearance of the dye and the mode of its disappearance provides valuable information about the presence of shunts or valve incompetence.

CARDIO-RESPIRATORY RESUSCITATION

In emergencies the heart and lungs tend to work as a unit: where there is circulatory failure, both pulmonary and cardiac resuscitation may be required. Although in the early stages of a crisis only one element may be affected, its failure very rapidly implicates the other. Thus, respiratory failure rapidly leads to cardiac arrest and vice versa.

Circulatory failure is common in civil life—electrocution, drowning, asphyxia, poisonous gases and major trauma account for many cases. In addition, circulatory arrest may occur in any hospital department. It is more commonly associated with major surgical procedures especially those performed for cardiac conditions, but minor operations and diagnostic procedures are not immune. If lives are to be saved, it is essential that the mechanism of production and the methods of correction of circulatory arrest should be clearly understood. Not only should vulnerable departments be supplied with the necessary basic instruments for dealing with the emergency, but both medical and nursing personnel should be thoroughly familiar with the procedure of resuscitation.

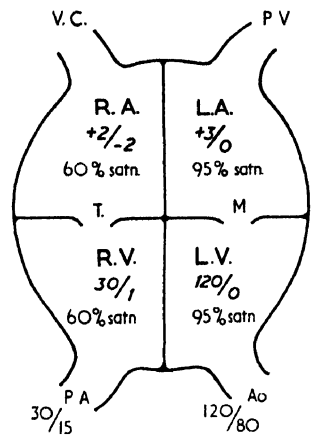


FIG. 884.—Normal intra-cardiac pressures and oxygen saturations.

Causes of Cardiac Arrest.—The following are the factors usually responsible for cardiac arrest :

(1) **Anoxia.**—Myocardial anoxia is probably the most frequent and important precipitating cause of arrest. It can be produced by a variety of means, such as respiratory obstruction, serious hæmorrhage, a sudden severe fall in blood pressure or coronary air embolism. Hæmorrhage from the heart is always more serious than that from the peripheral circulation owing to a marked decrease of cardiac output and rapid fall in blood pressure. Most cardiac manipulations produce a temporary fall in blood pressure, but if they are repeated or prolonged, severe and irreversible changes may develop.

(2) **The State of the Myocardium.**—A healthy myocardium will stand moderate manipulation and anoxia without ill effect, but a diseased myocardium has less reserve, and relatively minor disturbances may cause failure.

(3) **Toxic and Irritating Drugs.**—Certain drugs have a direct depressing or irritating action on the myocardium whilst others affect it indirectly through the circulation. In the latter group, the vasodilators are the most important ; these include many anæsthetic agents, procaine, etc. Chloroform, cyclopropane and adrenaline may produce cardiac irregularities, whilst marked excess of potassium may lead to cardiac arrest.

Types of Cardiac Arrest.—Sudden acute cardiac failure may be produced by :

(1) **Circulatory Obstruction.**—By a massive pulmonary embolism, air embolism, or accidental occlusion of one of the main vessels.

(2) **Asystole.**—This is usually due to severe myocardial depression. The heart beat becomes slow and feeble and finally stops. The myocardium is flabby, dilated, and cyanosed.

(3) **Ventricular Fibrillation** is usually associated with an irritable heart as a result of manipulation, trauma or drugs. In such cases, the myocardium is less depressed and more likely to recover than in the former group.

Management of Cardiac Arrest.—Speedy recognition is essential if the circulation is to be restored before irreversible damage has been done to the brain. Diagnosis is based on three signs (*a*) absent carotid pulse (*b*) absent or gasping respiration (*c*) dilated pupils. Disappearance of the pulse is the most important sign. If the carotid pulse cannot be felt, cardiac arrest must be assumed and appropriate action taken. If the circulation can be restored within five minutes of cessation, recovery is possible, but it is unlikely after an interval of ten minutes.

Management is divided into two phases :

(*a*) **Emergency.**—These measures are carried out by the man on the spot and consist of *mouth to mouth ventilation* and *closed cardiac massage*. These can both be performed by one operator in the ratio of one breath to ten chest compressions, but are more efficiently carried out by two. (Full technical details are available in all first-aid manuals.) These two measures should be continued without interruption until a heart beat or spontaneous respirations return or until expert help arrives. If performed effectively, the vital organs can be kept alive for long periods by these measures alone.

(*b*) **Subsequent Management.**—Further management depends on the arrival of expert help and appropriate equipment (endotracheal tubes, ventilating apparatus, cardiograph, electrical defibrillator and thoracotomy set).

1. **Ventilation:** At the earliest possible moment an endotracheal tube should be passed and mechanical ventilation started.

2. *Cardiograph*: Electrocardiographic monitoring should be provided as soon as possible in order to determine the type of cardiac arrest (asystole or ventricular fibrillation) and demonstrate any signs of recovery.

3. *Cardiac massage*: Closed massage should be continued until cardiography is available. Further action depends on the type of rhythm.

(a) If ventricular fibrillation is present, attempt external defibrillation. If this is unsuccessful, give an intracardiac injection of 100 mg. lignocaine or 250 mg. of procaine amide, or 5 ml. of 10 per cent. calcium chloride, and attempt defibrillation after an interval of two to three minutes. If still unsuccessful, open the left chest and perform open cardiac massage and repeat the procedures outlined above.

(b) If asystole has developed, inject 10 ml. of 1:10,000 adrenaline into heart. This should induce ventricular fibrillation in which case proceed as in 3(a) above. If there is no response, open the chest and carry out open massage and further injection of adrenaline.

(c) **Open Cardiac Massage**.—The chest should be opened through the fifth intercostal space on the left side by an intercostal incision extending from the sternum to the axilla. The wound does not bleed as there is no circulation. The only instrument required to permit bi-manual massage is a rib-spreader, but should this not be immediately available massage is quite possible with one hand. Massage is most efficient with two hands with the pericardium widely opened. One hand should be placed above and the other below the ventricular mass, and massage should be carried out with the flat of the hand and fingers rather than with the finger-tips. The rate should be about 50 times per minute, though this is guided by the need for a sufficient pause to permit diastolic filling. Massage should always be carried out gently as considerable damage to the myocardium is possible if excessive force is used. The object of cardiac massage is to improve the tone of the myocardium, to increase the coronary flow, and ensure a supply of oxygenated blood to the nervous centres. The arrested heart is distended, flaccid, and cyanosed, but after massage it should become smaller, firmer, and pinker. When tone has returned, attempts should be made to obtain normal beating.

4. *Correction of Metabolic Acidosis*: All patients develop acidosis after cardiac arrest and its presence may prevent resuscitation. Sixty ml. of an 8.4 per cent. solution of sodium bicarbonate (1 m.eq. per ml.) should be given intravenously as soon as possible and further doses administered to restore the pH to normal levels (p. 96).

Later Management.—After the restoration of normal rhythm and a normal blood pressure, a careful watch should be maintained in case of recurrence. Adequate steps should be taken to correct any metabolic acidosis. The main late complications are related to cerebral and renal damage resulting from the period of circulatory arrest. If return of consciousness is delayed, cerebral dehydration as described on p. 360 should be carried out. Total body hypothermia with reduction of temperature to 32°C. may help to limit cerebral damage (p. 360). If renal activity is impaired, the measures described under renal anuria (p. 1069) should be instituted.

THE SCOPE OF CARDIAC SURGERY

Operative treatment of cardiac abnormalities is advancing steadily, particularly with the advent of new techniques for controlling the circulation. The procedures can be, for practical purposes, placed in one of three groups:

(1) **Extracardiac Operations.**—These are carried out on the main vessels outside the heart or on the pericardium. The ventricles or atria are not directly interfered with so that cardiac function is not unduly disturbed. Examples include ligation of a patent ductus, excision of coarctation, systemic-pulmonary anastomoses, resection of aneurysms, pericardectomy and certain operations for ischæmic heart disease.

(2) **Closed Intracardiac Operations.**—These are blind intracardiac procedures performed by instrument or finger and controlled by touch. Access is obtained to the interior of the heart through either the ventricular or atrial walls or through the base of one of the great vessels. Cardiac action is interfered with to some extent so that irregularity of heart action is liable to be encountered. These operations are mainly designed for the relief of stenosis of the pulmonary, mitral or aortic valves.

(3) **Open Cardiac Operations.**—The desire of every surgeon is to operate with safety under direct vision on the open and motionless heart. The following procedures have come near to achieving this goal :

(a) *Extra-corporeal Circulation.*—Several clinically proven heart-lung machines are now in regular use. Basically each consists of a pump and an oxygenator. Blood is withdrawn from the venæ cavæ, passed through the oxygenator and returned into the arterial circulation through the femoral artery (fig. 885). The blood is thus diverted completely from the heart and lungs but a good supply of well oxygenated blood is made available to the vital organs. These machines provide the surgeon with longer periods for intracardiac surgery than is available with hypothermia. By the use of an extra-corporeal circulation more complicated cardiac anomalies can be corrected and such conditions as ventricular septal defects, Fallot's tetralogy (p. 697) and valvular incompetence are being so treated.

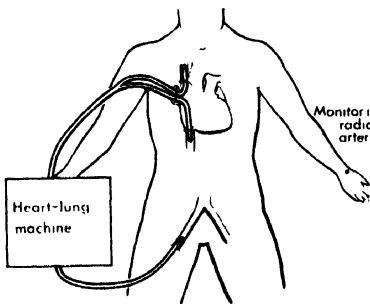


FIG. 885.—Orthodoxy cannulation for heart lung machine.

(b) *Hypothermia.*—By cooling the body to 29 to 30° C. the basic oxygen requirement of vital organs is so reduced that they can withstand periods of up to ten minutes of complete circulatory arrest without suffering irreparable damage. This time is sufficient to enable the surgeon to correct straightforward conditions such as atrial septal defects and pulmonary valve stenosis, but the technique is used less frequently as the efficiency and ease of operation of heart-lung machines increase.

The desired temperature is obtained either by cooling the whole body (surface cooling) by immersion in cold water or by cooled blankets; alternatively the blood alone can be cooled (veno-venous cooling) by cannulation of both venæ cavæ, removing blood from the superior cava, passing it through a cooling chamber and returning it to the inferior cava.

(c) *Deep Hypothermia.*—It is possible by cannulating each side of the heart separately to reduce the body temperature to 10° C. or lower. The heart fibrillates at about 25° C., but the circulation is maintained by separate pumps in each circuit until the desired temperature is reached. The patient's own

lungs are used for oxygenating the blood. At these low temperatures the circulation can be safely stopped for forty-five to sixty minutes.

(d) *Left Heart By-Pass*.—Left atrio-femoral or left heart by-pass is a useful technique which facilitates operations on the thoracic aorta. The left atrium is cannulated and blood diverted with a pump into the femoral artery (fig. 886). The aorta can now be cross-clamped and the left ventricle continues to supply the head and upper extremities with blood whilst the by-pass caters for the lower limbs and abdominal viscera.

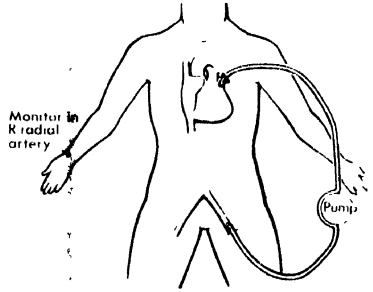


FIG. 886.—Left heart by-pass.

THE PERICARDIUM

Aspiration of the Pericardium.—The pericardium should be explored with a short-bevelled needle introduced alongside the xiphoid process in an upward and backward direction with the needle at an angle of 45 degrees to the skin. This site has the following advantages :

- (a) It does not transgress the pleura.
- (b) It is more likely to encounter fluid which frequently gravitates between the diaphragm and the heart.
- (c) It is less likely to damage the coronary vessels.

Cardiac Tamponade.—A rapid accumulation of fluid in the pericardial sac will compress the heart and prevent diastolic filling ; this results in an increase of the venous pressure and a reduction of the cardiac output. Occurring rapidly (e.g. after injuries or cardiac surgery), it produces a shock-like state and if not relieved may prove fatal. The cardiac dullness and silhouette are enlarged and the heart sounds weak ; the jugular venous pressure is raised and the pulse may be paradoxical (weaker on inspiration). Immediate relief can be obtained by aspiration but, if symptoms recur, pericardial exploration will be required.

Acute Pericarditis.—Purulent pericarditis is now a rare complication of septicaemia, pyaemia, pneumonia or empyema. Signs and symptoms may be vague, consisting of fever, tachycardia or retrosternal pain. A friction rub may be heard and felt. X-rays may show a large pear-shaped cardiac outline, whilst the electrocardiograph shows a characteristic elevation of the S-T segment. The pericardium should be aspirated and suitable antibiotics injected. Drainage of the pericardium is required if aspirations prove ineffective in controlling the infection.

Chronic Pericarditis (Constrictive Pericarditis ; Pick's Disease).—In this condition there is marked thickening, fibrosis and calcification of the pericardium which confines the heart in a rigid inelastic casing preventing it from filling in diastole and emptying in systole. The majority of cases are a sequel to a tuberculous pericarditis, but occasional cases follow purulent pericarditis or a traumatic hæmopericardium.

Hæmodynamics.—Decreased diastolic filling leads to an accumulation of blood on the venous side. The jugular veins are engorged, pleural effusions may occur, the liver is enlarged, and ascites and oedema of the ankles may appear. On the arterial

side, the blood pressure is low with a small pulse volume due to a reduced cardiac output. Fluoroscopy shows decreased or absent cardiac pulsation and pericardial calcification is commonly seen (fig. 887).



FIG. 887.—A calcified pericardium.

Treatment.—Surgical removal of the constricting pericardium is the only effective treatment; this will allow the heart to fill and empty normally.

The patient should be prepared by salt and water restriction, diuretics, acupuncture, Southey's tubes and aspiration of pleural or ascitic fluid. At operation (pericardectomy) it is essential to remove the thickened pericardium from the ventricles, but thickened or calcified plaques covering the atria or venæ cavæ should also be removed (Holman).

CONGENITAL HEART DISEASE

Approximately six babies in every thousand live births have a congenital cardiac abnormality. Many are severe and complicated so that survival beyond a few weeks or months is unlikely. Of those who do survive infancy, the expectation of life is often markedly reduced so that surgical correction is desirable wherever possible.

The lesions are divided into those associated with a normal arterial oxygen saturation (acyanotic group) and those with reduced saturation (cyanotic group).

Acyanotic Congenital Heart Disease

1. PATENT DUCTUS ARTERIOSUS

This is one of the commonest abnormalities, accounting for 15 per cent. of all types. It may occur as an isolated condition (simple patent ductus) or as part of a more complex abnormality (complicated patent ductus). The mechanism of normal closure of the ductus and the causes of patency are not fully understood.

Hæmodynamics—The ductus connects the left pulmonary artery with the aorta just distal to the origin of the left subclavian artery. As the pressure is higher in the aorta than in the pulmonary artery, blood will flow from the former into the latter vessel. As much as 10 to 20 litres of blood per minute, representing two to four times the normal cardiac output, can flow through a patent ductus. On the systemic side, the systolic blood pressure rises and the diastolic pressure falls, producing a water-hammer or collapsing pulse. The amount of blood in the lungs is markedly increased and the pulmonary vessels are dilated (pulmonary plethora) and their pulsation increased (hilar dance). The additional circulation of blood through the lungs and the left side of the heart results in left ventricular hypertrophy.

Clinical Features.—Many cases are only discovered on routine examination in childhood, but a minority give rise to cardiac failure during the first

year. Soon after birth, when the pressures in the pulmonary and systemic circuits are nearly equal, only a systolic murmur is audible and diagnosis is difficult. As the child grows, the difference between pressures increases and the typical continuous machinery murmur develops; this is best heard in the second and third spaces to the left of the sternum. Breathlessness on exertion and retardation of growth may be present in childhood, but symptoms are often absent until complications ensue. Fluoroscopy shows enlargement of the left ventricle with markedly enlarged pulsatile pulmonary arteries. The diagnosis is usually obvious from the presence of the continuous murmur, an increased pulse pressure with a water-hammer pulse, and the X-ray findings. Atypical cases should be investigated by cardiac catheterisation.

Complications.—(1) *Cardiac Failure.*—This is the most important complication and probably accounts for one-third of the deaths. Failure is preceded by progressive cardiac enlargement.

(2) *Bacterial Endocarditis.*—Before the advent of antibiotics this was a frequent and fatal complication accounting for one-third of all deaths. Chemotherapy now permits control of the majority, but relapse is likely unless the ductus is ligated.

Prognosis.—The outlook for a simple patent ductus is difficult to determine. The disease is common in children and rare in adults. It is unlikely that a patent ductus ever closes spontaneously, and it must be assumed that some patients die from cardiac failure in early adult life.

Treatment.—The results of surgical closure are so satisfactory and the mortality rate is at such a level (less than 2 per cent.) that ligation can be recommended in all cases. Recanalisation is rare. Division and suture is practised by Gross and others in order to reduce the risks of recanalisation, but is probably only necessary for the short wide ductus which cannot be safely ligated.

The operation is carried out through a left-sided postero-lateral incision through the fourth space. The mediastinal pleura is incised over the aortic arch and the vagus nerve and its recurrent branch are retracted posteriorly. The areolar tissue is removed from the surface of the ductus and the latter gently mobilised by blunt dissection. Occlusion is achieved by using two stout non-absorbable ligatures placed at either end of the ductus with a transfixion ligature between (Blalock).

II. COARCTATION OF AORTA

Narrowing of the aorta may occur at any site, but, in the vast majority of cases, the stenosis lies immediately beyond the origin of the left subclavian artery in close relationship to the ductus or ligamentum arteriosum. The lesion accounts for 6 per cent. of all congenital cardiac anomalies.

Two main types are described according to the relationship of the ductus to the coarctation:

(1) **Post-ductal Type. Adult Coarctation.**—Sixty per cent. of all cases are of this type. The ductus is patent in a minority and enters the aorta above the coarctation. The stenosis is abrupt with an internal lumen of 1 to 2 mm. Externally the aorta shows a characteristic indentation.

(2) **Pre-ductal Type. Infantile Coarctation.**—Here a patent duct enters the aorta below the coarctation. Unoxygenated blood may pass from the pulmonary artery into the aorta and thence to the lower trunk and extremities. These areas are cyanosed, whilst the head, neck and upper extremities are pink (differential

Robert Edward Gross, Contemporary. Ladd Professor of Children's Surgery, Harvard University Medical School Boston, U.S.A.
Frederic Blalock, 1899-1964. Professor Emeritus of Surgery, Johns Hopkins University, Baltimore.

cyanosis). The stenosed aortic segment may be long and include one or more of the great vessels. Forty per cent. of patients have other cardiac lesions. The mortality in the first year of life is considerable.

Clinical Features.—The obstruction results in hypertension above and hypotension below the constriction with collateral vessels linking the two areas. Symptoms can be divided into three groups :

(1) Those of hypertension above the block ; headaches, irritability, excessive heart action, throbbing and pulsation in head and neck.

(2) Those due to hypotension below the block ; cold legs and intermittent claudication.

(3) Complications secondary to hypertension ; left ventricular failure, intracranial hæmorrhage, rupture of the aorta.

Characteristically there is marked pulsation of the carotid and subclavian arteries with evidence of a left ventricular enlargement. Systolic murmurs are frequently audible over the præcordium and elsewhere over the thorax, due usually to the collateral vessels, although sometimes caused by associated aortic valve disease. Blood pressure in the arms is considerably elevated. Collateral vessels can be seen and felt over the thorax, particularly in the scapular region. Femoral, dorsalis pedis and posterior tibial pulses may be absent or weak. The femoral pulse, if present, is delayed when compared with the radial. Oscillometry reveals defective pulsation in the lower limbs. X-rays show evidence of left ventricular enlargement, a prominent ascending aorta and an absent or abnormal aortic knuckle (fig. 888).



FIG. 888.—Coarctation of aorta showing a prominent ascending aorta, double aortic knuckle and rib notching.



FIG. 889.—Coarctation of aorta. Aortogram showing the stenosis and the marked collateral vessels.

Notching of the ribs due to large tortuous intercostal vessels is seen in adults but rarely in children.

Investigations.—Venous angiocardigraphy or retrograde aortography (fig. 889) are employed to demonstrate the actual site and type of the

coarctation, but these investigations are by no means essential pre-operatively if an aortic graft is available at the time of operation.

Prognosis.—The condition is a serious one and it is estimated that very few patients survive beyond the age of forty-five years; the majority die from the effects of hypertension.

Treatment.—Both Crafoord and Gross independently in 1945 demonstrated that the stenosis could be excised and an end-to-end anastomosis safely performed. The operation is now a well-accepted one and the mortality should not be above 10 per cent. In the majority, excision with an end-to-end anastomosis is possible. Occasionally a graft is required to bridge a wide defect in cases with a long hypoplastic segment or with a post-stenotic aneurysm. Aortic homografts were originally employed but they calcify and narrow with age and have been superseded by grafts of crimped Teflon or Dacron (p. 127). All children and young adults should be operated upon. The results in children are excellent with the blood pressure usually returning to normal. In adults the blood pressure rarely falls to normal levels but the reduction is nevertheless important; symptoms are relieved and the risks of serious or fatal complications reduced.

The operation is carried out through a left postero-lateral incision through the fourth intercostal space. The large anastomotic vessels in the chest wall are ligated.

The aorta is mobilised widely both above and below the constriction, great care being taken not to injure the large thin-walled intercostal and mediastinal vessels arising from the distal segment. The ligamentum arteriosum is divided: the aorta is clamped above and below the constriction and the latter excised (fig. 890). An end-to-end anastomosis is performed using eversion sutures of fine (000) silk. The clamps should be released slowly and any leak from the suture-line controlled by pressure.

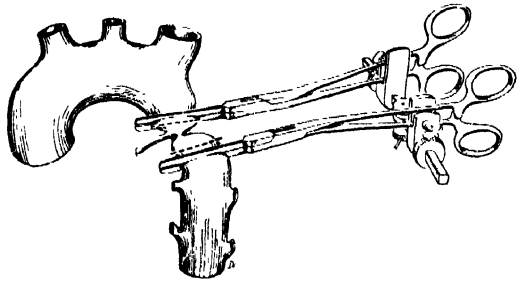


FIG. 890.—Coarctation of aorta. The mobilised aorta is clamped prior to excision and anastomosis.

Results of the Operation.—The over-all mortality rate for all cases is under 10 per cent., but in children it is considerably lower. A normal blood pressure in both upper and lower limbs is usual in children; in adults some elevation in blood pressure usually persists, but the pressure in the lower limbs often rises above that in the upper limbs and the femoral pulse is no longer delayed. Relief of symptoms is the rule.

III. PURE PULMONARY STENOSIS

The stenosis is usually at the valve but occasionally the subvalvar or infundibular region is the site of the obstruction. In the valvar type the cusps are fused to form a dome with a central orifice of 0.5 to 1 cm. diameter. The pressure in the pulmonary artery is low, whilst that in the right ventricle is considerably raised. The stenosis gives rise to a coarse systolic murmur and harsh thrill, maximal in the second and third left intercostal spaces. The right ventricle is enlarged and active. There is no intracardiac shunt so that cyanosis does not develop. The chief symptom is dyspnoea on exertion due to insufficient blood reaching the lungs. X-

rays show enlargement of the right ventricle and of the main pulmonary artery (post-stenotic dilatation). The obstruction can be relieved by closed pulmonary valvotomy (Brock). The valve is approached through the right ventricle and incised with valvotomes and dilated. Pressures should be taken before and after valvotomy to ensure that relief has been adequate. More accurate correction of the deformity is possible by open operations with extracorporeal circulation. Mild cases without symptoms or evidence of right ventricular strain rarely require operation, but severer ones should be subjected to valvotomy owing to the risk of cardiac failure, which is especially common during and after adolescence.

IV. ATRIAL SEPTAL DEFECT

Defects of the septum between the atria account for 7 per cent. of congenital cardiac anomalies; they allow blood to flow from the left to the right atrium (L-R shunt) so that the right side of the heart and lungs are over-filled whilst the left side receives less blood than usual. Although the deformity may cause little disability during childhood and early adult life, it is a serious condition with an average expectation of life in the untreated patient of thirty-five years.

Signs are due to over-filling and over-activity of the right side of the heart and lungs. The right ventricle is palpably enlarged and active. The pulmonary arteries are dilated and actively pulsatile (hilar dance) and increased flow produces a pulmonary systolic murmur. The second heart sound is widely split due to bundle branch block. The E.C.G. shows right bundle branch block and right ventricular hypertrophy.

Closure of the defect is readily performed by direct suture under vision with the aid of hypothermia or extracorporeal circulation.

V. VENTRICULAR SEPTAL DEFECT

Isolated defects of the ventricular septum are one of the commonest congenital lesions (22 per cent.). In addition, they occur frequently as part of certain complex anomalies (Fallot's tetralogy, transposition, etc.).

The defect is usually 1 to 3 cm. in diameter, most often situated in the fibrous part of the septum close to the aortic and tricuspid valves.

The defect allows the passage of blood from the left to the right ventricle resulting in over-filling of the right heart.

The lesion is a serious one, over 50 per cent. of patients dying during the first few months of life.

The defect can be repaired with the aid of an extracorporeal circulation or deep hypothermia using either direct sutures or inserting a patch of Teflon or pericardium.

The operative mortality is 10 per cent., and the most serious complications are due to the production of complete heart block or incomplete closure of the defect.

Cyanotic Congenital Heart Disease

The cyanotic heart lesions include a number of different abnormalities, the commonest of which is Fallot's tetralogy (fig. 891). The abnormalities are multiple and complex and often difficult to determine accurately, but all have central cyanosis due to an intracardiac shunt from the right to the left side. From the surgical point of view the state of the pulmonary cir-

ulation is by far the most important consideration. If this is defective (pulmonary ischaemia), then surgical means can be taken to improve it and thereby increase exercise tolerance and decrease cyanosis. If, however, the lungs are overfilled (pulmonary plethora), or the pressure considerably raised (pulmonary hypertension) it may be difficult or impossible to carry out a corrective procedure with safety. The exact type of surgical procedure will depend on obtaining accurate anatomical details of the abnormality by angiocardiology and cardiac catheterisation.

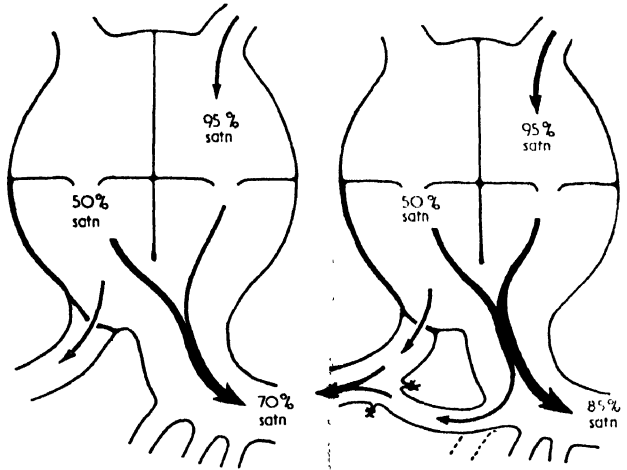


FIG. 891.—Hæmodynamics of Fallot's tetralogy before and after Blalock's operation. Note the ventricular septal defect, the over-riding aorta, pulmonary stenosis and right ventricular hypertrophy which make up the tetralogy (cf. fig. 884).

Fallot's Tetralogy

This is the commonest of the cyanotic conditions and accounts for about 10 per cent. of all cases of congenital heart disease.

The tetrad originally described by Fallot in 1888 consists of (1) stenosis of the pulmonary tract; (2) a ventricular septal defect; (3) an aorta which straddles the ventricular defect and over-rides both ventricles and (4) right ventricular hypertrophy.

The patient is usually undersized and mentally retarded, with central cyanosis and finger clubbing. The heart is small and quiet and there is a systolic murmur and thrill in the pulmonary area. All degrees of severity are encountered from those who are only cyanosed with exercise, to others who are deeply and permanently cyanosed in whom the arterial oxygen saturation rarely rises above 60 per cent. The mortality in infancy in this latter group is considerable, but those of less severity may survive into adult life.

All patients require careful investigation with cardiac catheterisation, angiocardiology and dye studies to provide accurate anatomical details of the lesion and indicate the most favourable type of operation.

The surgical management of patients with Fallot's tetralogy can be considered under three main headings—anastomotic procedures, closed direct operations on the pulmonary valve and infundibulum and open complete corrective procedures. The choice of operation is often difficult; there is much to be said in favour of the complete corrective procedures, and these are indicated for the less severe examples of the condition in children over the age of five years. In severe cases and young children the risks of the complete

operation are at present high and either an anastomotic or closed operation is preferred initially to be followed after an interval of one or two years by the complete corrective operation.

(1) **Anastomotic Procedures.**—These aim at increasing the blood flow to the lungs by creating a shunt between the aorta or one of its branches and the pulmonary artery. The Blalock operation is the most popular and consists of an anastomosis between the left subclavian artery and the left pulmonary artery.

A left posterolateral thoracotomy through the fourth space gives good access. The left pulmonary artery is mobilised and doubly clamped as it lies in the lung hilum. The left subclavian artery is fully mobilised and divided near the first rib. The proximal end of the subclavian is then anastomosed to the upper border of the pulmonary artery with fine silk (fig. 892). A continuous thrill can be felt over the anastomosis on releasing the clamps.

The Potts operation consists of a side-to-side anastomosis between the aorta and the left pulmonary artery. A specially designed Potts anastomosis clamp is used on the aorta so that a lumen is preserved in the latter during the operation to supply the lower half of the body with blood. The anastomotic opening should not exceed 4 mm. if pulmonary engorgement and heart failure are to be avoided. This operation is used mainly in infants and in those where the Blalock operation is impracticable for anatomical reasons.

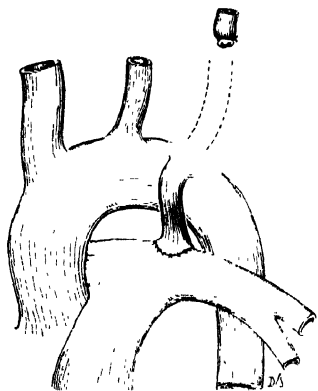


FIG. 892.—Subclavian-pulmonary anastomosis (Blalock) for Fallot's tetralogy.

(2) **Direct Closed Operation.**—This operation has been largely developed by Brock and aims at relieving the obstruction in the right ventricular outflow tract and pulmonary valve by direct means.

The heart is exposed by a left anterior thoracotomy through the bed of the fourth rib. The right ventricle is explored with a cannula capable of recording pressures. The obstruction is carefully assessed and relieved by valvulotomes, expanding dilators and punch forceps. This procedure is now recommended by Brock as a preliminary to complete open correction with the second stage performed two years after the first.

(3) **Complete Open Correction.**—This is now a practicable procedure with the aid of a heart-lung machine or deep hypothermia. It has much to recommend it, but in the young child and the more severe examples of the condition the operative risks are at present too high.

The operation is performed through a vertical sternum-splitting incision. The venæ cavæ are cannulated and the blood diverted through an artificial oxygenator and returned to the femoral artery. With the heart devoid of blood the right ventricle is opened, the ventricular defect is repaired by direct suture or with a patch, pulmonary valve stenosis is relieved and hypertrophied muscle beneath the valve excised.

The operative mortality is 10–15 per cent. but the survivors should be completely cured with a normal circulation.

ACQUIRED HEART DISEASE

Inflammatory and degenerative changes are responsible for the majority of the acquired lesions of surgical interest although cardiac injuries and

umours constitute a small but important group. Rheumatism has a predilection for the aortic and mitral valves, syphilis for the aortic valve and aortic wall, whilst degenerative lesions are frequent in the aortic valve, coronary vessels and myocardium.

Mitral Stenosis

Mitral valvulitis is the commonest sequel of rheumatic carditis. The original attack of rheumatism may be so mild as to pass unnoticed. Only half the patients with mitral stenosis give a history of rheumatic fever or chorea. The condition affects women more frequently than men, and symptoms are commonest during the third and fourth decades.

Pathology.—The cardiac manifestations of rheumatism are widespread with involvement of all three layers, but it is mainly the mitral and aortic valve involvement which leads to later disturbances. The essential lesion is the Aschoff nodule. These produce swelling and roughening of the valve cusps and fibrin is deposited on the roughened surfaces. Later organisation with fibrosis and calcification may occur. As a result the valve leaflets become fused, thickened, rigid and immobile. All grades are encountered from simple fusion of the commissures and mild fibrosis (but with preservation of mobility) to grossly calcified, fixed and functionless valves. The valve orifice is oval or slit-like with an average opening of 1 cm. in its long axis in those patients submitted for operation.

Clinical Features.—These can be briefly considered here under four headings :

(1) Those due to a low cardiac output: tiredness, shortness of breath on exertion, a small volume pulse and pale, cold extremities.

(2) Those due to disturbances of the pulmonary circulation: exertional dyspnoea, pulmonary oedema, nocturnal dyspnoea or asthma, hæmoptysis and bronchitis.

(3) Those due to failure of the right ventricle: congestive heart failure with engorged veins, large liver, ascites and oedema.

(4) Systemic embolism from clot dislodged from the left atrial appendage.

In all cases a full cardiological examination and investigation are required, not only to confirm the diagnosis but to determine the presence or absence of other cardiological abnormalities (fig. 893).

Indications for Operation.—Every case of mitral stenosis with symptoms which are beginning to cause inconvenience should be carefully considered from the point of view of operation.

The following factors will influence any decision regarding surgical treatment :

(1) **Active Carditis.**—Any suggestion of active rheumatic carditis constitutes an

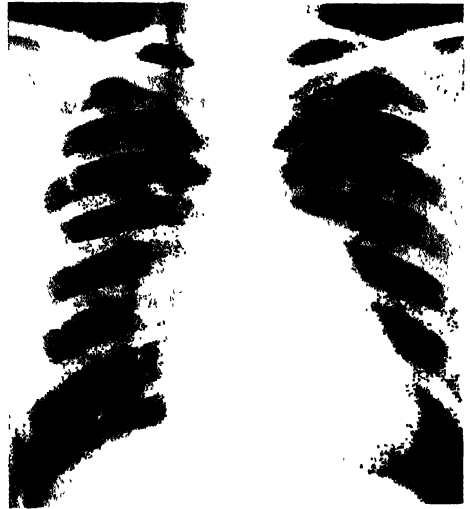


FIG. 893.—Mitral stenosis showing enlargement of the 'pulmonary bay' and the pulmonary arteries.

absolute, but perhaps temporary, contraindication to surgical treatment. Such evidence as fever, a raised E.S.R., unexplained tachycardia, unexplained recent cardiac enlargement, and a recent history of respiratory infection and joint pains all call for a postponement of operation.

(2) **Age.**—The ideal age for operation lies between twenty and fifty; below twenty the chances of further rheumatic activity are appreciable, and surgery should be delayed if practicable, but should never be withheld in severe cases. Over the age of fifty it is important to make sure that symptoms are due to mitral stenosis and not the result of myocardial ischaemia, hypertension, bronchitis and emphysema, etc.

(3) **Other Valve Abnormalities.**—Aortic stenosis, aortic incompetence, and mitral incompetence may be associated with mitral stenosis. They produce left ventricular hypertrophy which does not occur with uncomplicated mitral stenosis. Diagnosis and assessment in these cases with multiple valve lesions is sometimes extremely difficult and may require elaborate investigations. Where mitral stenosis is the dominant lesion, mitral valvotomy should be carried out, but if other valvular lesions are playing a significant part in the dynamics and symptomatology, mitral valvotomy may give disappointing results.

(4) **Mobility of the Mitral Valve.**—Best results are obtained when the mitral valve is still mobile where, after valvotomy, reasonable restoration of function can be expected. A mobile valve is suggested by marked accentuation of the first sound at the apex, and the presence of an opening snap. Rigidity is suggested by a poor first sound, the absence of an opening snap and calcification of the mitral valve.

(5) **Auricular Fibrillation.**—The operative risks are slightly higher, and the post-operative results slightly inferior in cases with auricular fibrillation. This is probably merely an indication of more severe myocardial damage in these cases.

(6) **Embolism.**—The majority of emboli are associated with auricular fibrillation; they constitute a severe complication and account for a significant number of deaths in the medical series. Cerebral, aortic bifurcation and femoral emboli are both frequent and serious. Multiple emboli are common and the chances of further emboli increase steeply with each incident. In the majority of instances the clots develop in the left atrial appendage. Valvotomy is urgently indicated to relieve stagnation in the left atrium with the removal of the appendage at the same time.

(7) **Pregnancy.**—Mitral valvotomy should always be considered in cases where symptoms of mitral stenosis are aggravated by pregnancy. Mild cases can usually be nursed through pregnancy without difficulty, though there are risks of pulmonary oedema and serious pulmonary haemorrhage even in these cases. More severe cases within the first three months of pregnancy can be treated by abortion or valvotomy, but after three months mitral valvotomy is preferable to termination of the pregnancy. After the seventh month of pregnancy, the chances of continuing to term are reasonable with rest, etc., and mitral valvotomy can be deferred until after delivery. Sterilisation should never be considered until the possibility of relief by valvotomy has been fully explored.

(8) **Size of Heart.**—The size of the heart, and particularly the size of the right ventricle, indicates the degree of strain thrown upon the heart and also the state of the myocardium. Operative risks in patients with large hearts are considerable, and even should a successful valvotomy be performed, subsequent improvement may be disappointing unless the myocardium is capable of considerable recovery.

(9) **Congestive Heart Failure.**—Persistent congestive heart failure is an indication that the heart has broken down completely and is unlikely to improve if a valvotomy is performed. The operative risks are high in this group.

Treatment.—Careful pre-operative preparation, with a period of rest, fluid and salt restriction, diuretics and breathing exercises is necessary. All cases should be digitalised as there is a 30 per cent. risk of auricular fibrillation developing in the post-operative period in cases in normal rhythm. Bronchopulmonary infections should be relieved by antibiotics. Patients with a history of embolism and those with pulmonary hypertension should be given anticoagulants (Dindevan) for several weeks before operation. The

anticoagulants should be continued through the operation into the post-operative period.

The operation is performed through an antero-lateral or lateral thoracotomy through the fifth intercostal space. The pericardium is opened widely by an incision parallel with the phrenic nerve. If there is clot in the appendage or atrium, the great vessels arising from the aortic arch should be isolated so that they can be temporarily occluded during intracardiac manipulations to reduce the risks of a cerebral embolus. The appendage can be controlled with an auricular clamp or with a purse-string suture placed around its base controlled by a special tourniquet (Rumel). The index finger enters the atrium through an incision in the appendage. An attempt should be made to split both commissures fully with the index finger with the production of an orifice of 3 or more centimetres. Many surgeons prefer instrumental valvotomy with an expanding dilator (fig. 894) inserted through a stab incision near the apex of the left ventricle. The right index finger remains in the atrium to guide and control the dilator. The appendage is amputated at the completion of the operation, the pericardium loosely closed and the chest drained.

All intracardiac manipulations should be carried out gently and carefully with adequate pauses between stages to allow the heart to recuperate.

In the post-operative period the most troublesome complications are :

- (1) Systemic embolism.
- (2) Suppurative bronchitis.
- (3) Cardiac failure.
- (4) Pulmonary embolism.

All peripheral vessels should be palpated at the end of operation to exclude embolism. Occlusion of one of the major vessels should be treated by immediate embolectomy (p. 131); involvement of smaller vessels is treated by antispasmodics (papaverine, Priscol) and by keeping the affected part locally cool and producing reflex vasodilatation by warming the rest of the body. The majority of emboli occur at the time of operation; late embolism is uncommon.

Active breathing and coughing exercises are started early and full mobility in bed is encouraged. Bronchitis should be treated vigorously with postural coughing and antibiotics.

A close watch is kept on the fluid balance;

if water retention occurs, if the venous pressure rises or if pulmonary oedema is imminent, a mercurial diuretic (mersalyl) should be given.

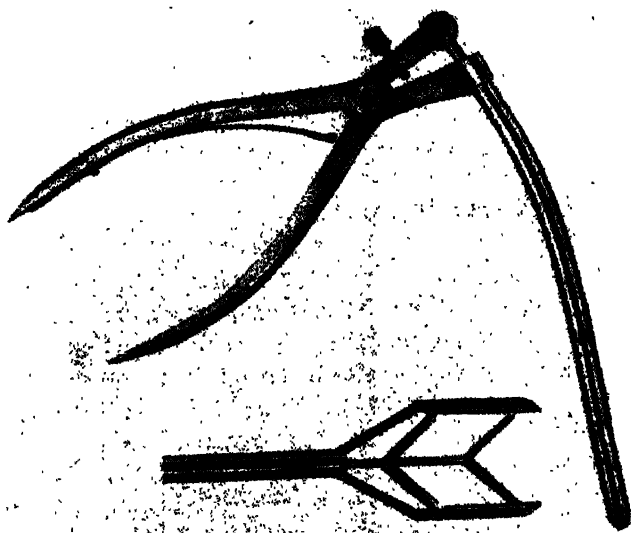


FIG. 894.—Transventricular adjustable mitral valve dilator. (Tubbs.)

Troublesome tachycardia may develop and is usually a sign of under-digitalisation. Small repeated doses of digoxin intravenously will usually control it.

Results of Treatment.—The operative mortality in reasonable cases should not be above 5 per cent., representing the accidents and irreducible hazards of the operation (cerebral embolism, cardiac arrest, auricular tears). Good results are obtained in 75 per cent. of all cases. The majority of the poor results are due to the production of mitral incompetence. About 2 per cent. of patients per annum develop signs of re-stenosis. A second valvotomy should be carried out in favourable cases.

Mitral Regurgitation

This may occur as an isolated lesion or more commonly in association with stenosis. A proportion complicate valvotomy for mitral stenosis and the remainder are developmental in origin.

The lesion impedes the forward flow of blood from the left ventricle leading to left ventricular hypertrophy and pulmonary venous congestion.

Repair of the leaking valve with pericardium is sometimes possible provided the cusps are not severely damaged. Competence can be restored in a dilated valve by annular plication (Wooler). In the majority of cases, however, the valve is grossly diseased and excision with replacement with an artificial ball valve (Starr) offers the only hope of success.

Aortic Valve Disease

The aortic valve may be affected by developmental, inflammatory or degenerative processes, resulting in stenosis, incompetence or combined lesions.

Aortic Stenosis.—The obstruction impedes the forward flow of blood from the left ventricle producing angina, syncope and effort dyspnoea. The left ventricle is hypertrophied and a coarse systolic murmur and thrill are discernible in the aortic area. The arterial pulse is slow-rising and sustained (anacrotic or plateau pulse) and the pulse pressure is small.

The lesion is serious; children are liable to sudden death and adults with symptoms have a life expectancy of only a few years.

Surgery, with the aid of extracorporeal circulation, plays an important role in the management of the condition. *Open valvotomy* is the procedure of choice; congenitally fused cusps can be accurately divided whilst calcified and fibrotic valves can be mobilised and decalcified. *Valve replacement* will be required for grossly destroyed or heavily calcified valves. Artificial valves are prepared from woven Teflon (McGoon), or Teflon impregnated with silastic rubber (Hufnagel). The most efficient artificial valve, however, is the ball valve designed by Starr (fig. 895). Reconstruction of the valve by fascia lata (Senning) and replacement by preserved aortic valve homografts (D. Ross and Barrett-Boyes) is proving an increasingly popular alternative to artificial valve replacement. *Closed valvotomy* with mechanical dilators is occasionally indicated for the seriously ill individual or for those patients with combined aortic and mitral stenosis of rheumatic origin.

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Albert Starr, Contemporary. Professor of Surgery, University of Oregon Medical School, Portland, U.S.A.
Dwight McGoon, Contemporary. Cardiac Surgeon, Mayo Clinic, Rochester, U.S.A.
Charles Hufnagel, Contemporary. Professor of Surgical Research, Georgetown University, Washington.
A. Senning, Contemporary. Professor of Surgery, Zürich, Switzerland.
Donald Nixon Ross, Contemporary. Thoracic Surgeon, Guy's Hospital and National Heart Hospital, London.
B. Barrett-Boyes, Contemporary. Thoracic Surgeon, Auckland, New Zealand.

Aortic Incompetence.—A leaking aortic valve produces considerable left ventricular dilatation in addition to hypertrophy. Symptoms are similar to those of aortic stenosis. In addition to an aortic systolic murmur, a diastolic murmur is audible along the left sternal edge. The pulse is characteristically quick rising (Corrigan or water hammer) with a high systolic and low diastolic pressure resulting in a large pulse pressure.

Isolated cases are capable of correction by suture (tears) or patching (perforations), but the majority are best treated by excision and replacement.

Cardiac Tumours

Tumours of the heart are relatively uncommon; they may occur in the ventricle or atrium. The commonest is the myxoma of the atrium (80 per cent. of all tumours) and of these the majority (75 per cent.) occur in the left atrium.

Left atrial myxomas simulate mitral stenosis; they obstruct the flow of blood through the left atrium producing pulmonary venous engorgement and hypertension. Systemic embolism and postural syncope are important symptoms.

The condition simulates mitral stenosis but is distinguished from it by a shorter and more progressive history, evidence of toxicity (fever, raised E.S.R., anæmia) and disturbances of plasma proteins.

The lesion can be demonstrated by angiography, and can be completely removed with the aid of an extracorporeal circulation.

Disorders of Rhythm

Heart block or complete atrio-ventricular dissociation may occur as a manifestation of certain congenital cardiac lesions, as a complication of ischaemic heart disease or the result of surgical treatment of ventricular or allied defects. In all cases, the atrial impulse fails to reach and stimulate the ventricle owing to interference with the conduction of the Bundle of His. The ventricles beat independently at a rate of 25 to 50 per minute.

The dangers of this situation are three-fold:

- (a) The slow rate impairs cardiac output and may lead to heart failure.
- (b) A sudden slowing or cessation of the beat may produce syncope or death (Stokes-Adams attacks).
- (c) A sudden change from sinus rhythm to heart block may produce syncope.

Although the ventricular rate can sometimes be increased by sympathetic-tonic drugs, of which ephedrine and iso-propyl nor-adrenaline (iso-prenaline) are the most effective, the response to both drugs becomes less with continued use.

Artificial electrical pacemakers have been designed to stimulate the heart



FIG. 895.—Ball and socket aortic valve prosthesis. (Starr.)

^S Dominic John Corrigan, Bart., 1802–80. Dublin Physician.
^P Wilhelm His, Junr., 1863–1934. German Physician.
^V William Stokes, 1804–78. Regius Professor of Medicine, Dublin.
^R Robert Adams, 1791–1875. Regius Professor of Surgery, Dublin.

at any desired rate. These can be *wholly external* using electrodes applied to the surface of the chest; *wholly internal* with a small implantable battery operated pacemaker (fig. 896), which is buried in the abdominal wall and

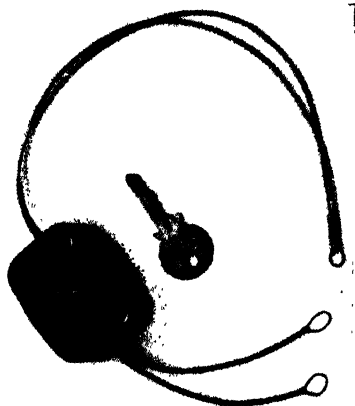


FIG. 896.—Battery-operated implantable pacemaker.

connected to the myocardial surface by wire electrodes, or *combined internal and external*. In the latter the apparatus is external and the heart is stimulated either by a catheter electrode passed along the external jugular vein into the right ventricle, or by wire electrodes attached to the surface of the ventricle. Artificial pacemakers are designed to work for about three years but failure of the battery, leads or electrodes may occur at any time and demand earlier replacement.

Cardiac Ischæmia

There are two aspects of cardiac ischæmia which have a surgical bearing, viz. the relief of the pain of angina pectoris, and the improvement of the blood supply to the myocardium.

Relief of Angina.—The afferent sympathetic fibres from the heart pass through the stellate and upper three or four thoracic ganglia on both sides. Interruption of these pathways relieves anginal pain in many instances.

Alcohol injection of the stellate and upper thoracic ganglia has the attraction of being a simple procedure not normally classed as an operation, but it carries considerable risks. Not only is it difficult to inject the alcohol accurately, but it is easy to inject it into undesirable or dangerous places (lung, spinal canal, brachial plexus). It is not a method which can be generally recommended.

Cervico-dorsal Sympathectomy.—This is performed through either a supra-clavicular or a posterior approach. The former operation is described on p. 140.

The posterior approach is carried out with patient prone with arms hanging down. A paravertebral incision is made to expose the third rib, and a short segment is removed. The parietal pleura is mobilised from the apex to expose the dome and mediastinum. The sympathetic chain is readily identified and the stellate and upper four or five thoracic ganglia are resected. The operation is repeated on the opposite side without moving the patient. The pleural cavity should be drained if opened.

Revascularisation of the Myocardium.—The coronary arteries and their branches are virtually end arteries with practically no communication between them. Occlusion of one artery or a branch usually results in infarction of the cardiac muscle. An infarct not only weakens the ventricle but may act as an irritable focus with the production of ventricular fibrillation.

Much experimental work has been carried out by Beck, O'Shaughnessy and others to overcome the inherent shortcomings of the coronary circulation and thereby improve the blood supply to the heart muscle but no universally satisfactory method has yet been developed.

The procedures employed fall into three categories :

(1) Those which aim at producing pericardial obliteration so that mediastinal vessels can pass through the adhesions into the myocardium. Abrasion of the

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 Laurence O'Shaughnessy, 1900-1940. Surgeon, London County Council Cardiovascular Clinic, London. Died from wounds during the evacuation from Dunkirk.

epicardium and pericardium and powdering with asbestos or talc or painting with phenol or silver nitrate have all been employed.

(2) Those which introduce an extracardiac source of blood to the myocardium;omentum, lung, muscle, and the internal mammary artery (Vineberg) fall into this category.

(3) Direct operations on localised constrictions of the coronary arteries (Effler).

Ventricular Aneurysm.—Myocardial infarcts may lead to such weakening of the ventricular wall as to produce an aneurysm. The presence of the latter grossly impairs left ventricular function by paradoxical pulsation but in addition clots form frequently in the aneurysm and are a constant menace to life.

Aneurysms can be safely treated with the aid of an extracorporeal circulation. The sac is excised and the defect closed with multiple mattress sutures and overlapping edges.

SURGERY OF THE AORTA

The surgery of the aorta has made considerable strides since the introduction of aortic grafts and the advent of hypothermia and methods of cardiopulmonary by-pass. It is now possible to occlude the aorta with safety for reasonable periods, and to bridge considerable defects, so that the two chief deterrents to reconstructive aortic surgery have largely disappeared.

Aortic Aneurysms.—Aortic aneurysms due to syphilis or degenerative changes may be fusiform, saccular (fig. 897) or dissecting. The diagnosis may be straightforward, but is often notoriously difficult, although angio-cardiography has provided a useful diagnostic procedure.



FIG. 897.—Saccular aneurysm of the ascending aorta.

Treatment of Aneurysms

Insertion of Colt's umbrella and Blake-more's wire and wrapping of aneurysms in polythene has been entirely superseded as methods of treatment by more direct procedures made possible by the use of heart-lung by-pass or left atrio-femoral by-pass. (pp. 690 and 691).

(1) *Excision and Repair. Aneurysmorrhaphy (Matas).*—Applicable to smaller saccular aneurysms. Consists of excision of the sac with direct suture of the neck. Not applicable to fusiform aneurysm or saccular aneurysms with a wide neck (fig. 898).

(2) *Excision and Grafting.*—This involves excision of the aneurysm and adjacent aorta and its replacement with a prosthesis (woven, crimped Dacron) (fig. 899). Heart-lung by-pass is required for aneurysms of the ascending aorta or aortic arch, but the simpler left atrio-femoral by-pass is all that is required for aneurysms of the descending thoracic aorta.

(3) *Excision and Plastic Repair.*—The aneurysm is excised and the aortic

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 Effler, *Contemporary. Thoracic Surgeon, Cleveland Clinic, Cleveland, Ohio.*
 George Herbert Colt, 1878-1957. *Surgeon, Royal Infirmary, Aberdeen.*
 Arthur Hendley Blakemore, *Contemporary, Surgeon, Presbyterian Hospital, New York.*
 S.S.—45

defect is repaired by an insert of woven Dacron (fig. 900). This method is applicable to saccular aneurysms of the aortic arch and avoids the elaborate anastomoses required for replacement of the arch.

Dissecting Aneurysms

Produced by a split in the intima and internal portion of the media, allowing blood to enter the media and extend both distally and proximally

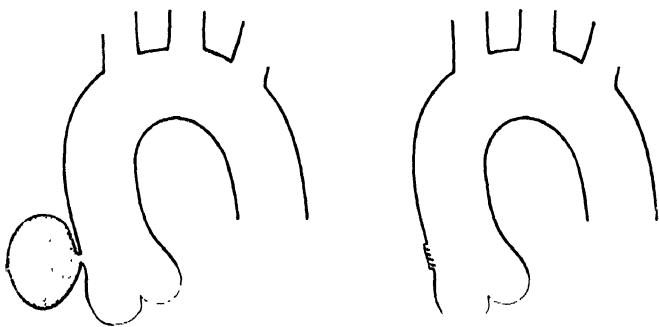
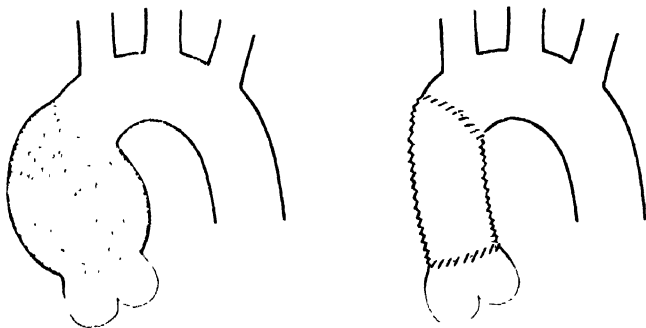


FIG. 898. — Saccular aneurysm. Excision and repair.

FIG. 899. — Fusiform aneurysm of the ascending aorta. Excision and grafting.



resulting in a double lumen aorta. The aneurysms may rupture externally or into the pericardium with fatal results, may undermine and dislodge the attachments of the aortic valve, producing aortic incompetence or occlude vital branches of the aorta (carotid, renal, etc.) with serious consequences. Dissection may be related to systemic hypertension or defective aortic wall as in Marfan's disease.

The lesion is a serious one with a high mortality within a few days of onset, but a minority survive for some years. In the early stages dissection may be limited by the use of hypotensive drugs. Operative treatment with excision of the point of entry and re-anastomosis offers some hope of limiting further dissection and allowing blood to clot in the entire lumen (fig. 901). Some form of by-pass circulation is necessary.

Thrombosis of Aortic Bifurcation (Leriche Syndrome) (pp. 134 and 136).

Dysphagia Lusoria¹ (Vascular Rings).—Abnormal development of the aortic arch and its branches (e.g. double aortic arch, right subclavian arising from descending aorta) may result in obstruction of the trachea and œsophagus by vessels which surround them both in front and behind. The anomaly may pass unnoticed, but occasionally in children severe dyspnoea and stridor and dysphagia may develop and require urgent treatment. Occasionally symptoms only occur later in life due to enlargement of the vessels.

FIG. 900.—Aneurysm of the aortic arch. Localised excision and plastic repair.

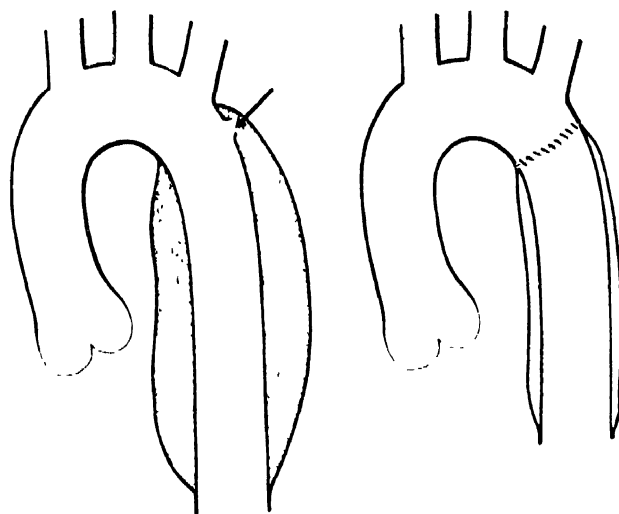
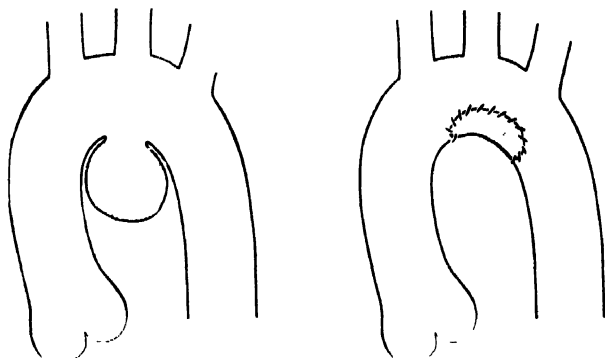


FIG. 901.—Dissecting aneurysm of the descending aorta. Excision of area of rupture and reconstitution of the aorta. The arrow indicates the site of the intima tear.

A barium swallow reveals a posterior indentation of the œsophagus by the abnormal vessel. If symptoms are severe, thoracotomy should be performed and the abnormal vessel divided or transposed.

¹ *Lusus* = game, sport or jest. Condition originally described as '*Lusus naturæ*'—a jest or sport of nature.

CHAPTER 31

THE ŒSOPHAGUS

Surgical Anatomy.—The œsophagus is a fibromuscular tube 25 cm. long occupying the posterior mediastinum and extending from the cricopharyngeal sphincter to the cardia of the stomach; 3 cm. of this tube lies below the diaphragm. The musculature of the upper one-third is mainly striated, giving way to smooth muscle below. It is lined by squamous epithelium which is replaced by specialised epithelium at the level of the hiatus similar to gastric mucosa but without oxyntic and peptic cells (fig. 902). This specialised mucosa lines the lower 3 cm. The parasympathetic nerve supply is mediated by the vagus through an extrinsic and intrinsic plexus. The intrinsic plexus has no Meissner's network which is present elsewhere through the alimentary canal, and Auerbach's plexus is present only in the lower two-thirds.

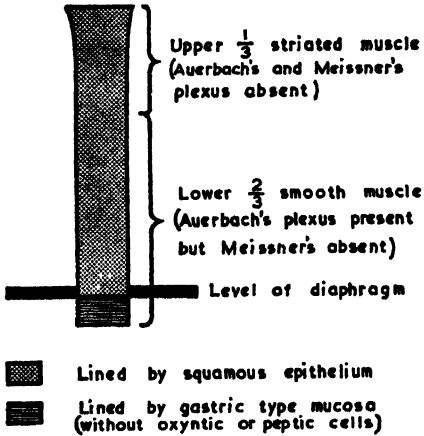


FIG. 902.—The anatomical structure of the œsophagus.

culty may be experienced in the passage of instruments and where foreign bodies may be arrested. They are also the sites of election for innocent strictures and for carcinoma of the œsophagus.

COMMON LESIONS OF ŒSOPHAGUS

		COMMON LESIONS OF ŒSOPHAGUS			
		Neoplasms	Strictures	Arrest of foreign bodies	Miscellaneous
0	incisor teeth				
15 cm.	cricopharyngeal constriction	carcinoma	traumatic strictures.	F. B.	pharyngeal pouch, webs.
25 cm.	aortic and bronchial constriction	carcinoma	peptic or traumatic strictures, congenital stenosis.	F. B.	atresia, traction diverticulum, dysphagia lusoria.
40 cm.	diaphragmatic and "sphincter" constriction	carcinoma	peptic strictures.	F. B.	œsophagitis, (hiatus herniation) achalasia, scleroderma.

FIG. 903.—Common lesions at various levels of the œsophagus.

(R. Celestin, F.R.C.S., Bristol.)

Physiology.—Swallowing is accomplished by two mechanisms, 1, *oropharyngeal* which is voluntary, and 2, *œsophageal*, which is involuntary.

Georg Meissner, 1828–1905. Professor of Physiology, Göttingen, Germany.
Leopold Auerbach, 1828–1897. Professor of Neuropathology, Breslau.

INVESTIGATION OF THE DISEASES OF THE ŒSOPHAGUS

The œsophagus being inaccessible the history is of special importance but the physical signs are of lesser value.

Dysphagia is the term used to describe difficulty (not pain) in swallowing; there are two types—*oropharyngeal* and *œsophageal*. The type of dysphagia is important. It may be dysphagia for solids or fluids, intermittent or progressive, precise or vague in its appreciation. *Pain* may be present. Painful dysphagia is usually due to *œsophagitis*. *Regurgitation*—it is important to record the volume, contents, presence of blood or bile and the reaction to litmus. Loss of weight, *anæmia*, *cachexia* and change of voice are also important symptoms.

Radiography is a most valuable investigation. A *straight film* will show an opaque foreign body and the site of its arrest (figs. 908, 909). A *barium swallow* is essential: screening will show the motility and films, taken as necessary, will delineate size, distortion or the presence of a space-occupying lesion. It is important to take a film with the patient in the prone head-down position to assess the degree of regurgitation.

Œsophagoscopy is essential. Endotracheal anaesthesia with a short-acting relaxant permits of easy access. The key to safety lies in the correct position of the patient on the table. A sandbag is placed behind the third dorsal vertebra in the supine position. An assistant holds the head and controls its position during the various manœuvres of the tube (fig. 904). Every stage must be accomplished under *direct vision*—at no time must the instrument be passed *blindly*.

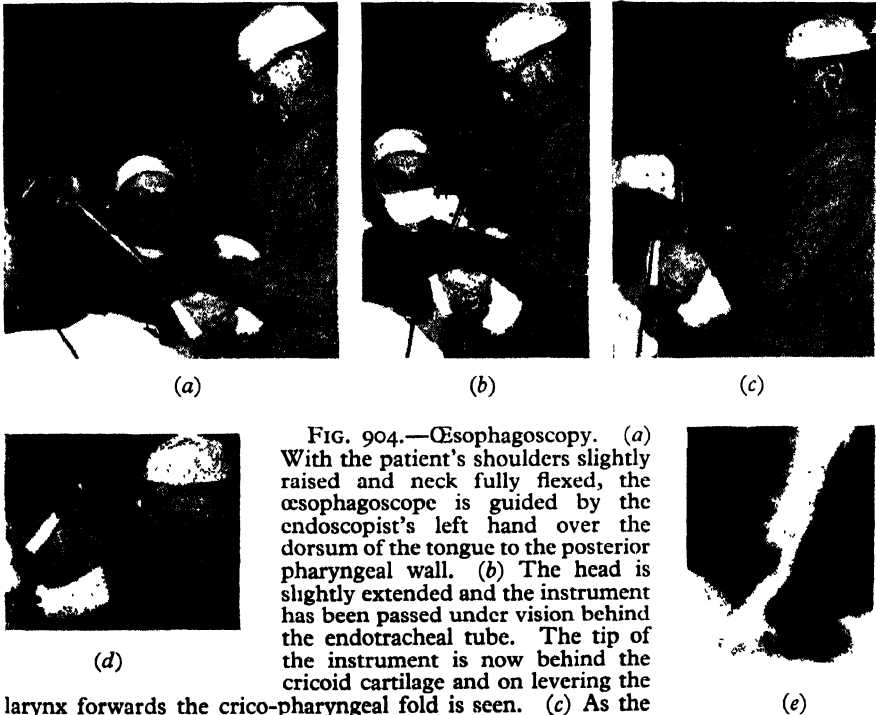


FIG. 904.—Œsophagoscopy. (a) With the patient's shoulders slightly raised and neck fully flexed, the œsophagoscope is guided by the endoscopist's left hand over the dorsum of the tongue to the posterior pharyngeal wall. (b) The head is slightly extended and the instrument has been passed under vision behind the endotracheal tube. The tip of the instrument is now behind the cricoid cartilage and on levering the

larynx forwards the crico-pharyngeal fold is seen. (c) As the head and neck are gradually extended the instrument is advanced under vision. The mid-thoracic œsophagus is now being inspected. (d) The head and neck are now completely extended and the œsophagoscope has advanced until the 40-cm. mark is opposite the upper incisor teeth. At this level the cardia normally comes into view but in this case the œsophageal carcinoma (e) is seen.

(Norman C. Tanner, F.R.C.S., London.)

Adequate suction and a good light are very important. After œsophagoscopy the patient should have nothing to drink for twelve hours. If there is any fever or pain within thirty-six hours of the procedure, massive doses of penicillin should be given. This will usually prevent the development of a retro-pharyngeal cellulitis or mediastinitis from a perforation (p. 712) which is a possible complication.

CONGENITAL ABNORMALITIES

Common abnormalities are:

1. Atresia with or without tracheo-oesophageal fistula.
2. Stenosis.
3. 'Short' oesophagus with hiatus hernia.
4. Congenital achalasia.
5. Dysphagia lusoria.

Congenital atresia of the oesophagus is usually associated with a tracheo-oesophageal fistula. Referring to fig. 905, it will be seen that in 85 per cent. of cases it is the *lower* pouch that communicates with the trachea.

It is highly important to be cognisant of this abnormality, because its recognition within forty-eight hours of birth, and subsequent surgical correction, is the only hope of survival.

Clinical Features.—The new-born babe regurgitates all its first, and every feed. Saliva pours, almost continuously, from its mouth. This is *the* sign of oesophageal atresia—to no other condition does this phenomenon appertain. Attacks of coughing¹ and cyanosis are prone to occur (stomach

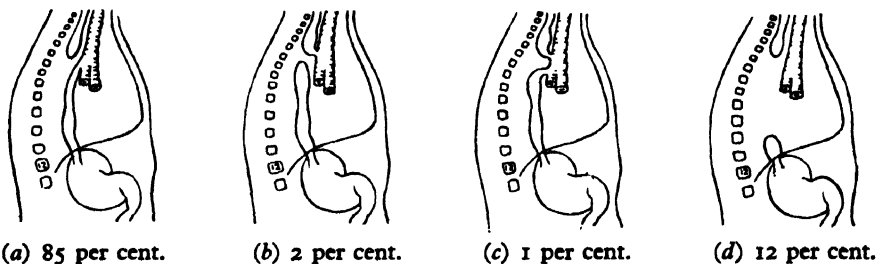


FIG. 905.—Congenital oesophageal atresia. (a) Lower pouch opening into the trachea. (b) Upper pouch opening into the trachea. (c) Both pouches opening into the trachea. (d) Both pouches ending blindly. Usually the mid-oesophagus is missing entirely.

contents regurgitating into the trachea). The abdomen becomes distended (dilatation of the stomach), due to air swallowed from the tracheal fistula (fig. 905 (a)).

Clinical Confirmation of the Diagnosis.—A No. 10 soft rubber catheter is introduced into the oesophagus through the nose. Should an obstruction be encountered about 4 inches (10 cm.) from the nostril, the diagnosis is practically certain.

Radiological Confirmation.—On no account should barium emulsion be given in these cases. Injection of 1 ml. of lipiodol down the catheter will demonstrate the obstruction. During this examination the supine position is advised, because in the rare event of the atresia belonging to categories (b) or (c), the medium is less likely to enter the trachea. In all cases the lipiodol should be aspirated after the radiograph has been taken. Another radiographic finding of significance is air in the stomach and jejunum, which is indicative of a communication between the lower pouch and the trachea.

Pre-operative Treatment.—Surgical intervention is urgent. Aspiration pneumonia is nearly always present and penicillin should be given. Everything by mouth is forbidden.

¹ In type (a) the patient coughs up frothy mucus, perhaps tinged with bile.

Operation.—The most satisfactory anæsthesia is a small dose of relaxant, together with gas and oxygen administered through an endotracheal tube. During the operation about 70 ml. of blood (not more) can be transfused with advantage.

The best approach is through a right-sided thoracotomy incision at the level of the fifth intercostal space (fig. 906). The azygos vein having been divided between ligatures, the upper segment of the œsophagus is located by a catheter within it, and freed gently from the surrounding structures. Having been separated from the trachea, the lower segment is transected just sufficiently far from the abnormal communication to permit the opening in the trachea to be closed (fig. 907 A) without causing a subsequent stricture, 00000 silk on an eyeless round-bodied needle being used for these, and all subsequent, sutures.

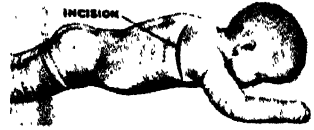


FIG. 906.—Thoracotomy for congenital œsophageal atresia.

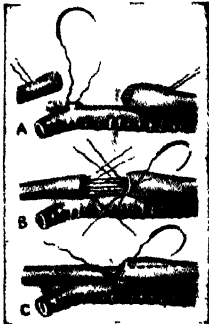


FIG. 907.—Stages in the repair of a tracheo-œsophageal fistula.

An opening is made into the blind upper segment and an anastomosis carried out between this opening and the lower segment. After the first three or four interrupted sutures have been introduced through all layers posteriorly (fig. 907 B), a catheter is introduced downwards into the stomach and upwards into the pharynx, where it is grasped and drawn out of the mouth by the anæsthetist. The presence of this catheter greatly facilitates the completion of the anastomosis (fig. 907 C). The stomach is emptied of air and the catheter is withdrawn through the mouth before closing the thorax. A drainage tube is inserted through an intercostal space and clipped prior to connecting it to a water-sealed bottle. Should leakage subsequently occur, gastrostomy is performed.

When the case belongs to type (d), anastomosis of the two ends being impossible, gastrostomy and bringing the upper pouch out through an incision in the neck, and then opening it, is the best temporary expedient. If all goes well, reconstruction of the œsophagus with a colon transplant can be undertaken at a later date.

Post-operative Care.—A special nurse skilled in aspirating the pharynx, and an oxygen tent-incubator are highly desirable.

Prognosis.—Aspiration pneumonia with acid digestion of the lungs and infection superadded is exceedingly likely to supervene when there has been delay in making the diagnosis. Added to this there is a danger of post-operative leakage from the anastomosis into the pleural cavity, a disaster that is minimised by freeing the distal segment for a considerable distance and thus decreasing tension. These dual complications result in a mortality approximating 40 per cent.

Œsophageal Stenosis.—This is a congenital organic narrowing of the lumen of the œsophagus and is a cause of dysphagia. Commonly it occurs at the same site as atresia and is treated by dilatation. Those in the lower one-third are usually not congenital but due to such conditions as œsophagitis, ulceration or achalasia.

Congenital Short Œsophagus (p. 715).

Dysphagia Lusoria or Œsophageal Compression by an Abnormal Artery.—Exceptionally, the right subclavian artery arises as a branch of the arch of the aorta, and passes either behind or in front of the œsophagus to reach its destination. Another rare abnormality is a double arch of the aorta with the œsophagus and trachea sandwiched between them. Pressure from such abnormal vessels is liable to produce dysphagia. Some cases are remediable by ligation and division of the abnormal artery (p. 707).

FOREIGN BODIES IN THE ŒSOPHAGUS

All manner of swallowed foreign bodies have become arrested in the œsophagus: coins, pins, and dentures (fig. 908) head the list. When the object contains radio-opaque material, an urgent X-ray examination is called for. The presence of a non-opaque foreign body in the œsophagus can

often be confirmed by a barium swallow (fig. 909). Whenever possible the patient should be screened before œsophagoscopy is undertaken.

The foreign body having been visualised (fig. 910), and, if necessary, manipulated into a favourable position, it is grasped with suitable forceps introduced through the œsophagoscope. The œsophagoscope, together with the forceps still grasping the foreign body, is then gently withdrawn.



FIG. 908.—False teeth impacted in œsophagus. (The late Dr. James F. Brailsford Birmingham.)



FIG. 909.—Damson-stone outlined by barium, indicated by the arrow.

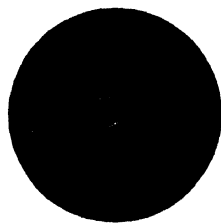


FIG. 910.—A penny in the œsophagus as seen through an œsophagoscope.

INJURIES

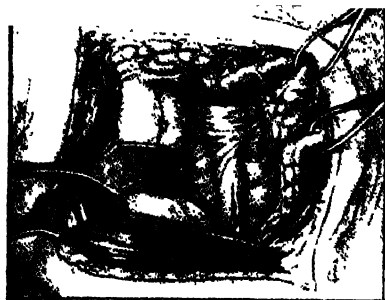
Perforation of the œsophagus can result from the inexperienced use of an œsophagoscope or a gastroscope; usually the beak of the instrument is thrust through the thin posterior wall of the pharynx just above the cricopharyngeal sphincter or the wall is crushed between the shaft of the instrument and a rigid, osteoarthritic cervical spine. Perforation just above an œsophageal stricture can occur from œsophagoscopy or bouginage. It also can result from removal of a piece of growth for biopsy, but probably the most frequent cause of perforation is a sharp foreign body plus instrumentation to remove it.

Often the perforation goes unrecognised by the œsophagoscopist until general distress and great pain on swallowing saliva or fluid, and dyspnœa, become apparent. In such circumstances the possibility of a tear should be considered at once. An X-ray examination is essential; if a perforation has occurred, this frequently discloses the presence of air in the mediastinum, the pleural cavity, or in the neck. In doubtful

cases the radiograph should be repeated after the patient has swallowed a small quantity of lipiodol. When the wall of the œsophagus has been perforated, urgent operation under antibiotic cover is indicated. When the cervical œsophagus has been ruptured, an incision along the anterior border of the sternomastoid (usually the left), with lateral retraction of the carotid sheath, gives access to the site of perforation (fig. 911), which is closed in two layers. If the mid or lower œsophagus is involved, thoracotomy must be performed. In non-malignant cases timely suture is usually followed by recovery.

When the symptoms are delayed, and radiography is negative, the perforation is in all probability a very small one. In this case massive antibiotic treatment with parenteral feeding is usually successful.

FIG. 911.—Exposure of a tear in the cervical œsophagus. In this case there was surgical emphysema on the right side of the neck.



Burns and scalds of the œsophagus from swallowing corrosive fluid, if not rapidly fatal, are often the precursor of a dense stricture. The surgeon's aim should

be to minimise the contracture of the inevitable scar and this is best achieved by the use of hydrocortisone and antibiotics. As soon as the patient's general condition permits, gastrostomy is performed. About a week later it is advisable to ascertain the extent of the damage by œsophagoscopy performed with great gentleness.

It is wise to prohibit bouginage for at least three weeks. More than 70 per cent. are controlled by dilatation. The remainder should have resection of the stricture and colon replacement if necessary. The enormous improvement in the outcome of burns and scalds of the œsophagus is due chiefly to the introduction of antibiotics, cortisone, and to the development of œsophageal surgery.

SPONTANEOUS RUPTURE OF THE ŒSOPHAGUS

Ætiology.—Instead of the cricopharyngeus relaxing, as is usual during vomiting, it contracts. The pharyngeal and pyloric sphincters being closed, the pressure within the œsophagus rises so steeply that the organ bursts at its weakest point. The condition is similar to the linear tears of the cardia in the Mallory-Weiss syndrome (p. 754).

Pathology.—The necropsy findings¹ are remarkably constant. There is a longitudinal tear 1 to 4 cm. in length in the posterior wall of the extreme lower end of the œsophagus, nearly always on the left side. Following perforation, the mediastinum becomes filled with air and gastric contents. Usually about six hours later the pleural membrane gives way, and air and gastric contents gush into the pleural cavity.

Clinical features are also remarkably constant. Following a meal, vomiting occurs. During vomiting, agonising pain is experienced in the thorax, followed by dyspnoea. Board-like rigidity similar to that accompanying perforation of a gastric or duodenal ulcer is often present. Soon surgical emphysema appears in the supra-sternal notch; it spreads up and around the neck. Crepitus is often detected here within one hour after rupture. Hyper-resonance and absence of breath-sounds over one lung indicate a pneumothorax. The pain is so intense that even morphine fails to relieve it.

Radiography.—In very early cases no abnormality is demonstrated; after a few hours mediastinal emphysema is seen; in later cases a hydro-pneumothorax is present.

Paracentesis Thoracis.—Several hours after the perforation, when the extravasated fluid in the mediastinum has ruptured into the pleural cavity, if a hollow needle is inserted into the chest, air and liquid vomitus, acid in reaction, are withdrawn.

Treatment.—Recovery has followed immediate thoracotomy, opening widely the mediastinum, suture of the perforation, and closure of the thorax with drainage. Supportive fluid therapy must be given before, during, and after the operation.

ŒSOPHAGEAL DIVERTICULUM

Pulsion diverticulum is rare, and occurs where a blood vessel enters the œsophagus. They are small and the majority are associated with neuro-muscular inco-ordination which is the cause of occasional dysphagia.

ŒSOPHAGEAL VARICES

The lower end of the œsophagus is one of the principal regions where the portal and systemic venous systems anastomose. Dilatation of these anastomotic channels occurs in portal hypertension (p. 808) and is a cause of hæmatemesis. The treatment of œsophageal varices is considered on p. 810.

PARALYSIS

The passage of food along the gullet is dependent entirely upon involuntary muscular peristalsis. When the neuromuscular mechanism of deglutition is paralysed, as occurs occasionally, notably as a complication of tetanus or diphtheria, ingested material is regurgitated. In established cases the difficulty must be overcome by feeding through a stomach tube.

¹ Spontaneous rupture of the œsophagus has been found at necropsy in cases of severe head injury and other cerebral lesions, doubtless due to vomiting and retching.

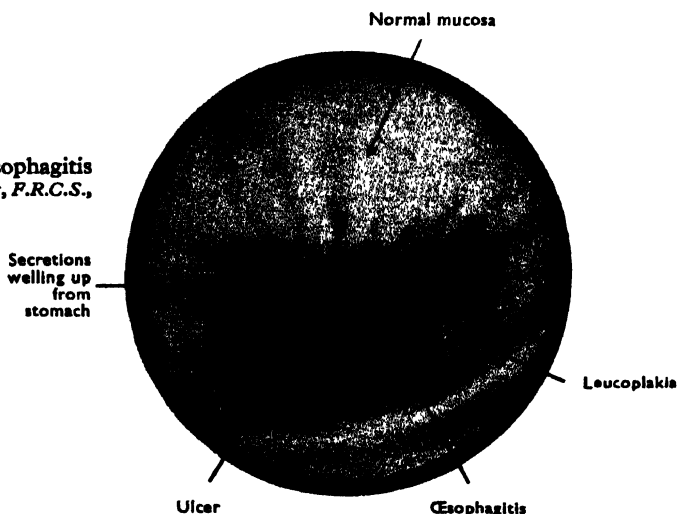
ŒSOPHAGITIS

This may be *acute*—following burns or scalds, infective (spreading from the pharynx), or peptic, sometimes from the trauma of an indwelling stomach tube; or *chronic*—the most important cause is reflux, either of acid or alkaline juices; the latter following œsophago-duodenostomy or œsophago-jejunosotomy without a Roux-en-y loop.

Ætio-Pathology.—The changes are maximal at the lower end of the œsophagus. Essentially there is loss of epithelium with replacement by bleeding granulation tissue. Although the symptoms are experienced by day the damage is done by night when the recumbent position allows the peptic juices to flow into the œsophagus. As soon as inflammation of the epithelium occurs, there is vagal hyperactivity so that the longitudinal muscle contracts and draws the cardia more and more up into the thorax and also hyperacidity is increased. Thus a vicious circle of œsophagitis—longitudinal muscle spasm—upward displacement of the cardia—increased regurgitation ensues. It is likely that the reflux œsophagitis so often encountered during pregnancy is due to a temporary sliding hiatus hernia resulting from increased intra-abdominal pressure; after delivery the hernia usually reduces itself, but it sometimes recurs apart from pregnancy later in life.

Œsophagoscopy.—The mucous membrane of the lower end of the œsophagus is velvety, scarlet, and bleeds readily. Additional white areas of desquamating epithelium may also be seen in the inflamed area. Later one

FIG. 912.—Reflux œsophagitis with ulcer. (N. R. Barrett, F.R.C.S., London.)



or more ulcers are present (fig. 912) and progressive cicatrization with narrowing of the terminal part of the œsophagus occurs.

Clinical Features.—Reflux œsophagitis is the commonest affection of the œsophagus, but its manifestations are not always clear-cut.

Pain.—In the majority, pain is the presenting symptom. At first, it is retrosternal and later may radiate between the shoulder blades, down either arm or up the side of the neck to the ear. This referred pain may simulate angina and is an expression of the sympathetic innervation. It tends to occur when the patient lies down to sleep and keeps him awake (in contradistinction to the pain of duodenal ulcer which wakes the patient from a sound

sleep at about 2 a.m.). It is relieved by sitting upright and sometimes by taking alkalis.

Heartburn with regurgitation of bitter fluid is very common.

Dysphagia.—A complaint that food sticks in the region of the lower œsophagus is a fairly frequent symptom of œsophagitis. It occurs long before any stenosis has developed, and is probably an indication of œdema and spasm. Later, dysphagia is constant and becomes progressively worse as the stenosis develops.

Occult blood is present in the stools in a large percentage of cases.

Secondary anaemia is not uncommon. The œsophagitis may only be uncovered when a routine search is made for the cause of such anæmia.

Diagnosis.—A barium swallow X-ray of the œsophagus reveals no characteristic sign until stenosis commences. *Œsophagoscopy* is essential in order to make the diagnosis. The *treatment* is that of the cause, notably that of hiatus hernia (p. 717).

HIATUS HERNIA

TYPES.	Sliding or œsophago-gastric	85%
	Rolling or parœsophageal	10%
	Mixed or transitional	5%

Ninety-eight per cent. of diaphragmatic herniæ (p. 682) occur through the œsophageal hiatus. Sir Astley Cooper was the first to insist that œsophageal hiatus hernia was acquired, and not congenital. While probably this is true in the majority of cases, the fact is that it may occur in early infancy, and rapidly proceed to stricture and œsophageal shortening, a condition erroneously described as 'congenital short œsophagus'.

Sliding Hiatus Hernia

Anatomy of Cardiac Sphincter.—There is undoubtedly a functional intrinsic sphincter-like mechanism at the cardia although it cannot be demonstrated anatomically. This is expressed as a zone of high intra-luminal pressure which prevents

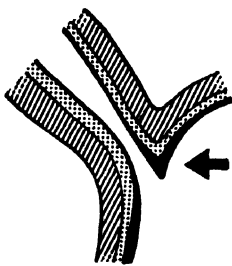


FIG. 913.—The valvular mechanism of the cardia. (After W. S. Lyons.)

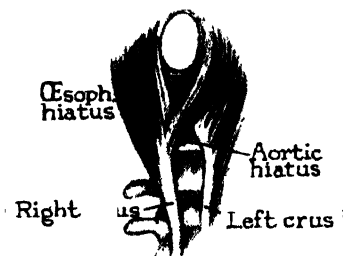


FIG. 914.—The right crus forms the entire musculotendinous diaphragmatic ring around the œsophagus. (After J. L. Madden.)

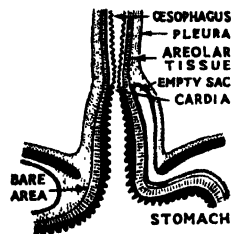


FIG. 915.—Sliding œsophageal hiatus hernia. (After N. R. Barrett, F.R.C.S., London.)

regurgitation from the stomach. This important function is maintained by four factors: 1, the valvular effects of the œsophago-gastric angle (fig. 913); 2, the pinch-lock action of the right crus (fig. 914); 3, the 'rosette-like' folds of the gastric mucous membrane at the cardia; 4, the presence of a length (about 2 inches (5 cm.)) of intra-abdominal œsophagus.

Mechanism of Herniation.—The hernia is small, and usually remains so. The cardiac orifice and a portion of the stomach immediately adjacent pass into the posterior mediastinum, carrying with them a small peritoneal sac applied to the left side

of the stomach. The right side of the hernia is derived from the 'bare area' of the stomach, and consequently is bereft of peritoneum (fig. 915). Branches of the left gastric artery supplying the prolapsed stomach also pass through the hiatus. The sac is always empty, and its fundus lies opposite the junction of the prolapsed stomach (lined by columnar epithelium) and the œsophagus (lined by squamous epithelium). It should be noted that the prolapsed stomach, with its columnar-celled epithelium (black) hangs from the œsophagus proper like a supradiaphragmatic bell. In a less common variety there is massive herniation upwards of the stomach.

The final result is that the phreno-œsophageal ligaments are stretched and the cardio-œsophageal junction is distorted.

Ætiopathology of Herniation.—There are several factors in the development of this hernia:

1. Muscular degeneration of increasing age.
2. Increased intrabdominal pressure as in large ovarian cysts, pregnancy, increasing weight and the wearing of tight corsets.
3. Increase of fatty tissue in the hiatus with decreased elasticity of the crus as occurs in obese women.
4. Once regurgitation is established, œsophageal spasm and later even œsophageal fibrosis will pull more and more stomach into the chest.

Clinical Features.—These are similar to those of reflux œsophagitis (see p. 714). The majority of the patients are over fifty years of age, and women are slightly more often affected than men. A duodenal or gastric ulcer may be present as well. Gall stones, diverticulosis coli and hiatus hernia are not uncommonly associated (Saint's triad, p. 836). An aspiration pneumonitis may develop.



FIG. 916. — Radiograph showing a sliding hiatus hernia. (Graham Airth, Bristol.)

Hiatus Hernia in Infants

Hiatus hernia with œsophagitis is not uncommon in infants. The outstanding clinical feature is effortless vomiting of small amounts, often blood-tinged, starting soon after birth.

Radiography.—In order to demonstrate a sliding hiatus hernia radiologically, technique is important. During the course of a routine barium meal the patient is turned into a semi-prone position on the right side and the table tilted into a 20° Trendelenburg position. In the case of a true hiatus hernia, barium will regurgitate into the hernia without the aid of any additional pressure (fig. 916). If there is no regurgitation from the hernia into the œsophagus, there are usually no symptoms and no treatment is necessary.

Œsophagoscopy reveals varying degrees of inflammation of the lower end of the œsophagus (p. 709). The most valid sign of a hiatus hernia is reflux of gastric juice through the cardia, best seen during œsophagoscopy under local anæsthesia. Furthermore the cardia will open on inspiration, whereas it normally closes and descends.

Differential Diagnosis.—Now that the prone head down position is routine in all barium meals, hiatus hernia and reflux are a not infrequent finding. Above the age of 60 years about 50 per cent. of all patients coming for a barium meal examination have a demonstrable hiatus hernia at some time or other (Airth). One must therefore be vigilant lest cholelithiasis.

peptic ulcer, or appendicular dyspepsia be overlooked in a patient with an incidental hiatus hernia.

Factors which influence Treatment

In all cases medical treatment should be tried and faithfully followed. Many cases do not need surgery, and it is unwise to advise operation in the absence of definite indications. Operation undertaken in patients who are obese or who have chronic bronchitis often gives unsatisfactory results.

Medical Treatment.—The principles to be followed are:

1. The patient must sleep in a semi-recumbent position propped up with pillows and with the lower end of the bed raised on a chair.

Five out of six patients benefit from lying on the left side at night.

2. He must avoid heavy work, lifting weights and excessive bending. Surgery may be essential in these cases.

3. Six small, non-bulky meals should be taken daily, instead of three; food should be masticated thoroughly, and eaten slowly. The maintenance of the upright position after meals is sometimes of benefit.

4. The sucking of antacids such as Nulacin may control symptoms.

5. If the patient is obese, reduction of weight is very important.

6. Correction of anæmia.

Especially when the patient is old or follows a sedentary occupation, these simple measures may bring about and maintain such a remarkable improvement that the question of operation can be postponed indefinitely. In other circumstances operation should be advised.

Medical Treatment in Infancy.—The first injunction is to nurse the baby in an upright position, and the second is to thicken the feeds, giving semi-solid diet as soon as possible. The most practical method of maintaining the sitting position is by an almost legless chair made of plaster of Paris, which can be used both in the cot and the perambulator. Some children lose all symptoms when they start to walk; some of the remainder will require hiatus herniorrhaphy.

Indications for Surgery

1. Severe symptoms which cannot be controlled medically or which interfere with patients' occupation.

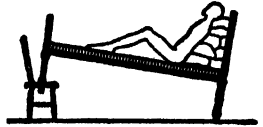
2. Complications such as stenosis, hæmorrhage and œsophagitis.

3. "Causative" lesions such as gallstones, ovarian cysts and pyloric ulcers should be dealt with.

Operative Treatment.—There are two approaches in common use—thoracic and abdominal. The guiding principle of surgical repair is to ensure a length of at least 2 inches of intra-abdominal œsophagus and to prevent this segment from returning to the chest. The positive intra-abdominal pressure of inspiration closes this segment of gullet by compression against the right crus thus preventing the reflux of gastric juice into the thoracic œsophagus (Keen).

The choice between the abdominal and thoracic operations is vexed. There is little doubt that the production and maintenance of a segment of intra-abdominal œsophagus can often only be achieved after mobilisation of the œsophagus as far as the aortic arch. Peri-œsophagitis will frequently prevent adequate mobilisation from the abdominal approach. Early cases uncomplicated by œsophagitis however may be approached from below and this approach certainly facilitates surgery on the gall bladder and duodenum if necessary. Furthermore it avoids the intercostal neuralgia which may follow the thoracic approach.

Thoracic Approach.—With the patient lying on the right side, an incision is made parallel to the left 8th rib and the chest is entered by sub-periosteal stripping of the upper border of this rib. The pleura over the hiatus and lower œsophagus is incised and the œsophagus mobilised. In the presence of peri-œsophagitis and fibrous



œsophageal shortening, it may be necessary to mobilise it as high as the aortic arch. If this is not done it may be difficult to reduce the hernia and where the hernia is repaired under the condition of a taut fibrous œsophagus, recurrence is certain. A rubber catheter is placed around the œsophagus to act as a retractor and helps to pull the herniated stomach into the chest. The crural muscles are cleaned and freed from the cardia and the lesser sac of peritoneum is opened. During this procedure, the phreno-œsophageal ligament is divided. This 'ligament' is more frequently seen in diagrams than at operation and although it exists as an anatomical entity, too much reliance must not be placed on its use during the repair. The stomach is freed from the hiatus, and the large fatty bursa at the cardia should be cleaned from the œsophagus and stomach. There are 3 essential steps to the mechanism of repair (Belsey). Step 1. Three or four linen sutures are placed between the two limbs of the right crus behind the œsophagus (fig. 917(1)) but are not tied at this stage. Step 2. The gastro-œsophageal angle is then created by three linen sutures placed at the same level transversely between the fundus of the stomach and the œsophagus in such a way (fig. 917(2)) that the fundus is wrapped around the lower inch (2.5 cm.) of the left side of the œsophagus. Step 3. A further three or four linen sutures are placed also transversely (fig. 917(3)), picking up in turn the underside of the left leaf

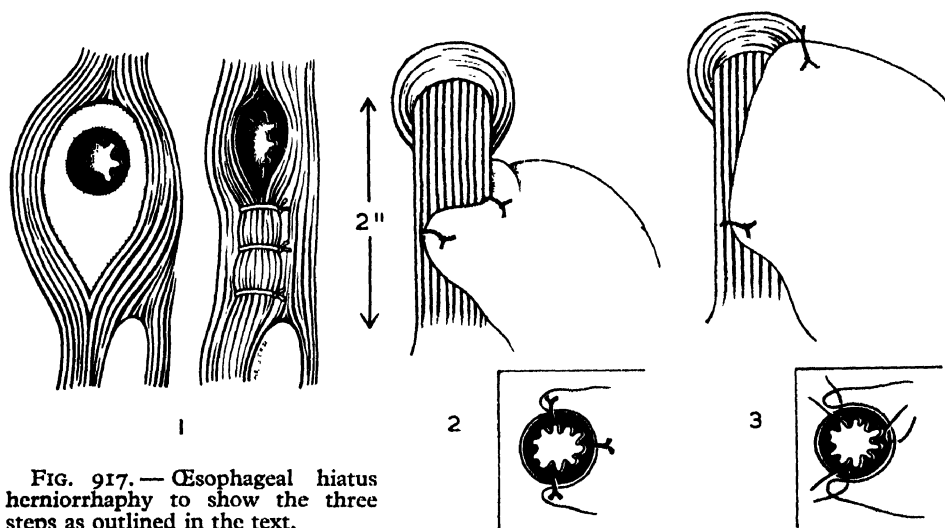


FIG. 917.—Œsophageal hiatus herniorrhaphy to show the three steps as outlined in the text.

of the diaphragm $1\frac{1}{2}$ in. (3.8 cm.) from the hiatus, the fundus of the stomach and the œsophagus. When these sutures are tied, the reconstituted angle is snugly sutured below the diaphragm ensuring the required 2 in. (5 cm.) segment of intra-abdominal œsophagus. The vagi are not disturbed during this operation.

Belsey prefers to work through the opened hiatus and regards a separate diaphragmatic incision as not only unnecessary but predisposing to further herniation through the resulting scar. After reduction of the hernia, the previously placed crural sutures are tied in such a fashion that the tip of the index finger can be inserted through the hiatus behind the gullet, thus avoiding post operative dysphagia. The chest is closed with underwater drainage.

Abdominal Approach.—The left upper paramedian incision makes it possible to mobilise the left lobe of the liver to expose the cardia in the usual way. The stomach is drawn down to reduce the hernia and a transverse incision made at the lowest margin of the diaphragmatic fascia over the œsophagus. The lower end of the œsophagus is thoroughly mobilised with the finger passing up into the mediastinum and the crura completely displayed. Repair is achieved by the three steps defined above. Lesions of the stomach, duodenum, gall bladder and colon may be dealt with at the same time.

Treatment of Complications. Stricture and Penetrating Ulcer.—Here the stenosed portion must be resected and continuity restored by œsophago-gastrostomy or by interposition of jejunum or colon. These patients are often bad surgical risks. Very elderly and frail patients may be fit for regular dilatation only.

Hæmorrhage.—Although chronic œsophagitis can cause a severe iron deficiency anæmia and is an indication for repair, yet severe hæmorrhage is usually due to an accompanying duodenal or gastric ulcer and not from the œsophagus.

Para-œsophageal ('rolling') hernia is a true hernia into which the greater curvature of the stomach (figs. 918 and 919) or, very rarely, the whole stomach (fig. 920) ascends into a preformed sac lying in the mediastinum (fig. 918). Acute dilata-

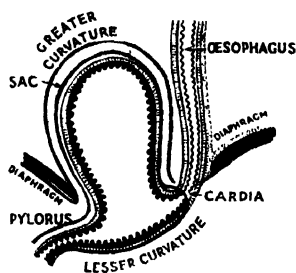


FIG. 918.—Para-œsophageal hernia. (After N. R. Barrett, F.R.C.S., London.)



FIG. 919.—Radiograph showing para-œsophageal hernia. (Dr. Oliver Smith, Birmingham.)



FIG. 920.—Gastric volvulus showing inverted stomach in the thorax. Case of para-œsophageal hernia.

tation or volvulus of the intrathoracic stomach may result in serious symptoms or death and such herniæ are best repaired early.

Clinical Features.—Regurgitant œsophagitis and peri-œsophagitis are not features of this condition and therefore the cardia remains in its normal position (fig. 918). The gastro-œsophageal angle is not disturbed and the competence of the cardia is not impaired. Symptoms therefore rarely appear until the hernia is large (fig. 920), and they are variable: (a) intermittent dysphagia; (b) cardiac symptoms due to pressure on the heart; (c) occasionally bouts of hiccup from irritation of the phrenic nerve. These symptoms are most frequently due to a recurrent gastric volvulus (p. 735).

Operation.—The muscular fibres of the crus are so attenuated by stretching that the operation described for sliding hernia is usually inapplicable.

For a right-sided hernia the thoracic approach is chosen, because the liver prevents a satisfactory abdominal exposure of the hernial site. In the case of the left side (much more common) the abdominal approach is chosen. The upper abdomen having been opened, a stomach tube is passed by the anæsthetist and all gaseous and fluid stomach contents are aspirated before reduction of the hernia is undertaken. Reduction having been effected, the hernial sac is usually excised. A very large adherent sac may have to be detached from the stomach, but the intrathoracic portion is left *in situ*.

The hernial orifice is repaired by overlapping (Harrington). The first layer of sutures is of thread (fig. 921(a)); the second and third layers are of fascia lata (fig. 921(b)).



(a)



(b)

FIG. 921.—(a) Repair of a para-œsophageal hernia. First layer of sutures. (b) The second and third layers of sutures of fascia lata, so placed as to imbricate the pillars of the hernial orifice. (After S. W. Harrington.)

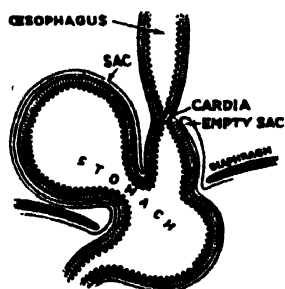


FIG. 922.—Mixed hiatus hernia. (After N. R. Barrett, F.R.C.S., London.)

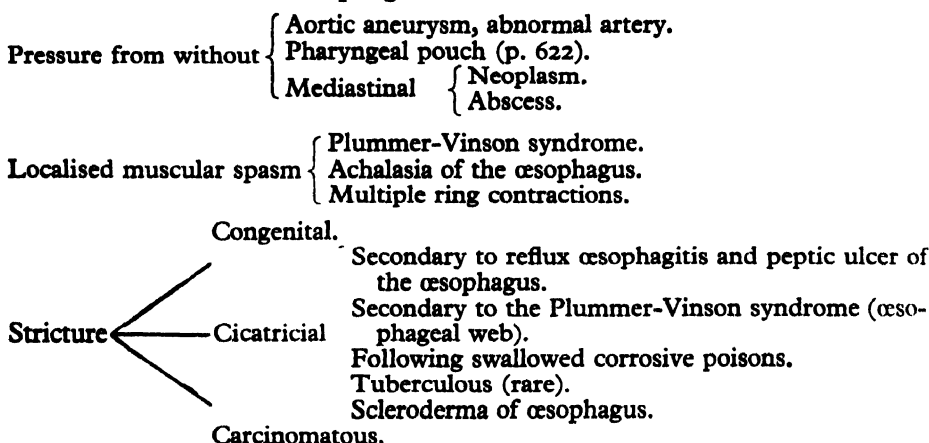
Mixed Hiatus Hernia.—This is a combination of sliding hernia and para-oesophageal hernia (fig. 922).

Clinical Features.—These depend on the degree of angulation of the cardia and the bulk of the para-oesophageal component. If angulation is disturbed and the para-oesophageal component is small, the main symptoms are those of reflux oesophagitis. Alternatively, normal angulation will be associated more with symptoms of a para-oesophageal hernia.

Treatment.—Operative treatment is strongly indicated. For the repair of the hernia some prefer the transthoracic route and others the abdominal route as described on p. 719.

CHRONIC OESOPHAGEAL OBSTRUCTION

The causes of chronic oesophageal obstruction can be classified as follows :



LOCALISED MUSCULAR SPASM

There are two important clinical entities associated with muscular conditions of the oesophagus. One affects the pharyngo-oesophageal junction and is known as the Plummer-Vinson syndrome. The other affects the extreme lower end of the oesophagus and is called cardiospasm, or achalasia.

THE PLUMMER-VINSON SYNDROME

This syndrome was first described by Paterson and Kelly in 1909. It is however commonly called after Plummer and Vinson who described it again in 1919. The patient is nearly always a middle-aged woman who comes complaining of difficulty or inability to swallow. Severe retching spells are wont to occur and classically the patient fears choking. Provided the clinician is aware of the existence of the syndrome, thoughtful examination nearly always provides clues to the diagnosis, but not all the following are necessarily in evidence in a given case.

The tongue is usually devoid of papillæ, smooth and pale, but rarely inflamed or sore.

The lips and corners of the mouth are often cracked, giving the mouth a pursed appearance.

The finger-nails are brittle, and tend to be spoon-shaped (koilonychia) (fig. 923).

The spleen is enlarged in the same ratio as in other iron-deficiency anæmias.

The bone marrow is devoid of stainable iron stores.



FIG. 923.—Spoon-shaped finger-nail.

The Blood.—Hypochromic¹ anæmia is always present, the serum iron levels being particularly low. Usually dysphagia precedes the anæmia, which is due to lack of iron and other requirements of a balanced diet.

Achlorhydria is often present, as is usually the case in iron-deficient anæmia.

Dysphagia is due to spasm of the circular muscle fibres at the extreme upper portion of the œsophagus. This is associated with the formation of webs. The mucous membrane is hyperkeratotic in places, and desquamated in others; it is extremely friable and easily traumatised by the passage of an œsophagoscope. In long-standing cases the orifice looks like a mere pin-hole. This lesion is definitely a pre-carcinomatous condition.

Treatment.—The dysphagia yields readily to dilatation of the stricture through an œsophagoscope. As always, gentleness must be used; even so, in cases of some standing, bleeding will occur. The administration of tab. ferrous sulphate, 5 grains (0.3 G.) t.d.s., together with vitamins, is indicated. Some patients require intramuscular or intravenous iron. Sometimes blood transfusion is necessary. Hyperalimentation with a liquid diet through a gastric aspiration tube helps to bring about regeneration of the desquamated epithelium of the mucous membrane of the œsophagus. Once the anæmia is under control and the patient can swallow an adequate diet, rapid improvement occurs and usually is maintained.

ACHALASIA OF THE ŒSOPHAGUS (syn. 'CARDIOSPASM')

Ætiology.—Achalasia is a disease of the œsophagus, the exact cause of which still remains unsolved. It may be congenital.

It consists essentially of a failure of integration of the parasympathetic impulses so that œsophageal peristalsis is disorganised, and there is failure of relaxation of the cardia. These result in a *functional* obstruction. An epidemic variety has been described in Breslau and was associated with Vitamin B₁ deficiency on the one hand and trypanosomiasis on the other (Chagas' disease). Stress and emotional upsets are also associated with achalasia.

The œsophagus consists of a dilated tortuous sac above and a narrow neck below. The elements of Auerbach's plexus are defective though ganglia are present (cf. Hirschsprung's disease (p. 893) where they are completely absent). There is marked stasis and a retention œsophagitis due to the presence of a foul smelling fluid. Like stasis anywhere, it predisposes to diverticula and it is pre-cancerous.

Clinical Features.—Usually achalasia of the œsophagus occurs in women about forty years of age, but it can occur at any time of adult life and in both sexes. The history is one of progressive dysphagia, there are several special features.

The onset is insidious and, more often than not, the patient seeks relief only after the symptoms have been present for many years. Although the patient says she vomits, on closer interrogation it becomes apparent that there is regurgitation of food, often several hours after the meal. In advanced cases mucus and froth are brought up in considerable quantities. There may be retrosternal discomfort, fœtid flatulence and aspiration pneumonitis. A toxic 'rheumatoid' arthritis may occur.

As a result of the obstruction the patient fails to obtain sufficient nourishment, and consequently remains in a state of continual ill-health, rendering normal activities impossible.



FIG. 924. — Achalasia of the œsophagus. Necropsy specimen showing the typical flask-shaped dilatation.

The *radiographic findings* are most important. Characteristic features are:

1. A smooth pencil-shaped narrowing of the lower segment (fig. 925).
2. Dilatation and later tortuosity of the lower œsophagus.



FIG. 925. — Typical X-ray appearance after a barium meal in a case of achalasia. (Graham Airth, Bristol.)

3. Lack of a gas bubble in the stomach (fig. 925).
4. Incoordinated peristalsis which is hypersensitive to acetyl-choline (when Carbachol 0.25 mg. is injected intramuscularly abnormal peristaltic contractions occur).

Œsophagoscopy.—Once the instrument has passed the cricoid cartilage it appears to enter a gaping cavity partially filled with dirty water, which laps to and fro with respiratory movement. When the fluid has been aspirated the cardiac orifice is located with difficulty, owing to its contracted state.

Treatment.—The essential is the disruption of the constricting fibres of the cardia either from within by Plummer's bag or from without by Heller's operation.

Plummer's Hydrostatic Bag.—A silk thread with a shot on the end is swallowed, and when the shot has entered the stomach, suitable bougies are passed by the 'rail-road' method. Once the narrow



FIG. 926. — Plummer's hydrostatic bag *in situ*.

neck has been dilated sufficiently, the hydrostatic bag (fig. 926) is inserted. Plummer's bag often succeeds where dilatation fails, because the hydrostatic bag can be distended to a transverse diameter of 5 cm. This ruptures the circular muscle fibres of 'the neck'.

Operative treatment becomes necessary in at least 20 per cent. of cases.

Heller's Operation (Œsophagocardiomyotomy).—Aspiration of the dilated œsophagus is commenced twenty-four hours before operation during which time the patient is on a water diet. The approach may be thoracic or abdominal but the essential procedure is the same. In the abdominal operation a long high left paramedian incision is made. The left lobe of the liver is retracted to the right and the triangular ligament, thus rendered taut, is severed. The stomach is retracted downwards so as to display the abdominal portion of the œsophagus, the peritoneum overlying which is incised, permitting the lower end of the œsophagus to be liberated circumferentially. A soft rubber catheter is passed around the cardia to facilitate retraction. When the lower end of the œsophagus has been mobilised sufficiently, a longitudinal incision is made extending 2 inches (5 cm.) proximal and 2 inches distal to the constricted portion. The incision is deepened to the mucosa throughout the length of the incision (fig. 927). After its completion, the mucous membrane bulges, as occurs in Ramstedt's operation (p. 732). The gauze sling is removed and the abdomen is closed. The advantage of the thoracic operation is that the myotomy can be carried up much higher on the œsophagus.

In 80 per cent. of cases the results of Heller's



FIG. 927.—Heller's operation, showing the length and position of the incision dividing the muscular coats. (After P. Thorek, Chicago U.S.A.)

operation are most satisfactory; in the remainder, symptoms due to reflux œsophagitis mar the benefits conferred by the operation.

ABNORMAL RING CONTRACTIONS OF THE THORACIC ŒSOPHAGUS

Multiple segmental ring-like spasms of the smooth muscle leading to alternate contractions and dilatations (ripple œsophagus) sometimes occur, usually in middle life. They are responsible for mild dysphagia and regurgitation and are discovered on radiological examination. The condition, which is sometimes familial, is presumably the result of autonomic dyskinesia. Antispasmodics can be tried in early cases. Later cases need regular dilatation with bougies.

BENIGN STRICTURE

Compared with obstruction due to carcinoma, simple stricture is rare in this country. As set out in the table on p. 720, the causes of benign stricture are numerous. When consequent upon swallowing corrosive poisons, the strictures are usually multiple, the densest being at the level of the crossing of the left bronchus. The sole symptom is increasing dysphagia, and the diagnosis is established by a barium swallow X-ray and by œsophagoscopy.

Treatment.—In acute corrosion the use of cortisone has been found valuable. In the established condition treatment consists in dilatation with bougies (fig. 928). In the first instance this should always be carried out under vision; in most cases it is advisable to continue to dilate the stricture at regular intervals under the vision afford-

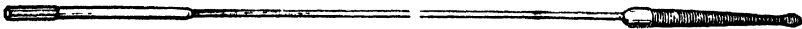


FIG. 928.—Chevalier Jackson's carrot-shaped œsophageal bougie.

ed by an œsophagoscope. When the stricture has a very small lumen a special guide is passed, to the end of which a larger bougie is attached by a screw. By these means nearly all simple strictures can be dilated sufficiently to permit bougies to be passed in the usual manner, but treatment must be continued at regular intervals suited to the individual case for the remainder of the patient's life.

In cases where a bougie cannot be passed from above or threaded along a swallowed string weighted by a small shot, retrograde bouginage can be attempted through a gastrotomy. If this measure is successful, bougies can be passed afterwards in the usual way. In extreme cases, where dilatation of a dense stricture is impossible, colon replacement of the strictured segment is advisable (p. 726).

BENIGN TUMOURS

Simple tumours of the œsophagus are exceedingly rare. Usually they are discovered as a space-occupying lesion in a radiograph following a barium swallow, ordered on account of dysphagia. The diagnosis is confirmed by œsophagoscopy with, perhaps, biopsy. Papilloma of the œsophagus is usually solitary, and occurs in the upper third of the organ. Diathermy excision through an œsophagoscope is a satisfactory method of treatment. A polyp with a narrow stalk can be removed with a snare. Occasionally a leiomyoma or a submucous lipoma causing symptoms of dysphagia has been removed successfully by the transthoracic approach. The most difficult benign neoplasm of the œsophagus to treat is a cavernous hæmangioma which, as might be expected, usually gives rise to hæmatemesis. Injection of a sclerosing agent is sometimes successful; in others, partial œsophagectomy is required.

CARCINOMA OF THE ŒSOPHAGUS

Carcinoma of the œsophagus presents a disturbing problem. Most cases are diagnosed only after œsophageal obstruction has developed and the tumour has progressed beyond the anatomical limits of the organ and has involved the regional, if not distant, lymph nodes. In spite of modern methods of treatment, only 2 or 3 per cent. of patients with this disease survive for five years after the diagnosis has been made.

The œsophagus is a common site for a carcinoma in men but is exceeded in incidence, by the bronchus, stomach, colon, prostate and pancreas. In

Japan, carcinoma of the œsophagus occurs even more frequently, and at a younger age, than in Western races. It may be primary or secondary. The latter is becoming more common with the increased incidence of bronchial carcinoma.

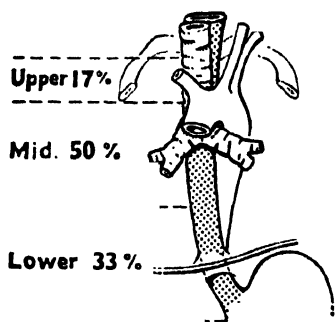


FIG. 929.—The relative frequency of squamous-celled carcinoma in various portions of the œsophagus.

2. An epitheliomatous ulcer with everted edges.
3. A fungating cauliflower-like friable mass.

Spread: (a) *Direct*.—This is the main method of spread and the most important to the surgeon. It is both transverse and longitudinal in direction and erodes the muscular wall to invade the important structures of the neck and posterior mediastinum, as well as the lungs and trachea. It may perforate and lead to mediastinitis; rarely it causes massive fatal bleeding from the aorta.

(b) *Lymphatic*.—Submucosal lymphatic permeation may lead to satellite nodules away from the main tumour. Similarly, embolic spread to surrounding lymph nodes occurs. From the cervical œsophagus the spread is to the lymph nodes of the supraclavicular triangles. From the thoracic œsophagus metastases pass to the para-œsophageal and tracheo-bronchial lymph nodes, with downward extension to the subdiaphragmatic nodes. In the case of the abdominal œsophagus, spread is to the lymph nodes along the lesser curvature of the stomach, and thence to those around the celiac axis. In the main, lymphatic metastases occur in a *downward* direction.

(c) *By the blood-stream* is exceptional; occasionally metastases occur in the liver or the lungs.

Clinical Features.—Men between forty-five and seventy are the usual victims, their average age being sixty-three. About 28 per cent. of the patients are women, and in them the neoplasm is situated most frequently in the upper third of the œsophagus; their average age is in the late fifties.

The leading symptom—indeed, often the only symptom—of this fell disease is dysphagia. Sometimes, owing to sloughing of a portion of the growth, for a time swallowing becomes easier, but as a rule the difficulty is steadily progressive. Only 40 per cent. of patients report within three months; too often the patient procrastinates seeking advice until he can no longer swallow milk pudding. By this time the growth must have encircled at least three-quarters of the lumen of the œsophagus and, as might be expected, loss of weight is obvious. Regurgitation (œsophageal pseudo-

Pathology.—The lesion may be squamous celled or an adenocarcinoma. The latter occurs essentially at the lower end from the lower 3 cm. lined by columnar epithelium (fig. 902). or may be secondary to carcinoma of the stomach. The sites of predilection for the squamous-celled growth are the three areas of constriction (fig. 903). The incidence is shown in fig. 929.

Macroscopically three types can be recognised:

1. An annular stenosing lesion usually found at the cardia.

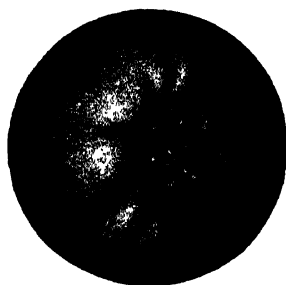


FIG. 930.—Carcinoma of the œsophagus as seen through an œsophagoscope. (A. Lawrence Abel, F.R.C.S., London.)

vomiting) is a fairly common symptom. The regurgitated material is alkaline, mixed with saliva, and possibly streaked with blood. In growths situated in the lower part of the œsophagus, anorexia is sometimes a feature. Pain, if it occurs, is usually a late manifestation, but it is not in itself a contraindication to an exploratory operation.

Radiography after barium emulsion has been swallowed should be carried out in every case of dysphagia. By this investigation the presence of a carcinoma of the œsophagus (fig. 931), or, more usually, the stenosis produced by the neoplasm, is displayed (fig. 932). Nevertheless, complete reliance must not be placed on the radiological findings, particularly if the examination is negative.

Œsophagoscopy is usually diagnostic. A piece of the tumour must always be removed for histology.

Bronchoscopy should be undertaken when there is a cough with expectoration of large quantities of purulent sputum which suggests involvement of the bronchial tree, and possibly a fistula.

Exfoliative Cytology.—Lavage of the œsophagus and examination of the fluid for malignant cells has led to the discovery of an early carcinoma when radiology and œsophagoscopy were negative.

Treatment.—Dysphagia is a most distressing symptom and somehow it must be relieved. Gastrostomy is a most unsatisfactory procedure and is mentioned only to be condemned. Treatment is by operative resection, radiotherapy or palliation.

Pre-operative Treatment.—Hourly feeds, as much as the patient can take, of fortified milk (2 eggs, 2 ounces (60 G.) sugar, 1 teaspoonful salt, 2 ounces dried milk, and 1 ounce of butter to 2 pints (1·14 l.) of milk) are given. Breathing exercises and, when necessary, postural drainage are carried out. The patient's teeth, if present, are scrutinised as hygiene of the mouth is most important. Suitable preparations of iron and vitamins are prescribed. Antibiotic therapy is commenced three days before the operation.

Operation.—It is only after the region of the growth has been displayed and the extent of its fixity to surrounding structures ascertained that it is possible to tell if partial œsophagectomy is practicable. As a result of exploration, about 50 per cent. of growths will be found to be irremovable, in which case a short-circuiting operation is performed. If the growth is operable, the methods of procedure are as follows:

(a) **Growth at the Cardiac Orifice.**—Through a thoraco-abdominal approach (fig. 933) a block dissection of the stomach, together with the spleen, omenta, and tail of the pancreas, is undertaken. Restoration of the continuity is by œsophago-jejunostomy, employing a Roux-en-Y anastomosis.

(b) **Growth situated in the Lower Half of the Thoracic Œsophagus.**—Again a thoraco-abdominal incision is made, but at a slightly higher level. Because of the danger of hæmorrhage from branches of the thoracic aorta, Allison frees the aorta as a first step by dividing between ligatures the upper six left and right intercostal arteries. The thoracic aorta can then be lifted up with tapes while the dissection



FIG. 931.—Early carcinoma of the œsophagus. (The late Dr. G. R. Mather Cordner, London.)



FIG. 932.—Advanced carcinoma of the œsophagus, D.8 level. (Dr. Oliver Smith, Birmingham.)

of the œsophagus from its bed is carried out. Once the tumour-bearing area of the œsophagus has been mobilised, the phrenic nerve is crushed and the diaphragm is incised from the œsophageal hiatus to the lateral thoracic wall. Resection and restoration of the continuity of the alimentary canal are effected by mobilising the stomach,

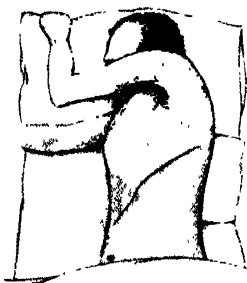


FIG. 933. — Usually the eighth rib is resected subperiosteally. If more room is required, the posterior ends of the seventh, sixth, and fifth ribs are sectioned.

division of the termination of the œsophagus with closure and invagination of its distal end, followed by attaching the fundus of the stomach to the œsophagus just above the proposed line of section. Resection having been completed, the proximal end of the œsophagus is joined to the fundus of the stomach, a circular area of about 3 cm. in diameter being removed from the stomach for that purpose (fig. 934). The diaphragm is repaired around the stomach. The lung is re-expanded, and the

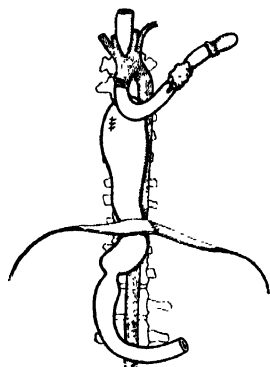


FIG. 934. — Resection of the œsophagus for a growth in its middle third, utilising the mobilised stomach for restoring the continuity of the alimentary tract (semi-diagrammatic).

incision in the thoracic wall closed in layers with under-water seal drainage.

(c) **Growths in the Region of the Middle Third.**—

The œsophago-gastric anastomosis after resection of these growths often comes to lie behind the arch of the aorta. A right-sided approach is advisable. An abdominal incision is made first, and the greater and lesser curvatures of the stomach fully mobilised from cardia to pylorus, preserving the right gastric and gastro-epiploic vessels. The cardia is freed from its hiatus at this stage and a pyloroplasty is performed. The patient is then turned on to his left side, and the œsophagus approached via a thoracotomy at the level of the fifth rib. The vena-azygos is the only structure requiring division. The œsophagus is then fully mobilised after which the stomach is drawn up into the chest. The œsophageal resection extends from 6 cm. above the growth right down to the cardia to include a cuff of gastric wall. This gastric defect is closed in two layers, and the highest point of the fundus anastomosed to the cut end of the œsophagus. Debilitated patients often benefit from a two-stage procedure several days apart.

(d) **Growth lies in the Thorax above the Arch of the Aorta.** See Post-Cricoid carcinoma (p. 630).

* * *

Other Methods of bridging the Defect.—The great disadvantage of utilising the stomach as a substitute for the œsophagus is reflux œsophagitis above the anastomotic line. For this reason other methods are being used. The best of these is colon replacement. An isolated segment of the transverse or the right colon, with its blood supply intact, is brought up to connect the stump of the œsophagus to the stomach. An intrathoracic polythene prosthesis has been used but, in general, it has not been found to be satisfactory.

Radiotherapy.—In carcinoma of the upper and mid-œsophagus radiation frequently restores the ability to swallow, although this benefit lasts only a few months. Recent results with penetrating radiation are more encouraging and an increasing number of long-term survivals are being obtained.

Growths of the Upper Third.—The magnitude and severity of extirpation of the œsophagus in this region, and the radio-sensitivity of most of the neoplasms, render radiotherapy the method of choice.

Growths of the Mid Third.—The ⁶⁰cobalt teletherapy unit emits gamma radiation equivalent to the most powerful super-voltage X-ray machine, and a rotational

device has enabled a more uniform dose to be delivered to the growth. In a number of instances, as a result of this treatment, the tumour disappears; however, in spite of improvement in swallowing, the majority of patients soon die of metastases.

Growths of the Lower Third.—Radiotherapy should not be employed. Frequently the neoplasm is columnar-celled and radio-resistant. Irradiation of the liver cannot be avoided, and this makes the treatment distinctly dangerous.

The results of radiotherapy plus operation appear to be little better than either alone.

PALLIATIVE PROCEDURES

When Thoracotomy reveals that the Growth is Irremovable.—If the neoplasm is situated at the cardia, the œsophagus can be divided above the growth, the lower end closed, and œsophago-jejunostomy-en-Y (Roux) performed above the obstruction. When the neoplasm is higher in the thoracic œsophagus, after similar exclusion, the previously mobilised stomach can be anastomosed to the upper end of the œsophagus.

When Inoperability is decided upon at a Clinical Examination.—Inoperability is so frequent and the results of heroic surgery so disappointing that a simple palliative procedure assumes an added importance. The introduction of a tube not only allows the patient to take sufficient nourishment by mouth, but relieves him of his one distressing symptom: dysphagia. It also protects him from recurrent aspiration pneumonitis. Neither of these is palliated by a gastrostomy.



FIG. 935.—Celestin tube.

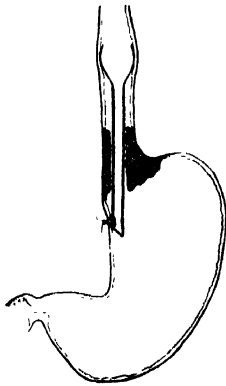


FIG. 936.—Method of fixation of tube.

The Souttar tube, made of German silver and pushed blindly into position after dilatation, has now been replaced by safer tubes that are *pulled* into position. The Celestin tube based on physiological considerations consists of a 25-cm. shaft carrying an oval barrel-shaped funnel about the size of a bolus. It is pulled into position by a detachable pilot-bougie (fig. 935). The latter is navigated through the growth under direct vision, no dilatation being required. The tip of the bougie is recovered in the stomach via high gastrotomy. The endo-œsophageal tube is then plugged on securely to the bougie and traction applied steadily to the latter so as to draw the tube through the tumour. The tube must be finally left in a comfortable vertical position, even if this means that the funnel lies several inches above the tumour (fig. 936); otherwise ulceration may result. It is then tethered to the lesser curve and any excess cut off. After the operation the patient may be allowed a fairly free diet provided it is chewed very thoroughly, and frequently washed down with aerated water. The tube may also be used in patients receiving high-voltage irradiation, or as a pre-operative measure.

Terminal Complications.—Unresected, the growth causes death in one of the following ways:

1. Progressive cachexia and dehydration.
2. Pneumonia from perforation into some part of the bronchial tree.
3. Mediastinitis from perforation into the posterior mediastinum.
4. Erosion of the aorta (rare).

CHAPTER 32

THE STOMACH AND DUODENUM

Surgical Anatomy and Physiology.—John Hunter described the stomach as 'a gland with a cavity', and it is conventional to subdivide the organ into various parts. The mucous membrane is composed of some 35,000,000 branched tubular secreting glands closely packed and perpendicular to the surface. The oxyntic and pepsin-secreting cells are confined to the body of the organ. The oxyntic cells secrete HCl to a pH of 0.9. It is fundamental to appreciate that the amount of acid secreted is determined by the oxyntic cell population. Normal parietal cell population is one billion and in duodenal ulcer cases 1.75 billion. The essential protective substance against HCl is mucus. The secretion of the antrum is, curiously enough, weakly alkaline (see antrectomy, p. 749).

Secretion of Gastric Juice.—There are two mechanisms of acid production: (1) *Nervous*.—Vagal stimulation is an important factor in the amount of acid-pepsin response. It conditions the oxyntic cells to gastrin and also the release of gastrin from the antrum. (2) *Hormonal*.—The hormone gastrin¹ is probably released from the modified ganglion cells of the antral mucosa and is responsible for the sustained response

following food. It is a polypeptide with a molecular weight of 2000. It is released by mechanical distension of the antrum, by the presence of food and by a high intraluminal pH. Release is inhibited when the pH falls to 1.5. Gastrin causes an increase in gastric acid, in pepsin production, in pancreatic secretion, and in gastrointestinal motility. The inter-relating nervous and hormonal activity is of fundamental importance to the magnitude and duration of response.

Acid production is inhibited (1) by the antrum when the pH of antral contents falls to 1.5 and (2) by the duodenum when the pH of duodenal contents falls to 2.5 or when fat is present—the latter actions are probably due to enterogastrone (fig. 937).

Innervation.—Apart from peristaltic waves, the stomach possesses muscular tone, controlled by the intrinsic nerve plexuses in the walls of the viscus. The vagus nerves are the motor nerves of the stomach. Fig. 938 shows the

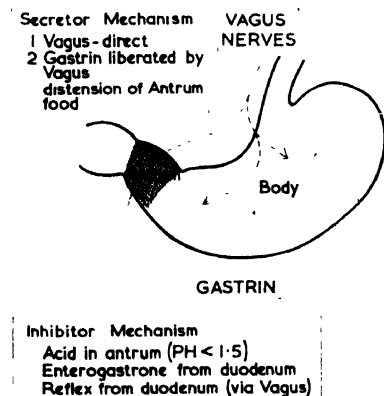


FIG. 937.—To show mechanism of gastric acid secretion.

general disposition of the nerves, and their branches. Familiarity with the common distribution and main variations of the nerves is important owing to the swing from total to selective or specific gastric vagotomy.

The sympathetic innervation of the organ may carry a proportion of pain-transmitting fibres and secretory fibres to the mucous glands.

Function of the Pylorus.—The pylorus itself is basically concerned not so much with gastric emptying as with the prevention of duodenal reflux back into the stomach. Duodenal juice is highly irritating to the gastric mucosa and causes an atrophic gastritis characterised by dysfunction of oxyntic cells and great reduction of mucus output. In early duodenal ulcer the pylorus is completely competent,

¹ Professor R. A. Gregory of Liverpool isolated and identified the nature of gastrin.



FIG. 938.—A, anterior and B, posterior vagus nerve. Showing distribution to stomach, celiac and subhepatic plexuses. (C. V. Ruckley, F.R.C.S., Edinburgh.)

there is no duodenal reflux, no atrophic gastritis, and hyperchlorhydria is present. Later, the duodenal cicatrization may make the pylorus rigid and incompetent, atrophic gastritis may occur, acid production fall, and a secondary gastric ulcer may form. In painful gastric ulcer the pylorus is incompetent; and there is free and extensive regurgitation. The widespread gastric change in the form of atrophic gastritis produced by regurgitation would seem to be a precursor of both gastric ulcer and gastric carcinoma. The consequent reduction in oxyntic cell population following reflux, results in hypo- or even achlorhydria.

Atrophy of Gastric Mucosa.—Atrophy may be due to duodenal regurgitation or it may be due to antibodies in the blood specific for the cytoplasm of the parietal cells. Such antibodies are found in pernicious anæmia in which there is also an antibody against intrinsic factor.

Arterial Supply (fig. 968).—(a) The left gastric artery, the smallest branch of the celiac axis, runs towards the cardiac orifice and thence along the lesser curvature, from left to right, to join (b) the right gastric artery, which arises from the hepatic artery and pursues a course from right to left along the lesser curvature. (c) The gastro-duodenal artery is the largest branch of the hepatic artery. It passes behind the first part of the duodenum, where it bifurcates into the superior pancreaticoduodenal artery and (d) the right gastro-epiploic artery. (e) The left gastro-epiploic artery is the largest branch of the splenic artery. (f) Vasa brevia (short gastric arteries) are five to seven small vessels that spring from the splenic artery towards its termination, and are distributed to the fundus of the stomach. An important feature of the gastric circulation is the large numbers of arterio-venous shunts in the submucosa of the lesser curve. These are opened up by vagotomy.

Veins.—Those corresponding to the right and left gastric arteries terminate in the portal vein. Those corresponding to the left gastro-epiploic artery and vasa brevia join the splenic vein, while the right gastro-epiploic vein empties into the superior mesenteric vein. In the living, the veins of Mayo (fig. 939) are helpful landmarks in distinguishing the pyloric canal from the first part of the duodenum.

Lymphatics are described on p. 769 in connection with malignant neoplasms of the stomach with which they are so intimately concerned.



FIG. 939.—The pre-pyloric vein of Mayo.

HYPERTROPHIC PYLORIC STENOSIS OF INFANTS

Ætiology.—The cause of the condition, which occurs 3 or 4 times in every 1,000 births, is unknown. Several theories have been propounded, the most acceptable being that the condition is due to an achalasia (primary failure of the pylorus to relax). In a small proportion (about 7 per cent.) of cases the abnormality appears in certain families with greater frequency than can be accounted for by coincidence. In these families one parent bears the scar of an operation for hypertrophic stenosis in infancy, and in 50 per cent. of cases it is the mother, which, when the much higher incidence in the male is taken into consideration, is remarkable. In these families a brother is affected similarly in 10 per cent. and a sister in 2 per cent. of cases.

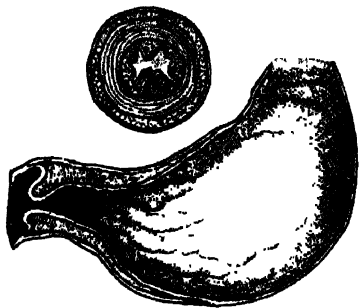


FIG. 940.—Pyloric stenosis of infants. Longitudinal and transverse sections of the stomach to show the enormous muscular hypertrophy. Note the abrupt termination.

Pathology.—The musculature of the pyloric antrum is always hypertrophied. Proceeding distally, the hypertrophy increases, to reach its zenith in the circular fibres of the pylorus. Here the muscle layer is so thick that the mucosa is compressed, and often the lumen admits only a fine probe. As estimated at operation, the size of the pyloric

lump is proportional to the duration of the symptoms. The hypertrophy terminates abruptly, the duodenum being normal.

Clinical Features.—Characteristically first-born male infants are most commonly affected.

The peak incidence for the onset of symptoms is between the third and sixth weeks of life; however, the condition can commence before that time, and as late as the seventh week. The pillars upon which the diagnosis rests are:

Vomiting is the presenting symptom in all cases and within two or three days it becomes forcible and projectile. The presence of bile in the vomit is unusual; exceptionally it contains a few streaks of blood (cf. cardiochalasia, p. 731). Immediately after vomiting the baby is often very hungry.

Visible Peristalsis.—After the child has been fed, peristaltic waves may be seen passing from left to right across the upper abdomen (fig. 941). A good light is essential for this; the baby should be watched throughout a feed until vomiting occurs.

The Presence of a Lump.—The palpation of the hypertrophied pylorus is the most essential step in reaching a diagnosis. The surgeon should sit on the left side of the child and palpate under the liver with a warm left hand. It may be necessary to examine the child more than once. The lump is most easily felt just after vomiting has occurred.

Constipation is usually present, and when a stool is passed it is small and dry, resembling those of a rabbit. It is important to ask the mother about napkins.

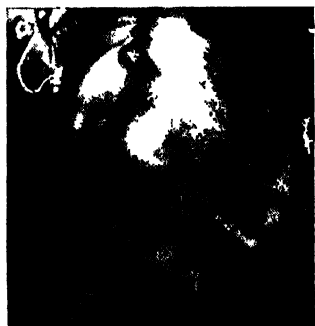


FIG. 941.—Visible peristalsis in hypertrophic pyloric stenosis; it is unusual for the waves to be so obvious. (Alan Kark, F.R.C.S., New York.)

If the child is dehydrated, they are not wet and the case is correspondingly more urgent.

Loss of Weight.—One of the most striking signs of infants suffering from hypertrophic pyloric stenosis is loss of weight. Moreover, it is not long before the infant begins to look emaciated and dehydrated.

Often a change from one type of feeding to another brings about a remission. Consequently a series of changes in diet are sometimes made before the diagnosis is established, by which time the infant's condition may be pitiable.

In premature infants, in whom the condition is not uncommon, the symptomatology is often paradoxical. There is anorexia instead of a voracious appetite; the vomiting is regurgitant rather than projectile, and so frequently is peristalsis normally visible in these attenuated subjects that its significance is liable to be disregarded. None the less, amidst this sea of bewilderment one diagnostic rock remains—a hypertrophied pylorus can be felt through the poorly-developed abdominal wall with comparative ease.

Radiography.—The diagnosis should be made on a careful clinical examination and an X-ray is rarely necessary. To be diagnostic, a barium meal should show persistent narrowing and elongation of the pyloric canal.

Differential Diagnosis.—The most difficult condition from which hypertrophic pyloric stenosis of infants has to be differentiated is *cardiochaliasia*¹ of the *œsophagus*, often due to a hiatus hernia (p. 715): a clinical entity that has been summarised pithily as one of 'persistent vomiting without bile and with no other signs of hypertrophic pyloric stenosis'. The mother sometimes volunteers the information that the infant does not vomit if it is held in the upright position. The presence of blood in the vomit is almost diagnostic of this condition. Here radiology is essential; it shows that the gastric cardia is in a state of continuous relaxation with frequent regurgitation of the barium that has been given.

Other conditions from which hypertrophic pyloric stenosis must be differentiated are (a) intracranial hæmorrhage; (b) duodenal atresia; (c) high intestinal obstruction, e.g. volvulus neonatorum (p. 937). In (b) and (c) bile will be present in the vomitus.

Treatment.—The majority of pædiatricians believe that surgery is the treatment of choice: the condition is immediately and completely corrected and the child is well within twenty-four hours. Babies who are admitted in a dehydrated condition should receive sufficient half-strength dextrose-saline solution, given subcutaneously with hyaluronidase, to restore the fluid balance. This results in a remarkable improvement in the general condition: the sunken eyes and cheeks and depressed fontanelle fill out; the dry skin and mucous membrane become moist, and the output of urine increases.

Medical Treatment

In cases complicated by infection, especially of the mouth, medical treatment is advisable because of the increased risk of post-operative gastro-enteritis, even if operation would otherwise be indicated.

In subacute cases the patient often reaches the age of two months before the symptoms become obvious and if, as a result of medical treatment, the gastric residue decreases in amount, a cure by non-operative means can be anticipated.

Eumydrin (atropine methylnitrate) 1 : 10,000 of water, freshly made, is given half an hour before each feed, beginning with 0.5 to 1 ml. and increasing to 2.5 ml. to control the symptoms. Toxic symptoms (erythema and hyperpyrexia), though less

¹ Cardiochaliasia is the direct opposite of achalasia of the cardia (cardiospasm).

common than when atropine is employed, do occur with Eumydrin, and are signs that the dose of the drug should be decreased or its use discontinued. Small, frequent feeds of milk (if possible the mother's milk), diluted with 5 per cent. dextrose, are given.

Eumydrin must never be given until a firm diagnosis has been made.

Surgical Treatment.—After correction of dehydration, operation is performed without delay. The recovery rate approaches 100 per cent. in early cases.

Ramstedt's Operation.—*Preliminary Preparation.*—The stomach is washed out with saline several times; finally, one hour before operation. Immediately before operation gastric aspiration must be performed, and continued throughout the operation. The prevention of chilling is of great importance. To this end the temperature of the operating theatre should be high (80° F., 27° C.) and the infant's body is encased in wool, the upper abdomen alone being accessible.

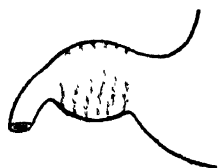


FIG. 942. — The upper part of the hypertrophied pylorus is comparatively bloodless.

Operation.—Local anaesthesia is advisable unless the child is in good condition and the services of an anaesthetist skilled in anaesthesia of infants are available. The abdomen is opened by a grid-iron¹ incision in the upper right quadrant of the abdomen. The hypertrophied pylorus is delivered and rotated so that its superior surface comes into view; thus the least vascular portion (fig. 942) can be selected for the incision.

In order to ascertain the distal limit of the hypertrophied pylorus the surgeon invaginates the duodenum with his index finger. The incision is made through the serosa only from this point along the whole length of the lump (fig. 943). Because the hypertrophied pylorus is the consistency of an unripe pear, splitting of muscle coats can be accomplished with a blunt dissector. On separating the edges with artery forceps, the pyloric mucosa bulges into the cleft which has been made in the muscle (fig. 944). Great care is taken not to penetrate the mucosa,



FIG. 943.—Ramstedt's operation.



FIG. 944.—Showing the hypertrophied muscle divided and the mucous membrane bulging into the incision.

an accident which is liable to occur while dividing the most distal part of the constricting fibres which are in the vicinity of the duodenal 'fornix' (fig. 940). In order to be sure that there is no perforation, some air is squeezed from the stomach into the duodenum. If a perforation has occurred, it is closed by a wisp of omentum held in place by three or four interrupted sutures of fine silk (fig. 945). The free entry of air into the duodenum indicates adequate division of the muscle. Haemostasis must be meticulous. The abdominal incision is repaired. The edges of the loose skin fall together and can be kept in apposition with strips of adhesive plaster. It is important to remove the wool from the infant's body as soon as the operation is over, or hyperthermia may occur.

After-treatment.—To minimise the risk of cross-infection, and consequent gastro-enteritis, the patient must be segregated. Further infusions of dextrose-saline need not be given unless the baby was insufficiently hydrated before operation. A small feed (not more than 3i (4 ml.)) should be given one hour after operation, and after that the feeds can be rapidly increased. The child should be feeding normally in forty-eight hours and if there is no vomiting at this time, it is the practice in many hospitals to send the child home and bring it back for removal of stitches. If the mucosa was accidentally opened at operation, it is wise not to feed the child orally for forty-eight hours.

Complications:

(a) *Post-operative pyrexia* is rather common and is not necessarily of serious consequence. If protracted, tepid sponging is advisable.

(b) *Gastro-enteritis* is rare in this country, and is minimised by segregation and sending the child home as soon as convenient.

(c) *Disruption of the wound* is also rare, and is more liable to occur in emaciated subjects. The use of the grid-iron incision in these cases has reduced the incidence of this complication.

Hypertrophic pyloric stenosis of adults is a definite entity and is often mistaken for carcinoma of the pylorus. When a diagnosis can be made with certainty, Finney's pyloroplasty or a gastro-jejunostomy gives excellent results; when doubt exists, a Billroth I gastrectomy is done. Microscopical examination of the specimen shows fibrosis of the myenteric plexus (contrary to the corresponding condition in the infant).

CONGENITAL OCCLUSION OF THE DUODENUM

There is stenosis, usually complete, across the duodenum (fig. 946). This occurs at the point of fusion of the fore- and mid-gut, and consequently lies in the neighbourhood of the ampulla of Vater. It is frequently accompanied by other congenital



FIG. 946.—Congenital septal duodenal obstruction at the commencement of the third part of the duodenum. The gut above is enormously dilated. (After W. E. Ladd.)

defects. The infant vomits *from birth*, and daily rapidly loses weight. In contradistinction to congenital pyloric stenosis *the vomit contains bile*. Laparotomy should be undertaken without delay. Duodeno-jejunostomy is the best procedure when, as is usually the case, the obstruction is in the second or third part of the duodenum (Ladd). In these cases the stoma is difficult to keep open and it is wise to introduce a fine plastic tube through a gastrostomy opening, through the stomach across the stoma and down into the jejunum for a few inches. This should be retained for several days for feeding purposes.

FOREIGN BODIES IN THE STOMACH

A variety of ingested foreign bodies reach the stomach. Fortunately, for the most part they are opaque to X-rays (fig. 947). Sharply pointed or long objects are best removed promptly by gastrotomy because they have difficulty in negotiating the curves of the duodenum. Rounded, smaller foreign bodies may be left to pass along the natural passages. Suitable doses of normacol form a gelatinous



FIG. 945.—Patching a perforation of the mucous membrane. (After C. E. Welch.)



FIG. 947.—Embedded in normacol, this foreign body was passed naturally in three days.

pabulum, in which the article becomes embedded during its transit along the alimentary tract. X-ray examinations should be undertaken sparingly. The first diagnostic examination should be sufficient in most instances.

Hair-ball of the Stomach (Trichobezoar¹).—An example of this rare condition is usually to be found on the shelves of pathological museums. Trichobezoars occur almost exclusively in females, and in 80 per cent. of cases the patient is below thirty years of age and is often feeble-minded. Trichobezoars can give rise to high intestinal obstruction, gastro-duodenal ulceration leading to hæmatemesis, perforation, peritonitis, or inanition. A trichobezoar shows well on radiography (fig. 948). The treatment is removal by gastrotomy.



FIG. 948.—Radiograph showing hair-ball in the stomach. (Dr. P. M. Wine, Birmingham.)

ACUTE DILATATION OF THE STOMACH

This complication can occur after any operation especially those on the biliary passages or pelvic organs or even after such trauma as a fractured femur or the application of a plaster-of-Paris jacket (p. 408). Fortunately this condition which used to be common has been greatly reduced by the routine use of a nasogastric tube. It can however appear quite suddenly and is still a menace.

Pathology.—In fatal cases the stomach is found to be enormously dilated (fig. 949). The organ is filled with air and dark, watery fluid. Sometimes the dilatation ends at the pylorus; more often it extends into the duodenum,

and in a few instances the dilatation involves the upper part of the jejunum. The gastric mucosa is bespattered with petechial hæmorrhages.

Clinical Features.—Signs of acute dilatation of the stomach may come on two or three hours, or as long as two or three days, after an operation or an accident. The first warning sign is often muffled hiccoughs. Soon the pulse-rate rises to 100 or 120 beats per minute and other signs of shock become manifest. Then the patient vomits; if the content of the stomach is not aspirated forthwith, as it should be, this is followed by enormous effortless vomits of dark, watery fluid which are characteristic of the condition. There is a danger of inhalation of vomit and cardiac arrest.

Prevention.—The use of a post-operative nasogastric tube should prevent this complication entirely.

Treatment.—In a fully established case, prompt action is imperative. The two principles are: (1) To empty the stomach and keep it empty; (2) to restore fluid balance by administering continuous intravenous normal saline, followed (after chloride loss has been satisfied) by dextrose-saline solution. The stomach should be emptied and kept empty by aspiration through an indwelling Ryle's or other small gastric tube passed, when possible, by way of the nose.

By these measures, unless effective treatment has been delayed unduly, the stomach soon regains its tone. If it does not do so, the possibility of hypopotasæmia (p. 86) being a contributory cause should receive immediate attention.



FIG. 949.—Acute dilatation of the stomach seen at necropsy.

¹ Bezoars, or masses of foreign material in the stomach or intestines of animals, are relatively common. In some primitive communities a gastric bezoar from a goat is accredited with magical healing properties.

GASTRIC TETANY

Tetany due to alkalosis may occur where there is long-continued vomiting, gastric aspiration, or excessive ingestion of alkali. The extracellular alkalosis is always accompanied by cellular acidosis, potassium depletion, and a fall in the blood calcium (parathyroid tetany, p. 568). Clinically there are muscle spasms in the extremities, shallow respiration, and cyanosis. The stomach must be kept empty by a tube and the blood electrolytes estimated so that the specific deficiencies may be rapidly corrected. Whilst these are being done, continuous intravenous dextrose saline with calcium gluconate should be given. Even when the chloride deficit is corrected, the alkalosis may persist and this is indicative of the need for potassium (p. 86).

RUPTURE OF THE DUODENUM

Traumatic rupture of the duodenum is a rare accident, usually the result of a blow on the right flank. The rupture may be intra- or extraperitoneal, or both.

Intraperitoneal Rupture.—The tear can usually be sutured.

Extraperitoneal Rupture.—The initial symptoms are often slight, and the condition is overlooked until an abscess forms. When such an abscess is opened, a duodenal fistula results.

Duodenal Fistula.—The most usual causes are as follows :

1. As a complication of gastrectomy (p. 759).
2. An abscess connected with a perforated duodenal ulcer.
3. Traumatic rupture of the duodenum.
4. As a complication of transduodenal choledochotomy, right nephrectomy, or right colectomy.

The fistula discharges bile and pancreatic juice which cause excoriation of the skin. Excessive discharge may quickly lead to dehydration, electrolyte imbalance, and hypoproteinæmia. The management consists essentially of three procedures:

1. Protection of the skin by 1 per cent. HCl in zinc oxide cream.
2. Continuous, adequate, efficient suction drainage of the fistula (fig. 1102) which may be facilitated by gravity—the patient lying prone.
3. Replacement therapy—when the fistula drainage is small, closure may be confidently expected within a few days using simple intravenous therapy. If the discharge is profuse, a severe illness can be prevented by a simple feeding jejunostomy on the left side of the abdomen. The valuable pancreatic and biliary juices may be returned to the jejunum by this route.

Traumatic fistulæ of the first part usually close spontaneously—those of the second and third part are formidable indeed. They present such a grave problem that it is worth considering a duodeno-jejunal anastomosis overlying the defect if this cannot be closed easily at the primary operation.

VOLVULUS OF THE STOMACH

Rotation of the stomach usually occurs around the axis made by its two fixed points, i.e. the cardia and the pylorus (fig. 950). Although the rotation can occur in a horizon-

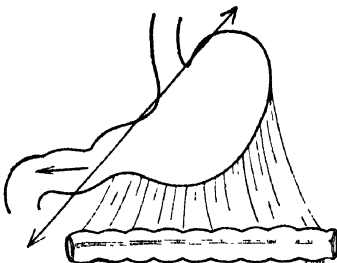


FIG. 950.—Volvulus of stomach—common axis of rotation.

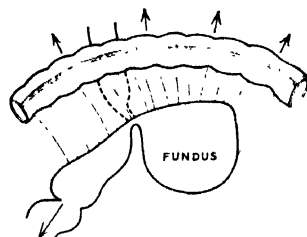


FIG. 951.—Volvulus of stomach—colon passes upwards and greater curve of stomach is inverted.

tal or vertical direction, in its common form the greater curve with the colon moves upwards to lie under the cupola of the left diaphragm (fig. 951). An important predisposing cause of this event is eventration of the diaphragm from whatever cause. It is probable that the colon moves up first and takes the stomach with it. The condition is usually intermittent, but it may present in an acute form. The patient takes a small amount of food and he feels full; there is slight epigastric pain and retching. He has to wait awhile, and he may find that if he lies flat he can then eat some more. The diagnosis is established by a barium meal (fig. 952). It is often impossible to pass a stomach tube.



FIG. 952.—Volvulus of stomach—barium meal findings.

Treatment.—Operation is the only satisfactory form of treatment. The greater curvature of the stomach must be completely freed from the colon by division of the gastro-colic omentum. It is probable that this of itself is adequate. A further safeguard, however, consists of fixing the greater curve to the

duodeno-jejunal flexure (or better, the fourth part of the duodenum), as in a posterior gastro-enterostomy but without a stoma.

Where the rotation occurs as the result of and into a large hiatal defect, the essential treatment consists of closure of the hiatus around the œsophagus.

ACUTE PHLEGMONOUS GASTRITIS

(*syn.* ACUTE SUPPURATIVE CELLULITIS OF THE STOMACH)

Both the anterior and posterior walls of the stomach are congested and swollen, and feel like wet blotting-paper; pus is present in its submucosal layer. In its most acute form phlegmonous gastritis invades the walls of the stomach from the cardia to the pylorus. There is a localised variety that, when situated in the distal part of the stomach, has been treated successfully by partial gastrectomy. The absence of characteristic signs and the rarity of the condition makes preoperative diagnosis impossible. Probably in some cases resolution occurs, and the true nature of the condition never comes to light. In the acute generalised variety, the diagnosis of either perforated peptic ulcer or acute pancreatitis is made. At laparotomy the stomach is found to be inflamed and œdematous. If a hollow needle connected to an aspirating syringe is introduced into the submucosa, thick, muddy pus is withdrawn which is sent for culture and antibiotic sensitivities. A small incision is made through the muscle coats only. Drainage is provided to this locality and to the lesser sac. Post-operative treatment consists of the administration of fluids intravenously, complete rest to the stomach, gastric aspiration continuously or at very frequent intervals, and intense antibiotic therapy, in which the main hope of recovery lies.

DUODENAL DIVERTICULUM

There are two varieties:

1. **Primary diverticulum** of the second and third parts of the duodenum. They occur on the concave border (fig. 1150), are usually single but may be multiple, and commonly arise at the portal of entry of the blood-vessels into the duodenal wall. In 90 per cent. of cases they are an incidental finding during the course of a barium meal. It is wise not to incriminate them as a cause of symptoms (fig. 953) but to make a thorough search for pathology elsewhere. Rarely, a large diverticulum near the ampulla of Vater may cause jaundice. In



FIG. 953.—Typical duodenal diverticulum.

addition, if a diverticulum is persistently tender when visualised during screening or if it retains barium for days, then it may be justifiable to operate. The duodenum is mobilised, turned medially, and the diverticulum dissected free. The duodenum is then opened opposite the diverticulum which is seized, inverted into the lumen, and is tied off. The duodenum is then closed and the fascia over the site of the

diverticulum oversewn. The duodenum is deficient of peritoneum in this area and the operation is not without risk as a fistula may follow.

2. **Secondary Diverticula.**—These occur in the first part of the duodenum and are the result of scarring following duodenal ulceration. They are responsible for the trifoliate duodenum seen on X-ray (fig. 954).

Chronic duodenal ileus is a rare condition which may mimic pyloric stenosis. The duodenum is dilated up to the superior mesenteric vessels. The dilated segment has a characteristically smooth outline (fig. 955). It is a radiological diagnosis rather than a clinical one. Some advocate duodeno-jejunostomy for this condition, but the results are disappointing.

Prolapsing Gastric Mucosa.—This again is a doubtful clinical entity and is really an X-ray finding. Hypertrophied gastric mucosa prolapses through the pylorus (fig. 956 (inset)). Pyloric obstruction has been attributed to this phenomenon.

Syphilis of the stomach produces thickening of the wall and mucosal ulceration. The differential diagnosis is difficult because ulcer or cancer of the stomach may be present in a patient with a positive Wassermann



FIG. 954.—Duodenal ulcer showing typical trefoil or shamrock deformity. (Dr. G. Stachurko, London.)



FIG. 955.—Duodenal ileus. Note the abrupt obstruction to the third part of the duodenum.



FIG. 956.—Showing the 'jockey-cap' deformity. Inset.—The prolapse viewed from the duodenal aspect of the pylorus. (L. H. Appleby, F.R.C.S., Vancouver.)

reaction. Laparotomy is indicated if the lesion does not respond rapidly to a course of anti-syphilitic treatment.

Annular pancreas may cause duodenal obstruction (p. 847).

Giant rugal hypertrophy of the stomach is a descriptive name given to a condition which manifests itself clinically as a protein-losing gastro-enteropathy. Recognition of this feature may necessitate gastrectomy. The thick folds can be demonstrated radiologically, seen at gastroscopy, and palpated at laparotomy. There is not the slightest evidence that this condition is premalignant, nor is it inflammatory. The aetiology is unknown (Johnson).

PEPTIC ULCER

There are two distinct types—duodenal and gastric. Either may present in an acute or chronic form.

ACUTE PEPTIC ULCERS

Ætiology.—As a clinical finding these ulcers appear as multiple erosions in stomachs which are not secreting acid at the time, have marked biliary reflux and the gastric mucus presents as large white globules instead of the usual opalescent fluid. About half the patients give a history of ingestion of aspirin or butazolidine.

August von Wassermann, 1866–1925. Director of the Institute for Experimental Therapy, Berlin.
H. Daintree Johnson, Contemporary. Surgeon, Royal Free Hospital, London.

Classically they present with hæmorrhage. Acute peptic ulcers have been found at post-mortem in hæmorrhagic diseases, uræmia, food poisoning, bacteraemia, and burns (see Curling's ulcer, below).

Pathology.—Acute peptic ulcers are frequently multiple—in 75 per cent. of cases more than three of these lesions are present. They can occur in any part of the stomach, but in the duodenum they are almost confined to the first part. These ulcers are oval or circular in shape, and vary in size from 1 to 2 mm. in diameter (when they are called erosions) to 1 or more cm. in diameter. They are shallow, punched out, and seldom invade the muscular coats. When healing occurs, acute peptic ulcers are unlikely to leave scars.

Clinical Features.—Acute peptic ulcers probably occur with great frequency. They give rise to short-lived attacks of dyspepsia which are not diagnosed, and the ulcer heals. Usually they are recognised only when they cause hæmatemesis. Occasionally an acute ulcer, particularly when it is situated on the anterior wall of the duodenum, perforates. By gastroscopy, it has been ascertained that acute peptic ulcers can be the cause of hæmatemesis at all ages and in both sexes. Such lesions have been seen to progress to chronic ulceration (Tanner).

Treatment.—If possible, the cause must be removed. Under medical treatment acute peptic ulcers tend to heal rapidly. Blood transfusion may be required for hæmatemesis. Dietetic irregularities must be corrected in order to prevent recurrence or chronicity.

Curling's ulcer is an acute ulcer of the posterior aspect of the first part of the duodenum which may follow severe burns. Often multiple, they are found in 9 per cent. of patients dying of burns and may cause severe gastro-intestinal bleeding some days after injury. Bleeding in the first forty-eight hours is due to severe congestion of the gastric and proximal duodenal mucosa which occurs after a burn—often with petechiæ and multiple superficial erosions.

CHRONIC PEPTIC ULCER

Ætiology.—The crucial question is, why do some acute ulcers become chronic? Many theories have been put forward but none is a satisfactory explanation for all the various forms of the lesion.

Incidence.—Peptic ulcer, rare before the age of sixteen, becomes more frequent as middle age approaches, and in England nearly 10 per cent. of men aged forty-five to fifty-four years are thus afflicted. Occupation has some bearing on the condition; thus significantly high incidences are found among doctors, foremen and business executives, while significantly low incidences occur among agricultural workers and sedentary workers, e.g. clerks, civil servants and draughtsmen (Doll). The popular belief that bus-drivers are especially liable to this condition has not been substantiated. In many parts of the world, e.g. U.S.A., Scandinavia, this disease is as rife as it is in Great Britain. Conversely, the incidence among Africans and Asiatics living in their native lands is comparatively low, except in the newly emerging nations where the local population are assuming increasing responsibility. Duodenal ulcer is found four times more commonly than gastric ulcer in patients under the age of thirty-five years, but after forty-five years of age it is only one to two times more common. In Scotland the ratio of duodenal to gastric ulcer is higher than in England, while in India the ratio of duodenal to gastric ulcers is 30:1.

All over the world the incidence of peptic ulcer in women is much lower than in men, and in women the two types of ulcer are about equal. Chronic gastric and duodenal ulcers not infrequently co-exist. In such instances the duodenal ulcer appears first.

Gastric Ulcer.—Chronic gastric ulcer is associated with either normal or hyposecretion, atrophic gastritis, and usually occurs in a later age group. It is a disease of the labouring classes rather than those subject to stress and responsibility. Here again there is a genetic factor, but the pattern of inheritance of gastric and duodenal ulcer is quite separate. With reference to the blood groups, again there is a relationship, but it is less precise than for duodenal ulcer.

Duodenal Ulcer.—The basic factor in this condition is the presence of a large oxyntic cell mass with associated hypersecretion of acid.

(a) *Genetic and Blood Groups.*—There is definite evidence that chronic ulcers occur in families. Where duodenal ulcer occurs in a young man, for instance, there is frequently a strong family history. Moreover, persons of blood group O are about

Thomas Curling, 1811–1888. Surgeon, The London Hospital.

Norman Cecil Tanner, Contemporary. Senior Surgeon, Charing Cross Hospital, London.

Richard Doll, Contemporary. Honorary Associate Physician, Central Middlesex Hospital, London.

three times more likely to develop a peptic ulcer than are persons of other blood groups. It seems possible that the ABO genes may either modify the size of the oxyntic cell mass and therefore the amount of hydrochloric acid that an individual can secrete, or else be associated with failure of the ulcer protective mechanism.

(b) *Neurogenic Theory*.—Stimulation of the vagus results in gastric hypersecretion and hypermotility. Stress and anxiety are undoubtedly a leading cause of duodenal ulcer and these probably exert their effect via the vagus.

(c) *Accessory Causes*.—Inadequate mastication, indigestible food, irregular meals, excessive smoking, and vitamin deficiency have, at one time or another, been blamed, and unquestionably are to some extent predisposing factors. It has been conclusively shown that smoking delays healing in gastric ulcers.

(d) *Endocrine*.—In general, it may be said that the effects of emotional, as well as physical stress are hormonally transmitted to the stomach via the pituitary-adrenocortical axis (fig. 752). There are, however, specific endocrine disorders which may be associated with severe or intractable ulceration. These include (i) the Zollinger-Ellison syndrome, where a non-beta cell tumour secreting 'gastrin' occurs in the pancreas (p. 861); (ii) the multiple adenoma syndrome, where adenomas occur in the pituitary, adrenal, pancreatic, and parathyroid glands (p. 570); (iii) hyperparathyroidism (p. 570).

Pathology.—Ulcers, whether gastric or duodenal, only occur in alkaline mucosa. The ulcer-bearing area is shown in fig. 957. This area is much smaller than the area of atrophic gastritis (fig. 958) which is the essential precursor of gastric ulcer. The ulcer itself occurs in the alkaline mucosa of the atrophic area adjacent to the acid-secreting fundic mucosa. A chronic peptic ulcer invades the muscular coats, which it tends

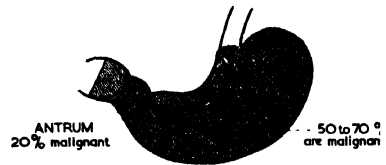


FIG. 957.—*Shaded Area*.—The common area for benign ulcers in stomach and duodenum. *Red Area*.—In this area ulcers carry a high risk of malignancy.

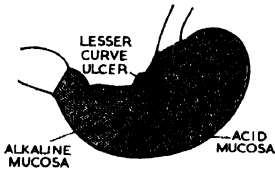


FIG. 958.—Area of gastritis (alkaline) in gastric ulcer. Red area — acid mucosa.

to penetrate. When a gastric ulcer heals, the site is covered by acid mucosa again. The line of demarcation between alkaline and acid-secreting mucosa is not fixed but migrates up and down in the stomach. Fibrosis, the result of recurrent ulceration, causes deformities, including pyloric stenosis and hour-glass contracture.

Chronic Gastric ulcer is usually larger than a chronic duodenal ulcer. It varies in size, but in a well-established case it will admit the tip of a finger. The floor of a chronic gastric ulcer is situated in the muscular coats of the stomach, and as time goes on the ulcer occupying the posterior wall becomes adherent to, and later invades (chronic perforation), the pancreas. In the same way a chronic ulcer situated on the antero-superior aspect of the stomach can penetrate the liver, while a saddle-shaped ulcer situated on the lesser curvature can, and often does, penetrate both the liver and the pancreas.

Chronic Duodenal Ulcer.—The ulcer is nearly always situated in the first part of the duodenum, and sometimes two ulcers are present: one on the anterior surface and one on the posterior surface of the first inch of the duo-

denum. An anterior ulcer may perforate, a posterior one carries the risk of secondary hæmorrhage by erosion of a large vessel.

Microscopical Examination.—There is nearly always greater destruction of the muscular coat than of the mucosa. The base of the ulcer is covered by a thin layer of granulation tissue. The arteries in the neighbourhood show evidence of endarteritis obliterans. Often there are no nerves in the floor of the ulcer but always many in the edge. The terminations of these nerves are bulbous, akin to those in an amputation stump (Kinsella). At the margin of the ulcer there may be epithelial proliferation, and downgrowths of glandular tissue are apt to be found beneath the muscularis mucosæ, which are sometimes misinterpreted as indicating a carcinomatous change.

A chronic duodenal ulcer never becomes carcinomatous. On the other hand, a chronic gastric ulcer may become malignant, but how frequently this change takes place is a matter of great difference of opinion. While a much higher figure has been given in many series, it would seem probable that it does not exceed 4 per cent., and, in the opinion of many, this estimate is too high. The problem is whether the carcinoma occurs in the ulcer or in the surrounding mucosa. Even **giant ulcers** (those with a crater of more than an inch (2.5 cm.) in diameter) are seldom carcinomatous.

Pathology at Operation.—A chronic peptic ulcer may present as a white scar under the peritoneal coat. Delicate vascular adhesions, salmon pink and fluffy in appearance, can often be observed in the immediate neighbourhood of the peritoneal aspect of the ulcer. At other times the ulcer must be sought for by palpation; induration, frequently extensive in the case of a

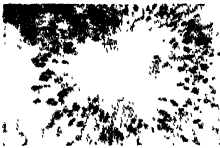


FIG. 959.—Petechial hæmorrhages around the peritoneal aspect of a chronic peptic ulcer. These become more obvious after gently rubbing the surface with gauze.

gastric ulcer, is centred over the mucosal lesion. When the ulcer is situated in the duodenum, the surrounding induration is not so evident, but if the ulcer is situated on the posterior wall it may be possible to feel the crater with the tip of the finger. A useful method of confirming the presence of a peptic ulcer, particularly one situated on the anterior wall of the duodenum, is to rub the peritoneal surface gently with a swab; the peritoneum overlying the ulcer becomes speckled, as though sprinkled with cayenne pepper (fig. 959), a characteristic phenomenon due to minute petechial hæmorrhages.

If there is any real doubt, a longitudinal gastrotomy incision should be made with the diathermy knife and the mucosal area inspected. Ulcers in the pyloric channel (p. 756) will be missed if this is not done.

It is sometimes difficult to be certain whether a given ulcer is gastric or duodenal. The veins of Mayo (fig. 939) are a helpful landmark. It is certain, however, that all benign ulcers lying immediately on either side of the pylorus are due to hypersecretion and will require the same treatment.

THE CLINICAL FEATURES OF GASTRIC AND DUODENAL ULCERS CONTRASTED

It is useful to record the patient's history under seven headings.

Chronic Gastric Ulcer.—The patient is usually beyond middle age, and by reason of a restricted diet is often thin. In many instances the patient appears anæmic, and this is often confirmed by a hæmoglobin

estimation. On careful enquiry certain features of the dyspepsia become manifest. Typically there is :

1. *Periodicity*.—The attacks last from two to six weeks, and are followed by intervals of freedom from two to six months. The attacks are more in evidence in the spring and autumn and should be recorded.

2. *Pain* is epigastric, and may occur immediately, or any time up to two hours, after food. Pain may radiate through to the back, is relieved by lying down flat and practically never occurs at night.

3. *Vomiting*.—In over 50 per cent. of cases vomiting is a notable symptom. It relieves the pain, and may be self-induced.

In the 'pyloric channel' ulcer vomiting may be the predominant or only symptom and the barium meal may be negative. If the symptoms are severe, pylorotomy may be essential in order to make the diagnosis. Pyloric channel deformity may be the precursor of a lesser curve gastric ulcer.

4. *Hæmatemesis and Melæna*.—At some time or other 30 per cent. of patients with gastric ulcers suffer from bleeding from the ulcer. The ratio of hæmatemesis to melæna is about 60 : 40.

5. *Appetite* is good, but the sufferer is afraid to eat.

6. *Diet*.—The patient learns to avoid fried foods, stews, curries, and twice cooked meat. Milk, eggs, and fish are the staple diet.

7. *Weight*.—Usually by the time the surgeon is consulted there has been some loss of weight.

On examination there is frequently deep tenderness in the mid-line of the epigastrium a few inches above the umbilicus.

Chronic duodenal ulcer can occur at any time during adult life, but is commonest between the ages of twenty-five and fifty. It is more common in men, who appear otherwise healthy.

1. *Periodicity* is usually well marked, and classically the attacks come on in the spring and in the autumn and are precipitated by 'work, worry, or weather'. These attacks usually last from two to six weeks, with intervals of freedom from one to six months.

2. *Pain* is severe, and may double the patient up. It usually occurs two to two and a half hours after food. As it is often relieved by food, the pain is known as 'hunger pain', and, classically, the patient carries biscuits, which he eats at frequent intervals to prevent this gastric torment. The pain, which is also relieved by alkalis, often awakens the patient round about 2 a.m. but is usually absent at the normal hour of rising.

3. *Vomiting* is rare in duodenal ulceration unless it is self-induced or stenosis has occurred. Regurgitation of burning fluid into the mouth ('water-brash') together with pain deep to the sternum ('heart-burn') due to reflux œsophagitis are common complaints (1 : 10).

4. *Hæmatemesis and melæna*, which occur in the ratio of 40 : 60, but sometimes together, are rather more frequent than in the case of gastric ulcer.

5. *Appetite* is exceptionally good, but the patient sometimes refrains from eating solid food during the attacks.

6. *Diet*.—In contradistinction to patients with a chronic gastric ulcer,

those suffering from duodenal ulcer who have not been ordered a special diet seldom display much dietetic discrimination, although some of the more intelligent find it advisable to avoid fried food.

7. *Weight*.—Usually there is no loss of weight ; indeed, the patient often becomes plump (partly because of the ingestion of milk to relieve pain).

On examination it is not unusual to find localised deep tenderness in the right hypochondrium.

After about two years the course of the disease becomes steady. There is 1 per cent. risk of perforation and 3 per cent. risk of massive bleeding—1 in 4 require surgical intervention in four to five years. The ulcer that starts with bleeding or perforation is likely to do it again.

SUMMARY

	<i>Gastric Ulcer</i>	<i>Duodenal Ulcer</i>
Periodicity	Present	Well marked
Pain .	Half an hour after food	Two hours after food
Vomiting	Considerable vomiting	No vomiting
Hæmorrhage	Hæmatemesis more frequent than melæna.	Melæna more frequent than hæmatemesis.
Appetite .	Afraid to eat	Good
Diet .	Lives on milk and fish	Eats almost anything
Weight .	Loses weight	No loss in weight

SPECIAL METHODS OF INVESTIGATION

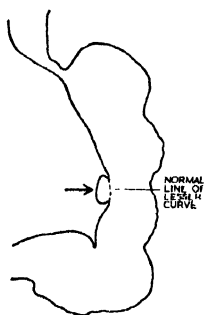


FIG. 960. — Barium meal X-ray showing gastric ulcer projecting from lesser curve. (Dr. Howard Middlemiss, Bristol.)

Barium Meal.—The radiological findings are often conclusive. In a *gastric ulcer* a niche will be seen projecting from the usually smooth outline of the lesser curve (fig. 960). The stomach is often hypotonic and J-shaped and hangs low in the pelvis. In *duodenal ulcer* diagnosis depends on demonstrating an ulcer crater filled with barium—this is positive evidence of an ‘active’ ulcer (fig. 961). In longstanding ulcers so much scar tissue is present that it is often impossible to show an ulcer crater with a persistent flake of barium. Where, however, deformity is *constant* the inference is that there has been a chronic cicatricial process due to an ulcer. In such a case the folds of scar tissue converge on the ulcer site, and if this ‘*rugal convergence*’ can be shown, there is a very strong suspicion that an ulcer is present. It is important to recognise the shape of the normal duodenal cap when distended with barium (fig. 961). The appearances of pyloric stenosis and hour-glass deformity are characteristic (figs. 981 and 982).

Blood Studies.—A hæmoglobin estimation may show evidence of chronic blood loss. A raised erythrocyte sedimentation rate in a gastric ulcer suggests malignancy.

Stool Studies.—Examination for occult blood is important and should be done routinely in such cases on the specimen of fæces on the finger-stall at rectal examination. If the patient has recently ingested meat or aspirin, or has used a stiff tooth brush, a false positive may be obtained.

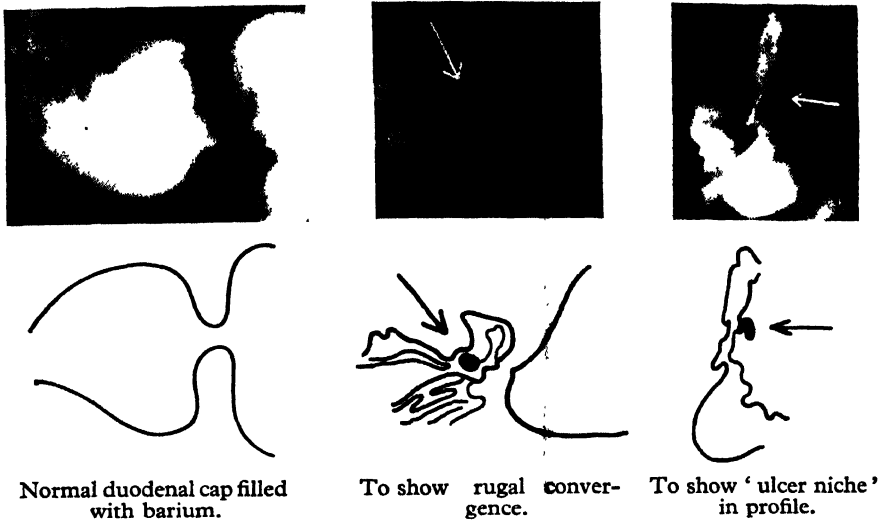


FIG. 961.—Various findings in barium meals of duodenal ulcers. (Dr. Howard Middlemiss, Bristol.)

Gregerson's Benzidine Test is the most useful for this purpose. A powder composed of barium peroxide and benzidine 10:1 is prepared and enough to go on the tip of a penknife is added to 5 ml. of 50 per cent. acetic acid in a test tube. If a few drops are added to a smear of faeces on filter paper, a blue colour appears in ten seconds when blood is present.

Hæmatest.—A drop of water is put on a tablet placed on a thin smear of faeces on filter paper. A blue colour develops in one minute if positive.

Studies of Gastric Function.—The fractional test-meal has been completely superseded. At present (Buckler), there are three tests in common use: Kay's test indicates the size of the oxyntic cell mass, Dragstedt's test the extent of vagal secretion at night, and Hollander's test the integrity of the vagus.

(1) **Augmented Histamine Test (Kay).**—This is useful in diagnosis, in pre-operative selection, and in screening cases of the Zollinger-Ellison syndrome (p. 861). In the latter there is a high basal output which is but little increased by histamine stimulation.

From a fasting stomach, the *basal secretion* is collected for one hour. Mepyramine malleate 100 mg. is given intramuscularly to antagonise the side-effects of histamine. Twenty minutes later, histamine (0.04 mg. per kg. body-weight) is given subcutaneously and the *maximal secretion* collected during the subsequent hour (Table 1). There are several variations of this test and these include continuous administration of histamine or the use of histalog when no antihistamine is necessary.

Achlorhydria, by definition, occurs when a stomach cannot produce juice with a pH of less than 7.1 even after augmented histamine stimulation. It occurs in 18 per cent. of patients with gastric carcinoma (Baron).

(2) **Night Juice (Dragstedt).**—This is a method of assessing the inter-digestive or resting secretion of the stomach. After sedating the patient with intramuscular phenobarbitone, continuous or intermittent low-pressure gastric suction is carried out through an indwelling gastric tube previously screened into position. Normally about 400-ml. secretion is aspirated in the twelve-hour period from 9 p.m. to 9 a.m. Volumes above this level are suggestive of duodenal ulceration, and in Zollinger-Ellison syndrome cases, volumes in excess of a litre occur. The estimation of free HCl content is valuable as an index of vagal activity (Table 2).

Keith Buckler, *Contemporary*. Research Fellow, Royal Infirmary, Bristol.
 Andrew Watt Kay, *Contemporary*. Regius Professor of Surgery, University of Glasgow.
 Jeremy Hugh Baron, *Contemporary*. Reader in Medicine, Middlesex Hospital, London.
 Lester Reynold Dragstedt, *Contemporary*. Research Professor of Surgery, University of Florida, Gainesville, U.S.A.

TABLE 1

Average HCl response in mEq. Free Acid/hr. to Augmented Histamine Test

	Basal	Maximal
Gastric Ulcer	2	14.6
Duodenal Ulcer	6	37.5
Anastomotic Ulcer	7.7	31
Zollinger-Ellison Syndrome	22	

TABLE 2

Free HCl in mEq. in twelve hours' night secretion

Normal	10-20
Gastric Ulcer	5-15
Duodenal Ulcer	40-80
Duodenal Ulcer with Gastric Ulcer	40-60
Zollinger-Ellison Syndrome	100-300

(3) **Insulin Meal (Hollander).**—Since acid production following hypoglycæmia is attributed to direct vagal action on the oxyntic cell mass, insulin given to a patient who has had a vagotomy performed should result in no increase in acid production. This test is thus valuable post-operatively to verify the completeness of vagal section. To a fasting patient, 15 I.U. soluble insulin are given intravenously, and serial quarter-hourly gastric aspirations performed for the next two and a half-hours. Blood-sugar levels are also measured at hourly intervals to ensure that the level falls below 30 mg. per cent.—the level necessary to guarantee adequate vagal stimulation. The test shows incomplete vagotomy if 20 m.eq. per litre of HCl appears in consecutive quarter-hourly samples in the two hours after insulin (Baron).

In addition, the glucose tolerance curve, an X-ray of the pituitary fossa, the serum calcium and phosphorus levels, and the urinary 17-ketosteroids may help to elucidate the difficult case with an endocrine background (multiple adenoma syndrome).



FIG. 962.—The blind areas of gastroscopy.

Gastroscopy requires special training and considerable experience but affords valuable information. By its employment, with the exception of the areas shown in fig. 962, the whole of the interior of the stomach can be adequately scrutinised by the eye of the gastroscopist.

Contraindications.—The passage of a gastroscope is contraindicated in the presence of disease of the œsophagus, aortic aneurysm, or an obvious spinal deformity. It may be difficult in cases with complete dentition, or with osteoarthritis of the cervical spine. In the latter case the posterior pharyngeal wall may be damaged and a retropharyngeal abscess may result.



FIG. 963.—Gastroscopic view of the lesser curvature showing a gastric ulcer above the entrance to the antrum. (W. W. Davey, F.R.C.S.)

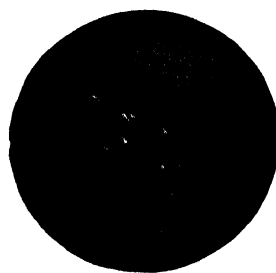


FIG. 964.—Gastroscopy—early carcinomatous change in a chronic gastric ulcer in a patient undergoing medical treatment. (Hermon Taylor, F.R.C.S., London.)

Indications.—Gastroscopy is valuable in the diagnosis of shallow gastric ulcers that do not show on radiography, in checking the results of medical treatment in cases of chronic gastric ulcer, in the differential diagnosis between a chronic peptic ulcer (fig. 963) and a carcinoma (fig. 964), in the diagnosis of a small gastric neoplasm, in the

Frederick G. Hollander, Contemporary. Associate Surgeon, Sharp Memorial Hospital, San Diego, California, U.S.A. Georg Wolf, Contemporary, Optical-instrument Maker, Berlin, and Rudolph Schindler, Contemporary. Associate Professor of Medicine, University of Chicago (formerly of Munich), invented the gastroscope in 1922.

detection of certain forms of gastritis, and in examination of the stoma in cases of suspected gastrojejunal ulcer.

Technique.—Local or general endotracheal anæsthetic may be used. With the patient lying in the left lateral position, the head supported and the knees drawn up, the gastroscope is passed and the stomach inflated with air. The flexible extremity of Hermon Taylor's instrument (fig. 965) can be moved into different parts of the

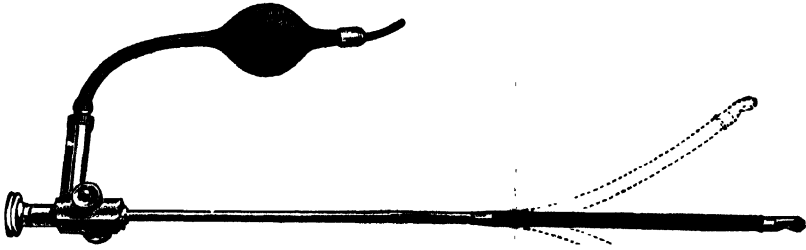


FIG. 965.—Hermon Taylor's flexible gastroscope.

stomach by means of a controlling wheel near the eyepiece, and with this instrument, with the exception of the blind spots referred to, all parts of the ulcer-bearing area can be examined. Positive radiographic evidence is more reliable than negative gastroscopic findings.

The *fiberscope* is an instrument similar in principle to the gastroscope but in which glass fibre provides the lens system. It is thus flexible and the technical difficulties associated with the introduction of the gastroscope are eased. It can be used to inspect the duodenal bulb (Hirschowitz) and to see beyond a gastro-duodenal anastomosis in some cases.

Gastro-photography.—The gastro-camera (fig. 966) was developed in Japan and is now widely used. It gives excellent colour photographs of every part of the gastric mucosa (fig. 967). These are not so easy to interpret as the direct view of the gastroscopist but a greater area of the stomach can be seen. The indications and contraindications

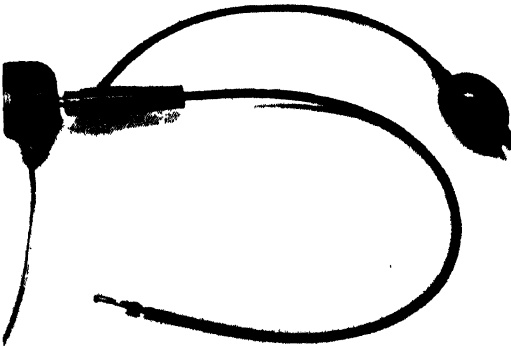


FIG. 966.—The gastro-camera. (From the London Clinic Medical Journal, Dr. G. D. Hadley, London.)

are as for gastroscopy (p. 744). The instrument is slim and flexible and can be passed under omnopon-scopolamine medication. It is passed to its full length and the stomach distended with air. It is withdrawn gradually, frames being shot in three directions at each level. The films are processed and returned in five days and give a permanent record of any lesion.

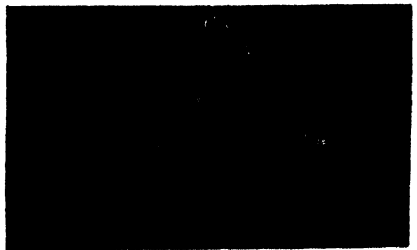


FIG. 967.—Gastro-photograph. A benign prepyloric ulcer. Note the clean yellow-white base and surrounding oedema. (Dr. G. D. Hadley, London.)

TREATMENT OF CHRONIC PEPTIC ULCER

All are agreed that in the absence of complications threatening life, the treatment of a chronic peptic ulcer should in the first instance be medical. When necessary, dental attention should be insisted upon, and as far as possible any other source of focal infection eliminated.

MEDICAL TREATMENT

Duodenal Ulcer.—The essence of treatment is rest in bed in peaceful surroundings with a two-hourly dietary regimen. In the case of a florid ulcer with severe and intractable pain, a trans-nasal intragastric milk drip through fine polythene tubing may be necessary to give relief. The doctor and the sister-in-charge must have ample time to talk to the patient, and thereby unfold any factor or factors that are causing stress, and strive to eliminate them. At least six weeks' rest is essential for the healing of a peptic ulcer with a resulting supple scar. In both duodenal and gastric ulcer the diet must be free from spicy foods and alcohol. All drugs such as butazolidine, aspirin, and cortisone should be avoided. Smoking must be prohibited.

To correct the hyperchlorhydria, it is useful to give alkalis such as magnesium trisilicate, magnesium carbonate, or aluminium hydroxide at frequent intervals, with belladonna. Antigastrin, (a phenylalanine compound), has been synthesised and may prove successful.

Probanthine is an anticholinergic drug and has a useful inhibiting action on secretion. Although it can be used during the day, its greatest value is to control night secretion (i.e. 30 mg. on retiring).

Gastric Ulcer.—Complete rest in bed in the *horizontal* position with only one pillow for three weeks is essential. This causes duodenal regurgitation to cease (p. 741). The diet must be soft, bland, and, where necessary, homogenised. Carbenoxolone is the only drug which has been proved to be of value in this condition.

In all gastric ulcers the effects of medical treatment must be reviewed after three weeks by X-rays, and gastroscopy where possible. If a gastric ulcer is not showing evidence of healing at this time, there is a strong suspicion of malignancy.

When the patient resumes his occupation he must persevere with frequent regular meals, regular hours, suitable food, and no cigarettes. He should lie down for one hour after every meal if practicable.

SURGICAL TREATMENT

Indications for Operation.—This is such an important decision that each patient must be considered individually and judged on his merits. It is wise to let the duodenal ulcer patient 'earn' his operation. If surgery is done early in the disease before he has had some pain or a complication, he will be likely to resent some of the sequelæ of surgery. In general, the following facts suggest the need for surgery:

(a) Intractable pain, or recurrence of pain after previous perforation or hæmorrhage.

(b) Complications, e.g. pyloric stenosis, hour-glass deformity, perforation, or bleeding.

(c) In a gastric ulcer where there is no sign of healing after three weeks' medical treatment or where there is other suspicion of malignancy.

(d) Where there is frequent loss of work.

(e) In general, ulcers which have been present for over five years are unlikely to heal (Watkinson).

Although it is true that the patient who responds well to medical treatment will also respond well to surgery, it is unwise to advocate surgery in women of the child-bearing age, in the young where retardation of growth may occur,

in the underweight, or in any who have a present or past history of tuberculosis.

Operative Treatment.—The aim of surgery in duodenal ulcer is the reduction of the acid secretion to a safe level. Hitherto this has been achieved by partial gastrectomy, an operation which has been widely practised for thirty to forty years. Better understanding of the mechanism of hypersecretion, however, has led to the introduction of other methods, i.e. vagotomy combined with gastro-jejunostomy, pyloroplasty or, more recently, with antrectomy.

Reduction of Acid Secretion by Various Operations

Gastrectomy	Variable (probably about 50%)
Vagotomy 65%
Antrectomy 65%
Vagotomy and Antrectomy 95%

Several factors should be borne in mind in selecting the right operation for any particular patient.

The average national mortality for gastrectomy is 2 per cent., but this figure is greater in the elderly. The mortality in vagotomy with a simple drainage procedure is 0.75 per cent. Gastrectomy is a more difficult operation and usually takes longer. The sequelæ of gastrectomy, both immediate and remote, are now well established. We are not yet sure of the remote sequelæ of vagotomy. From the standpoint of gastric physiology alone the operation of choice is obviously vagotomy with antrectomy.

In gastric surgery, the upper abdomen is usually opened by a right paramedian incision, but if a vagotomy is contemplated, a left paramedian is preferable. Having verified the diagnosis and ensured the absence of other pathology, the ensuing procedure differs according to the actual operation to be carried out.

Operations for Duodenal Ulcer

Historical

In January 1881, Billroth of Vienna performed the first successful gastrectomy, and in September of the same year Wolfier introduced the operation of gastro-enterostomy. The Billroth operations consisted of gastric resection followed by gastro-duodenal anastomosis (Billroth I technique).

Four years later, in 1885, the Billroth II operation was introduced as a two-stage procedure, more by accident than design. A preliminary gastro-enterostomy was done on a gravely ill patient with a pyloric carcinoma. Contrary to expectations, the patient improved, and the stomach was resected, distal to the anastomosis. It soon became evident that the use of a gastro-jejunal anastomosis after gastric resection could be safer and easier than the Billroth I procedure. Now there are some forty modifications in technique of the Billroth II procedure—the main variations involving the use of an antecolic or retrocolic anastomosis, the direction of peristalsis in the jejunal loop, and the use of a full-width stoma or a restricted stoma with a 'valve'.

I. Polya Gastrectomy (Billroth II type)

The lesser sac is opened through the gastro-hepatic omentum and the left hand introduced behind the stomach. Holding the stomach in this manner, the blood vessels and omentum along the greater curvature are divided and ligated. It is safer to do this between the gastro-epiploic artery and the greater curve. It is important at this stage to see that the spleen is mobile, otherwise damage may be done to the short gastric vessels or to the spleen itself. The dissection is carried on to the first part of the duodenum below, and up to the lower short gastric vessels above. The right and left

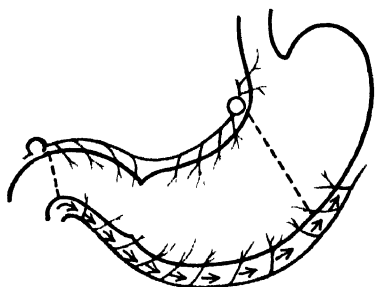


FIG. 968.—The main arteries of the stomach. Circles denote where the R. and L. gastric are divided. Arrows denote place of section of gastro-epiploic arteries. Dotted lines show where stomach is divided. Point of section on lesser curve is at the level of the transverse branch of the left gastric.

gastric arteries are then divided (fig. 968). The duodenum is divided beyond the ulcer if possible, and is closed by two layers of inverting sutures. (This is probably the most critical step of the operation.) The stomach is turned over to the left and a clamp is applied to permit removal of about 70 per cent. of the stomach. The line of section lies from a point on the lesser curve marked by the highest transverse arch of the left gastric artery to a point on the greater curve at the lower short gastric vessels. The cut edge of the gastric remnant is partially closed, leaving a stoma near the greater curve equivalent to the width of the jejunum. End-to-side gastro-jejunal anastomosis is now made, so that the afferent loop is as short as possible. This is achieved by a retrocolic anastomosis at the duodeno-jejunal flexure. If the anastomosis can be made into the fourth part of the duodenum so much the better. The closure of the mesocolic leaf around the anastomosis is of paramount importance.

There are still many surgeons who advocate an antecolic anastomosis. The addition of 'valves' offers no increased benefit (fig. 984). If the surgeon is not absolutely satisfied with the duodenal closure, a soft drainage tube should be put down to, but not touching, the duodenal stump.

II. Vagotomy (Vagus Nerve Section) with Gastric Drainage.—Vagotomy has the twofold effect of reducing hypermotility and hypersecretion of the stomach. Unfortunately it also causes gastric retention. It is therefore essential to perform some sort of 'drainage procedure' such as pyloroplasty or gastro-jejunostomy to correct this. Vagotomy moreover may be followed by severe episodic attacks of diarrhoea. This is less following (1) selective rather than total vagotomy and (2) drainage by pyloroplasty rather than by gastro-jejunostomy; but in any case vagotomy should be avoided in patients who suffer from diarrhoea.

There are two varieties of vagotomy:

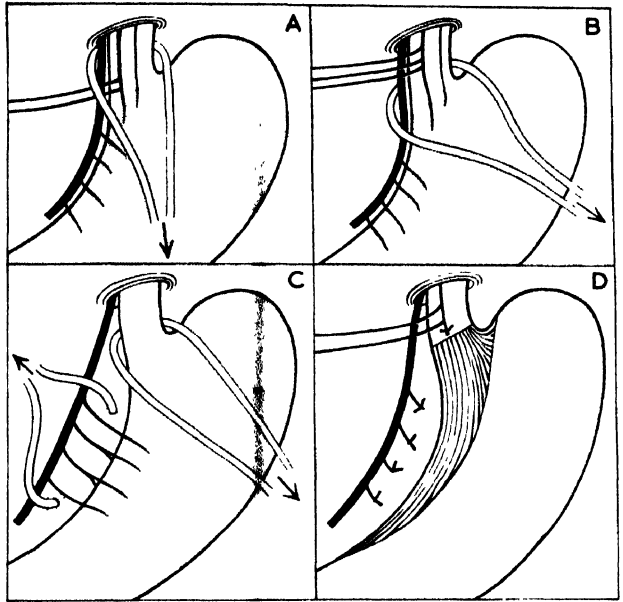
(a) *Total Abdominal Vagotomy* (fig. 969A).—The abdomen is opened by a left upper paramedian incision, the diagnosis is confirmed, and the other abdominal organs are examined. If necessary the left lobe of the liver can be mobilised by dividing the left triangular ligament close to its hepatic attachment. The peritoneum to the left of the œsophagus is incised and the right index finger encircles the œsophagus in order (a) to pass the soft rubber tube which is used for traction purposes, and (b) to identify the position of the posterior vagus trunk, which is thicker than the anterior nerve trunks, and lies almost entirely free from the œsophagus. With the œsophagus taut it is possible to make a thorough search for and divide all the anterior vagal fibres. Three centimetres of the posterior vagus are resected.

(b) *Selective (Specific Gastric) Vagotomy*.—This is best considered in two parts: *Selective Anterior Vagotomy* (fig. 969B). The cardia is exposed as above, but the encircling finger comes out beside the upper part of the lesser curve below the hepatic plexus which can be clearly seen. As the tissues on the front of the œsophagus are ligated and divided, at least 3 cm. of the muscle fibres are bared and cleaned of nerves. *Selective Posterior Vagotomy* (fig. 969C). The rubber traction tube is re-passed between the œsophagus and the separated posterior nerve, and the stomach is pulled to the left. Another rubber traction tube is passed behind the left gastric vessel pedicle, from below upwards, the posterior nerve being separated from the œsophagus as high as possible. All the gastric branches of the posterior nerve will be severed when all the structures in the pedicle are divided between ligatures, and the traction tube falls out.

Burge Test to Detect any Undivided Nerves

A cuffed plastic tube is passed into the stomach. The cuff is inflated to occlude the œsophageal lumen. An encircling electrode is placed around the lower œsophagus. Electrical stimulation, if a nerve is undivided, causes a slightly air-inflated stomach to

FIG. 969.—(See text.) A—Total vagotomy. B—Anterior selective vagotomy. The rubber sling is sited below the hepatic branches. C—Posterior selective vagotomy (after Harold Burge, F.R.C.S., London). The upper sling now excludes the posterior vagus. The lower sling encircles the left gastric pedicle. D—Bilateral selective vagotomy, showing the area cleared of nerve entry and of peritoneum.



contract, and the resultant pressure change registers on a sensitive manometer. Absence of manometer movement indicates complete nerve section. Any drugs having an anti-cholinergic effect must not be given before or during an operation using this test.

GASTRIC DRAINAGE PROCEDURES

There are three main types:

(1) **Gastro-jejunostomy.**—The essential feature of this procedure is to establish an anastomosis at the most dependent part of the stomach, which is the greater curve on the posterior wall. A short-loop posterior operation with a vertical stoma extending to the greater curvature (fig. 970) is the type of operation that gives the best results. In this operation the stoma should be larger than in gastrectomy. Tanner advocates

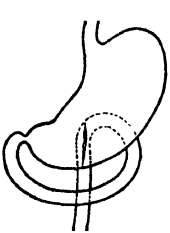


FIG. 970.—The posterior, vertical, short-loop retrocolic gastro-jejunostomy of Mayo.

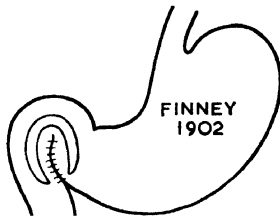


FIG. 971.—Finney pyloroplasty. The adjacent antrum and duodenum is approximated and an inverted U incision is sutured to give a wide gastro-duodenal anastomosis.



FIG. 972.—Heineke-Mikulicz pyloroplasty. A longitudinal incision of 6 cm. is closed transversely.

an anterior anastomosis made in the pre-pyloric area. He considers that the risk of recurrent ulcer is less, and that if it does occur, it is easier to deal with.

(2) **Pyloroplasty.**—The aim here is to produce free drainage by division of the pyloric ring. This can be done by two methods, that of Heinecke-Mikulicz or that of Finney (figs. 971 and 972).

(3) **Antrectomy.**—In addition to being a drainage procedure, antrectomy also abolishes the hormonal phase of gastric secretion. The difficulty lies in the precise delineation of this area. In general three methods are used—(a) excision of the distal

one-third of the stomach including the pylorus, (b) doing a gastrotomy and removal of the area where the mucosa is flattened and not rugose (this can be confirmed by immediate histology), (c) the most accurate method is to define the alkaline mucosa by applying universal pH paper through a gastrotomy incision. The alkaline zone is resected and continuity restored by use of a Billroth I anastomosis, except where the duodenum is grossly fibrotic. Under these circumstances gastro-jejunal anastomosis is safer.

Gastro-jejunostomy.—It would be remiss not to mention the great value of this operation as the *sole* procedure for the treatment of duodenal ulcer. It has been practised for the whole of this century and has given great benefit to hosts of patients. Its mortality is low, but unfortunately the ulcer-recurrence rate following this has steadily increased with time, e.g. it may be as much as 40 per cent. within twenty-five years after operation. It still has a value, however, in the elderly, especially women, with pyloric stenosis in whom the gastric acidity remains low after ten days thorough gastric lavage. Women do not do well after gastrectomy of the Polya type and rarely, if ever, should a woman be submitted to this operation for simple ulceration.

Gastro-jejunostomy	30%
Vagotomy alone	13%
Vagotomy + gastro-jejunostomy	5%
3/4 Gastrectomy	1-4%
Vagotomy + antrectomy (hemi-gastrectomy)	Less than 1%

FIG. 973.—Recurrence rate following operations for duodenal ulcer.

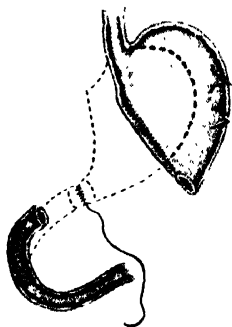


FIG. 974.—Billroth I operation. Dotted lines show rotation of the stomach, mobilisation of the duodenum, and the anastomosis in progress.

Operation for Gastric Ulcer.—The classical operation is the Billroth I partial gastrectomy. This usually includes the ulcer, all of the lesser curve, and one-half to two-thirds of the greater curvature. Recurrence after this operation is less than one per cent. Of recent years pyloroplasty and vagotomy has become increasingly popular for gastric ulceration. If there is any doubt, an immediate biopsy and frozen section must be done to make certain that the ulcer is benign.

The Billroth I Operation.—The stomach is mobilised by the division of the main arteries as shown in fig. 968. It is then partially resected and the remnant is closed, leaving a stoma at the lower end which measures up to the lumen of the duodenum. In addition, the duodenum may be mobilised by Kocher's method, i.e. division of the peritoneum on its convex border. The gastric stoma and the duodenum can then be anastomosed without tension (fig. 974). The important feature here is the construction of as large a stoma as possible.

COMPLICATIONS OF CHRONIC PEPTIC ULCER

- Acute**
 - Perforation
 - Hæmatemesis and/or melæna.
- Intermediate.**—Residual abscess.
- Chronic**
 - Stenosis
 - Pyloric stenosis.
 - 'Tea-pot' deformity.
 - Hour-glass contracture.
 - Penetration into neighbouring viscera, notably the pancreas.
 - Carcinoma.

PERFORATED PEPTIC ULCER

Sex.—The ratio is 19 men to 1 woman (Illingworth).

Age.—The highest incidence is between forty-five and fifty-five years.

Most often a peptic ulcer that perforates is situated on the anterior surface of the duodenum; much less frequently it is situated on the anterior surface of the stomach, usually near the lesser curvature or the pyloric antrum. Rarely an ulcer on the posterior wall of the stomach perforates into the lesser sac. In 80 per cent. of cases there is a history—often a long history—of peptic ulceration. In 20 per cent. there is no such history; it is a 'silent' chronic ulcer that perforates, especially in those patients who are being treated with cortisone. Usually the symptoms of perforation occur with dramatic suddenness.

The gastric or duodenal contents escape through the perforation into the general peritoneal cavity, resulting in peritoneal irritation (peritonism). At that moment the victim cries out in agony, and, at any rate if the perforation is a large one and the stomach is full, he is riveted temporarily to the spot where the perforation felled him. The peritoneum reacts to this chemical irritation by secreting peritoneal fluid copiously and this gives relief of pain for a short time. This stage of reaction lasts from three to six hours, and is followed by diffuse bacterial peritonitis. Following a perforation, gastric secretion is abolished and it is this loss of the acid barrier which permits bacterial invasion to occur.

Clinical Features.—1. *Stage of Peritonism.*—Examination reveals a pale, anxious individual obviously in such pain that he refuses to move. The temperature is usually subnormal, and the pulse-rate frequently in the neighbourhood of 80 or 90, and remains so during the first six hours.

The upper abdomen of a thin subject will be seen to be scaphoid, and it moves little or not at all with respiration (fig. 975). The palpating hand at once recognises an abdominal rigidity which is general and board-like. The whole abdomen is tender and usually dull to percussion. In a small percentage of cases, sufficient gas escapes to cause a diminution of the normal liver dullness in the mid-axillary line. A rectal examination sometimes reveals tenderness in the pelvis. When a comparatively small perforation is situated in the duodenum, the escaping fluid sometimes trickles down the right paracolic gutter to the right iliac fossa. The signs then simulate closely those of acute perforated appendicitis.

2. *The Stage of Reaction.*—The severe abdominal pain lessens, and the patient says he feels better. The temperature becomes normal or elevated slightly, but the pulse-rate is usually still in the neighbourhood

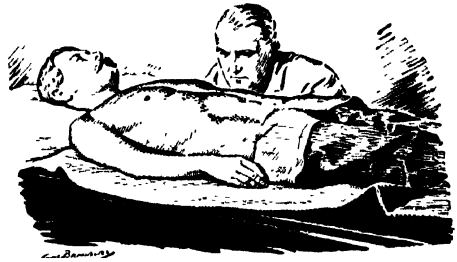


FIG. 975.—A sketch of Mr. Hamilton Bailey watching for abdominal movement on respiration. In cases of perforated peptic ulcer abdominal movement is restricted or absent.

of 90. This temporary improvement in the general condition has been termed the 'period of illusion', and it occurs between the third and sixth hours after the perforation. On examination there is a varying amount of rigidity, but it is not board-like; there is tenderness, and because of ileus associated with chemical peritonitis, bowel sounds are absent.

Radiography.—When a perforation is present, in about 70 per cent. of cases a plain radiograph in the sitting position will reveal a crescent-shaped translucent area beneath the right cupola of the diaphragm (fig. 976).



FIG. 976.—Plain X-ray of a perforated duodenal ulcer showing gas beneath the diaphragm. (Dr. R. Vecht, Bristol.)

3. *The Stage of Diffuse Peritonitis.*—

After six or so hours the signs gradually change to those of diffuse peritonitis. The abdomen slowly becomes distended and the rigidity tends to pass off. By this time enough free fluid may permit shifting dullness to be elicited.

After the sixth hour the pulse-rate increases gradually, and with each passing hour, in the absence of operative treatment, the general condition of the patient gradually deteriorates (p. 870).

Treatment.—Morphine should not be given until written permission for operation has been obtained. Operation, as soon as the general condition permits, is usually the best course. Laparotomy is performed and the perforation is closed with interrupted sutures. In the case of a gastric ulcer a biopsy *must* be taken as there is a high incidence of malignancy. In large perforations it is advisable to reinforce the suture line with a patch of omentum. Thorough peritoneal toilet is essential. With a mechanical sucker fluid and food debris are removed from the peritoneal cavity. Suprapubic drainage of the peritoneal cavity is employed in late cases. The immediate after-treatment consists of continuous gastric aspiration supplemented by intravenous fluid. Antibiotic therapy and breathing exercises are important in the elderly, and in cases where operation is more than six hours after perforation.

Pyloroplasty with vagotomy is being more and more frequently used in this condition especially when the perforation is recent.

Partial gastrectomy is occasionally indicated as an emergency procedure: (a) when the perforation is deemed to have occurred in a carcinomatous ulcer; or (b) when perforation is accompanied by severe hæmorrhage (hæmatemesis and/or melæna). The patient must be in good general condition and the surgeon experienced.

Conservative (Aspiration) Treatment.—Where the diagnosis can be made with assurance, conservative treatment is the method of choice in (1) patients with severe cardiac lesions, emphysema, or some other condition which renders operation dangerous, or (2) if proper facilities for surgery are lacking (Hermon Taylor). The stomach is aspirated hourly and repeated abdominal X-rays are carried out to show

the presence and size of the gas bubble and intraperitoneal or sub-diaphragmatic abscesses. After a few days, a perforation may be sealed by fibrinous lymph.

The treatment requires very frequent observation and control. The method is contra-indicated in the presence of bleeding, pyloric stenosis, after a heavy meal or in air swallowers. There is no conclusive evidence that this method has lowered the mortality even when excellent nursing facilities are available. A perforated carcinomatous ulcer will be missed by this method and not a few deaths following its use are attributable to a mistaken diagnosis. If surgery, in itself, is not likely to be lethal, it is the better procedure.

Follow-up of Patients after Perforation.—As might be expected, there is a transient remission of symptoms due to the rest in bed and the careful dietetic supervision during convalescence. Elderly patients and those of any age with a short dyspeptic history are likely to remain symptom-free after successful treatment of a perforation (Illingworth). Nevertheless, within one year 40 per cent. of patients relapse, and within five years 70 per cent. On this account, those who survive perforation should be followed up as out-patients, so that if symptoms suggesting renewed activity of the ulcer occur, timely treatment can be instituted.

Residual Abscess.—This may occur after perforation in any of the sub-phrenic spaces (p. 873) or in the pelvis.

HÆMATEMESIS AND MELÆNA

Ætiology. —Chronic peptic ulcer	65% of cases.
Acute peptic ulcer (p. 737) }	30% „ „
Multiple erosions }	
Œsophageal varices (p. 810) }	5% „ „
Carcinoma of the stomach (p. 765) }	
Mallory-Weiss syndrome }	
Peptic ulcer in a Meckel's diverticulum (p. 900) }	
Purpura (p. 782) }	
Hæmophilia (p. 76) }	
Pernicious and other anæmias }	

(Compiled mainly from statistics by Dr. F. Avery Jones, London.)

Special Types.—*Chronic Peptic Ulcer.*—Slight bleeding due to trauma from solid food occurs frequently from all chronic peptic ulcers; such bleeding is demonstrated by finding traces of blood during gastric analysis and occult blood in the stools. With advancing age and the associated arteriosclerosis there is an increasing risk of severe hæmorrhage due to the erosion of an artery in the base of the ulcer. Occasionally the artery is of considerable size, viz. the splenic or gastro-duodenal artery; more usually it is a branch of one of these vessels. Once a sclerotic artery has been eroded, even if it is sealed by clot, it is liable to bleed again (fig. 977). Even when a large vessel is eroded, death seldom results from the initial



FIG. 977.—A gastric ulcer with an eroded and aneurysmal artery temporarily sealed by clot. (Norman C. Tanner, F.R.C.S., London.)

Francis Avery Jones, Consulting Physician, Central Middlesex Hospital, London.

hæmorrhage. Far more frequently a large hæmorrhage is heralded by two or three smaller ones on consecutive days, as in other cases of secondary hæmorrhage.

Acute Peptic Ulcer (p. 737).—This may be solitary or present as multiple erosions all over the stomach. The diagnosis is established by gastroscopy and pH of the aspirate which contains no acid. It should be treated conservatively and all drugs withdrawn. If severe and prolonged blood loss occurs then a vagotomy with a Roux-en-Y should be done (Butler). The condition is probably due to acute regurgitation of duodenal contents and a Billroth I should never be used.

Mallory-Weiss Syndrome.—This condition has specific features—the patient, usually a man over fifty, has a prolonged vomiting bout, often after imbibing alcohol. Having vomited gastric contents, he suddenly starts to vomit blood profusely and persistently, and becomes exsanguinated. As a result of the straining and retching a longitudinal tear of the mucosa just below the cardia occurs and gives rise to the sudden onset of hæmatemesis (fig. 978). The diagnosis can be made on the history and is easily

confirmed by gastroscopy. If the condition does not respond to morphia and the ingestion of iced water, laparotomy must be done, when the linear tear can be exposed through a gastrotomy incision and sutured. The violent vomiting is sometimes due to migraine or vertigo.



FIG. 978.—A healing mucosal tear extending across the oesophagogastric junction. (Dr. Michael Atkinson, Worcester, and the Editor and Publishers of 'Gut'.)

Clinical Features in General.

—Initial features are usually faintness, sweating, and pallor; occasionally the patient collapses. Soon afterwards there is hæmatemesis, which is effortless vomiting of coffee-ground material or bright-red blood. Later, black tarry stools (melæna) or red clotted blood may be passed per rectum.

Treatment.—On admission the collapsed patient is laid flat, with one pillow under the head, and the foot of the bed is raised. He is accorded the usual number of blankets for the time of year, and not heated artificially in any way. If restlessness is in evidence, morphine grain $\frac{1}{4}$ (15 mg.) is given intravenously and repeated in four hours if necessary. Pending arrangements for blood transfusion, a plasma infusion is given. The important consideration is to replenish the circulation without overloading it, and a drip blood transfusion proportional to the estimated blood loss should be given. A pulse chart, if necessary half-hourly, is compiled, and a careful watch is kept on the blood pressure or central venous pressure (p. 69).

Very soon after a severe hæmorrhage the hæmoglobin concentration is often unchanged, and therefore no reliance should be placed upon it at this critical time. After three hours, estimations, repeated at frequent intervals, provide helpful information.

As a result of replenishing the circulation, improvement often occurs. Conversely, signs that bleeding has very seriously depleted the blood volume and is probably continuing are a cold nose, increasing pallor, increasing pulse-rate, beads of sweat on the forehead, and clammy palms of the hands (p. 66).

Management:

Medical or Surgical Regimen?—This decision should be made by joint consultation and co-operation between the physician and surgeon. In deciding whether conservative or operative treatment is the better course, they will take into consideration the following factors:

Age.—Seventy per cent. of cases of bleeding are over the age of forty-five. Under forty-five medical treatment is usually successful, but after this age operation is increasingly necessary.

Chronicity of Ulcer.—In general, the patient with a short history is more likely to respond to medical treatment than one whose history suggests a deep penetrating ulcer. Sometimes, on account of cerebral anoxia, the patient's history is unreliable, when it will be necessary to obtain all possible information from the relatives. If he has been treated at another hospital, no time should be lost in contacting that hospital because all-important X-ray evidence may be forthcoming.

Ingestion of Drugs.—The patient must be closely questioned about the ingestion of aspirin, butazolidine, or cortisone. If so, withdrawal of these drugs may suffice.

Condition of Arterial Tree.—The presence of arterio-sclerosis as evidenced by the radial artery, fundal and electrocardiographic changes suggest the likelihood of recurrent bleeding.

Response to Treatment.—Above all, if there is recurrent or profuse bleeding while the patient is under adequate medical treatment, the call for surgery is compelling.

Ancillary Aids.—As far as possible the exact cause of the bleeding should be made before operation. Help may be obtained from the following:

(a) *Barium X-ray.*—In all cases an emergency barium meal should be carried out. If necessary, the patient must be taken to the X-ray department with the blood transfusion still running. This will give a positive diagnosis in over 70 per cent. of cases.

(b) *Œsophagoscopy and Gastrosocopy.*—If the X-ray is negative and, in any case, before operation, the patient should be taken to the theatre and anæsthesia induced and a cuffed endotracheal tube put in position. Œsophagoscopy is then carried out to exclude œsophageal varices and reflux œsophagitis. If such are not present, a large stomach tube is passed and the stomach washed out with several pints of ice-cold water till bleeding abates. A gastroscope is passed when acute ulceration, erosions, or the Mallory-Weiss syndrome may be seen. If blood is seen coming back through the pylorus, it is highly likely that a duodenal ulcer is the cause of the bleeding.

(c) *Gastric Aspiration.*—A low pH (1 to 3) suggests a duodenal ulcer, nocturnal neutralisation, a gastric ulcer and anacidity an erosive gastritis.

Medical Treatment.—The essentials of medical treatment are :

Blood Transfusion.—When the estimated blood loss has been replenished, a drip transfusion is continued at the rate of 30 drops a minute for as long as is deemed necessary. The aim always is to render and maintain the reading of the hæmoglobin level at least above 75 per cent. of normal. In addition, frequent pulse-rate and blood-pressure readings are continued for a minimum three days after the apparent cessation of hæmorrhage.

Morphine.—Sufficient intramuscular morphine or phenobarbitone is prescribed to keep the patient just drowsy. When it is considered that the hæmorrhage has ceased, the dose is reduced very gradually over forty-eight hours.

Intra gastric milk drip is a valuable adjunct for the first forty-eight hours. It is discontinued when no further bleeding is manifest on gastric aspiration.

Diet.—Two-hourly 7-ounce (200-ml.) feeds of milk, egg and milk, alternating with Benger's Food or Ovaltine, are given.¹ After three days milky porridge is allowed. It is essential that an accurate fluid balance-sheet be kept. After a week the patient is given the routine medical treatment for peptic ulcer (p. 746).

Prevention of Pulmonary Complications.—These patients are prone to develop respiratory infection, and not a few of them have bronchitis, with or without emphysema. Regular chest physiotherapy and prophylactic antibiotics are essential.

Operative Treatment.—Should operation be deemed advisable a decision should be made within seventy-two hours of the commencement of bleeding. Experience has shown that when operation is delayed beyond that time the mortality rises steeply.

Before the patient is anæsthetised it is essential to ascertain that the blood transfusion is capable of rapid acceleration. In this connection, sometimes it is advisable, in order to provide a stream of blood equivalent to that of the splenic or the gastro-duodenal artery, to set up a second transfusion into another vein. The aim of the operation is to stop the bleeding. *If an ulcer is found*, a partial gastrectomy is performed including removal of the ulcer. Alternatively, vagotomy and a wide pyloroplasty may be carried out together with direct suture of the bleeding vessel in the ulcer base.

When no ulcer is found on external examination of the stomach, a wide gastrotomy and/or duodenotomy is necessary to expose acute ulcers, multiple erosions or the Mallory-Weiss syndrome (p. 754). To date the best results in multiple erosions come from diversion of duodenal secretion by a Roux-en-Y procedure and vagotomy to prevent an anastomotic ulcer developing (fig. 989). A 'blind' gastrectomy should not be done.

CHRONIC COMPLICATIONS

1. Pyloric stenosis is usually the result of cicatrisation of a duodenal or juxta-pyloric ulcer. Not infrequently it due to a carcinoma situated at or near the pylorus. Classically, pyloric stenosis occurs in women with a slow silent onset, but when there is a long standing history of ulcer the following modifications are evident:

Periodicity is lost.

Pain and fullness become more pronounced towards evening. The patients can usually eat breakfast, a very small lunch, and nothing thereafter because they feel full.

Vomiting.—Very large foul and frothy vomits are characteristic. They usually occur once a day, commonly in the evening. Classically, the patient recognises currants or other undigested particulate matter eaten one or more days previously. Vomiting usually gives considerable relief.

On Examination.—In thin subjects the outline of the enlarged stomach can sometimes be observed. Visible peristaltic waves passing from left to right (fig. 979) are characteristic. These patients may be mentally confused as a result of vitamin B deficiency or of alkalosis.

Barium Meal.—The stomach is large and low (fig. 980), it is never empty, and the barium is mixed with food residue.

Treatment.—Operation in this condition carries a high mortality unless the patient is carefully prepared. Preliminary treatment by gastric lavage with saline through a

¹ Einar Meulengracht, Emeritus Professor of Medicine, Copenhagen, introduced in 1935 early liberal purée feeding in these cases. Before that time absolute starvation by mouth for several days was the unwavering rule.

wide-bore tube, a high-protein fluid diet, and correction of electrolytes and vitamin deficiencies is necessary. Potassium and chlorides are usually deficient. A normal output of urine indicates adequate hydration (p. 82). In cases with a persistently low free HCl content, gastro-jejunostomy gives satisfactory results. If, however, the ulcer is active, partial gastrectomy is indicated; should the condition be due to carcinoma, radical gastrectomy will be required (p. 770).



FIG. 979.—Pyloric stenosis. Wave of peristalsis passing from left to right. Note that outline of stomach extends well below umbilicus.



FIG. 980.—Typical X-ray appearance of pyloric stenosis. Note peristaltic wave in mid-stomach. (Dr. Howard Middlemiss, Bristol.)

2. **'Tea-pot' Stomach.**—Cicatrization around a long-standing gastric ulcer often causes shortening of the lesser curvature, thus producing the 'tea-pot' deformity of the stomach, viz.→

'Tea-pot' stomach (Davey), called in the U.S.A. 'handbag' stomach, is not uncommon. Unless the deformity is known to the radiologist it is likely to be diagnosed as a congenital abnormality or as the result of a carcinoma. The pylorus being no longer in an advantageous position for complete emptying of the stomach, these patients may present with symptoms and signs of pyloric stenosis. Usually partial gastrectomy is required.

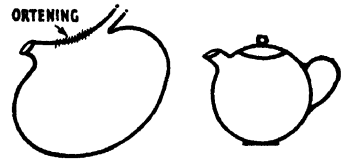


FIG. 981.—Hour-glass stomach.

3. **Hour-glass stomach** occurs almost exclusively in women, is usually silent, and is due to cicatricial contracture around a saddle-shaped lesser-curve ulcer. In extreme cases the stomach is divided into two compartments, united by a channel which barely admits a pencil (fig. 981). The condition is sometimes associated with pyloric stenosis.

History.—*Periodicity* is lost. The symptoms have become practically constant. *Vomiting* is more frequent, and gives no relief to the discomfort.

The *appetite* becomes poor.

Weight.—Loss may be so great that carcinoma is suspected.

Barium meal is often characteristic. We have known of cases of hour-glass stomach being reported as pyloric stenosis, owing to failure of the second pouch

to fill. True hour-glass stomach (fig. 982) must be distinguished from gastric spasm of the hour-glass type, which is sometimes associated with an uncomplicated ulcer on the lesser curvature.



FIG. 982. — Hour-glass contracture of the stomach. (Dr. Graham Airth, Bristol.)

Gastroscope.—The gastroscope enters the upper compartment of the stomach, and the narrow, scarred channel leading to the lower compartment is usually seen.

Treatment.—Billroth I gastrectomy, with removal of the second pouch and the isthmus, is the best treatment.

4. **Penetration of pancreas** may occur with posteriorly disposed gastric and duodenal ulcers.

History.—The main change is that pain is referred to the back and may be aggravated by stooping or exercise and *relieved by lying down*. The back pain may be so prominent that the patient is referred in the first instance to an orthopædic surgeon.

Treatment.—Recognition of this feature of the ulcer is an indication for surgery, not only for relief of pain but also because of the possibility of torrential hæmorrhage.

5. **Malignant Change.**—This occurs in 4 per cent. of cases and is limited to a gastric ulcer or the mucosa around it.

History.—This often gives no guide to the seriousness of the condition. The important point is a constant awareness of the possibility of malignancy at all times. In some cases the pain becomes constant and is unrelieved by vomiting or medical treatment. Any gastric ulcer which proves resistant to three weeks complete bed rest must be regarded as potentially malignant.

Barium Meal.—The size of the ulcer is no guide. The site is important (fig. 957).

Gastric Secretion Test (p. 743).

Gastroscope (p. 744) is essential because it is the most certain method of making an early diagnosis in the doubtful case. Failure of the ulcer to heal can be confirmed accurately by this manœuvre. In addition, direct evidence of malignant change may be seen, possibly confined to one sector of the edge (fig. 964).

COMPLICATIONS AFTER GASTRIC OPERATIONS

(1) Complications following any operation (e.g. pulmonary, cardiac, thrombotic).

(2) *Specific* complications of gastric operations.

A. Early

1. Hæmorrhage from anastomotic line.
2. Stomal obstruction.
3. Duodenal fistula.
4. Afferent loop distension.
5. Acute post-operative pancreatitis.

B. Remote

6. Recurrent ulcer.
7. Gastro-jejuno-colic fistula.
8. Post-gastrectomy syndromes.
9. Intestinal obstruction.
10. Pulmonary tuberculosis.
11. Carcinoma.

Specific Early Complications

1. **Hæmorrhage from Anastomotic Line.**—In the present day anastomotic clamps are seldom used and as a result this complication is rare. If it does occur, morphia should be given together with a slow blood transfusion. Adrenaline (1 ml. (1 : 1000) solution hourly) by mouth may be helpful. If the hæmorrhage is *severe and persistent*, the abdomen should be re-opened, continuous gastric lavage started and the anastomosis oversewn with continuous through-and-through catgut. The stomach is not opened. With control of the hæmorrhage, the washouts become clear.

2. **Stomal Obstruction.**—This includes two different conditions: (a) Due to oedema of the actual stoma or of adjacent small bowel. This occurs more frequently after a gastro-duodenal anastomosis—hence the need for a large stoma in this operation. The mechanism is unknown and it is important to persevere with gastric aspiration and intravenous therapy maintaining correct electrolyte balance, especially of potassium. Hydrocortisone may also be used.

(b) Due to the early occurrence of *retrograde jejuno-gastric intussusception*. This, of course, only occurs after a gastro-jejunal anastomosis and may appear immediately or after many weeks or months. The stomach completely fails to empty and a filling defect can be clearly seen on X-ray with a small amount of barium. The intussusception may reduce following the barium or gastric saline lavage or the adoption of the sitting or standing position. If it does not respond to these measures (and it usually does in a few hours), an operation must be done to reduce the intussusception by gentle downward traction. If the condition recurs, it is wise to convert the gastro-jejunal anastomosis to a Billroth I with vagotomy if necessary.

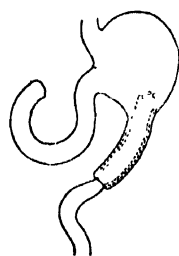


FIG. 983.—Jejuno-gastric intussusception following Polya gastrectomy. (After P. F. Early.)

3. **Duodenal fistula ('Blow-out')** is an infrequent, but very serious, complication of Polya partial gastrectomy with a maximal incidence about the fourth day. It may occur in cases where there has been difficulty in closing the duodenum so that avascular necrosis occurs; but the more usual cause is raised tension within the afferent jejunal loop, due to temporary obstruction at the site where that loop has been joined to the stomach; as a rule the point of obstruction is at A (fig. 984).

Prevention.—There are two important points in technique:

(a) Careful duodenal closure with meticulous turn-in. A crushing clamp should not be used.

(b) Construction of the stoma in such a way that afferent loop contents have free egress. This is achieved by minimal turn-in and the avoidance of so-called 'valves' (fig. 984). Multi-lumen tubes have been used to drain the afferent loop, but unless they are stitched into position, the tube is usually rejected. A tube drain down or into the duodenal stump is a worthwhile safeguard if trouble is anticipated. When a blow-out occurs the duodenal sutures give way and, if drainage was not provided at the time of the operation, there is intense thoraco-abdominal pain, which is not infrequently mistaken for acute basal pneumonia with pleurisy. Unless prompt action is taken, diffuse peritonitis ensues, and death is the usual sequel.



FIG. 984.—Obstruction to the afferent loop. A Polya operation with a Hofmeister-Finsterer valve has been performed.

Treatment consists in providing free drainage down to the duodenum with peritoneal toilet by suction. When drainage of the periduodenal tissues was not provided at the time of the gastrectomy, a small subcostal incision is made down to the duodenum. In either event, sump suction drainage (fig. 1102) is instituted until, and even after, the track of an external duodenal fistula has become defined. It is wise to do a temporary jejunostomy at the same time. The duodenal discharge can be collected and returned into the jejunostomy together with the necessary nutritive fluids (p. 735). The fistula usually closes spontaneously.

4. **Afferent loop distension** (p. 852) is a rare but very serious complication of gastrectomy (fig. 984). The symptoms which appear in the first few days after operation are difficult to distinguish from those of a leaking duodenal stump. The much elevated serum amylase suggests the diagnosis, which is confirmed by the absence of bile in the gastric aspirate. Immediate surgery is mandatory.

5. **Acute post-operative pancreatitis** is even more rare and the diagnosis usually is made in mistake for afferent loop stasis. Treatment is along the usual lines (p. 854).

REMOTE COMPLICATIONS

6. Recurrent Ulcer.—This includes the true anastomotic ulcer (gastro-jejunal, gastro-duodenal or jejunal ulcer) or a gastric ulcer in the remnant. True anastomotic ulceration has an incidence of 2 per cent. after a Polya gastrectomy, 5 to 7 per cent. after vagotomy and gastro-enterostomy, and

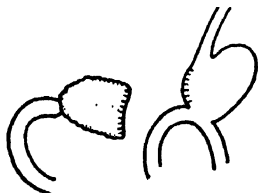


FIG. 985. — Finsterer-Devine operation. The antrum (left *in situ*) bathed in alkaline juice produces a constant secretion of gastrin.

up to 40 per cent. following gastro-enterostomy alone. If any part of the antrum is left *in situ* after a gastrectomy (Finsterer-Devine operation, fig. 985), the recurrence rate may rise to 30 per cent. The symptoms which appear usually within two years after operation consist of severe persistent pain which is worse within a few minutes of taking food. The pain passes down the left side of the abdomen but may be in the lower left chest especially following an ante-colic anastomosis. Bleeding manifest as hæmatemesis, melæna, occult blood, or secondary anæmia is common and perforation may occur. Quite often this condition defies identification by a barium meal and/or gastroscopy and, if the symptoms are severe, laparotomy must be done.

Management.—Relapse is common after medical treatment and re-operation is usually necessary. If the recurrence is a gastric ulcer in the gastric remnant, then a secondary gastrectomy is the operation of choice. If the ulcer is anastomotic, it is essential that acid secretory studies should be done. There are five possibilities:

(a) **Anastomotic ulcer following Polya with persistent hypersecretion** of more than 25 m.eq. HCl. If it is certain that all the antrum has been removed then vagotomy is adequate.

(b) **Anastomotic Ulcer after Polya with Low Secretion.**—This ulcer is often associated with fibrosis and has an arterio-sclerotic background. A secondary gastrectomy and a new separate anastomosis made without clamps is desirable.

(c) **Anastomotic Ulcer following Gastro-enterostomy.**—A vagotomy is usually all that is necessary. If there is hypersecretion, an antrectomy may be added.

(d) In the presence of **gross hypersecretion** a search must be made for the ulcerogenic pancreatic tumour (p. 861).

When severe hæmatemesis necessitates further operation, and it is found that the hæmorrhage is from the middle colic artery, in addition to measures to remedy the gastro-jejunal ulcer, ligation of the middle colic artery may so endanger the vascular supply of the transverse colon that this, too, must be resected.

(e) Finally, where a *Billroth I* gastrectomy has been done for a *duodenal ulcer* there is a 15 per cent. recurrence rate of stomal ulceration. The best procedure here is to convert the Billroth I to a retrocolic Polya gastrectomy.

7. Gastro-jejuno-colic fistula is a complication of gastro-jejunal ulcer, and is seen more often following simple gastro-enterostomy than partial gastrectomy. The ulcer penetrates and erodes the transverse colon. Usually the symptoms of anastomotic ulcer disappear soon after the fistula develops, but in their place the unfortunate patient is troubled with severe diarrhœa after every meal, and eructates foul gas. Exceptionally the patient vomits fragments of formed fæces. Loss of weight and strength, dehydration, and anæmia complete the picture. It is important to stress that this final picture may be of extremely rapid onset so that the patient becomes



FIG. 986. — Jejunal ulcer following gastro-jejunostomy. (Dr. K. J. Yeo, London.)

desperately ill within two to three weeks. Severe malabsorption is shown by the presence of cachexia, hypoproteinæmic œdema, and steatorrhœa. The chief factor producing the rapid deterioration of the patient is the fouling of the jejunum by colonic contents with resulting disturbance of the vital absorptive mechanisms. It is *not* that the gastric contents pass directly into the colon. The diagnosis is established by a barium enema; in more than half the patients a barium meal may fail to reveal a fistula. *The sudden onset of severe diarrhœa in a patient who has gastro-enterostomy must always raise the serious possibility of this condition.* Both diagnosis and treatment are urgent.

Treatment.—Some of these patients are extremely ill, in which case the first step is a lower laparotomy—almost an emergency operation—in which the ileum is divided near the ileo-cæcal valve and an end-to-side ileo-sigmoid anastomosis is done (Lahey). The purpose of this manœuvre is to deviate intestinal contents from the colon in order to prevent fouling of the jejunum. Proximal colostomy has the same effect. In many cases ileo-colostomy is all that is necessary. If, however, the patient has persisting diarrhœa, then a second-stage operation should be done. This consists of a resection of the fistula with repair of the colon and jejunum, together with either vagotomy or a high partial gastrectomy. Treatment of this condition can be one of the most difficult procedures that any surgeon may be called upon to do.

8. Post-gastrectomy Syndromes.—These are special complications directly attributable to altered alimentary function following the operation. The proper understanding of all these syndromes depends on a thorough knowledge of the physiology of intestinal absorption.

Physiology.—The main functions of the **Stomach** are: (1) It acts as a reservoir for food. (2) It churns and mixes the food to make it isotonic and sterile, and it adjusts the physical and chemical characters of the mixture to facilitate breakdown by enzymes. (3) It secretes intrinsic factor which is essential for the absorption of Vitamin B₁₂. (4) It passes its prepared contents in small amounts into the duodenum at regular intervals (about 12 ml. per minute).

Absorption is the prime function of the **Small Intestine** and it is essential to appreciate that there are *zones of maximal and selective absorption* (fig. 987). The first 100 cm. of small intestine distal to the pylorus are of great importance for in this zone the maximal absorption of carbohydrate, fat and protein occurs. The duodenum is the site of maximal absorption of carbohydrate, water soluble vitamins, iron and calcium. Vitamin B₁₂ is maximally absorbed in the terminal ileum. Folic acid is absorbed generally all over the small intestine.

The recognition of these essential functions of the small intestine is important for two reasons:

(1) Following gastrectomy food enters the small intestine rapidly without preliminary preparation by the stomach. This may bring into relief certain latent defects of absorption.

(2) When a gastro-jejunal anastomosis is made after gastrectomy a part of this important proximal 100 cm. is by-passed and in this vital matter of absorption even a few centimetres make a difference.

Summarising, therefore, gastrectomy may cause reduced secretion of HCl, pepsin, intrinsic factor and pancreatic enzymes, inadequate mixing of food with enzymes and bile, reduced absorption of protein, fat and iron, rapid absorption of glucose, altered

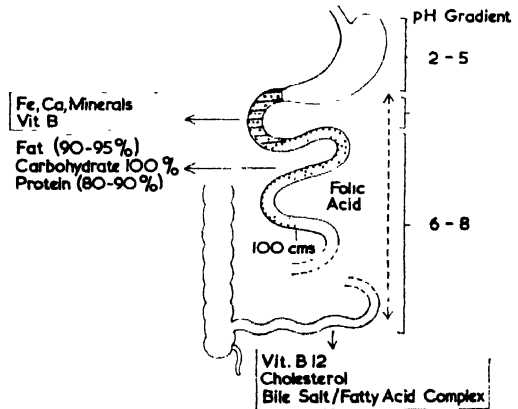


FIG. 987.—Absorption areas of small intestine.
(After T. J. Butler.)

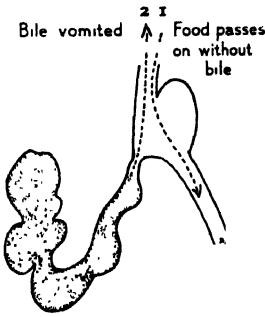


FIG. 988. — The afferent loop syndrome. The food (1) passes out of the gastric remnant before the bile (2) is ejected. (After Charles Wells.)

bacteria of small bowel, increased intestinal motility, and creation of a blind loop (i.e. the afferent loop, fig. 988). It is remarkable that most cases do so well!

The effects of some of those changes may appear a few weeks after operation, especially the post-cibal syndromes. It may be several years, however, before the absorption defects produce apparent nutritional disturbances.

The various **Post-gastrectomy Syndromes** therefore may be classified as follows:

A. Post-cibal Syndromes

- (i) Early post-cibal syndrome (*syn.* early dumping).
- (ii) Late post-cibal syndrome (*syn.* hypoglycæmia or late dumping).
- (iii) Bilious vomiting.

B. Nutritional Syndromes

- (i) Weight loss.
- (ii) Steatorrhœa.
- (iii) Diarrhœa.
- (iv) Iron-deficiency anæmia.
- (v) Megaloblastic anæmia.
- (vi) Vitamin B deficiency.
- (vii) Calcium deficiency.
- (viii) Gross malabsorption states.

By and large the higher the resection, the greater the risk of post gastrectomy troubles. In addition, these syndromes are best prevented if the normal duodenal pathway is maintained—and not by-passed.

Post-cibal Syndromes—Early and Late.

In general, these occur more frequently in women than in men, and are seen following the Polya operation rather than the Billroth I. Time relation to meals permits classification into early and late varieties; the main features of these are summarised in the following table:

	<i>Early</i>	<i>Late</i>
1. Incidence	5–12 per cent.	5 per cent.
2. Relation to meals	Immediately afterwards	During second hour afterwards
3. Duration of attack	Thirty to forty minutes	Thirty to forty minutes
4. Long-term	Severe cases indefinitely	Two to five years
5. Relief by	Lying down	More food, glucose
6. Aggravated by	More food	Exercise
7. Precipitating factor	Bulk of food, especially if wet	Carbohydrates
8. Chief symptoms	Epigastric fullness Sweating Sensation of warmth Tachycardia Occasionally colic, diarrhœa	Tremor Faintness Epigastric emptiness Nausea
9. Occurrence	Commonest after Polya	After any type of gastrectomy

The Early Post-cibal Syndrome.—This syndrome, consisting of abdominal and vasomotor symptoms, is seen in the majority of patients during convalescence, and persists in 5–12 per cent. The clinical features are best regarded as a result of a reflex, initiated in the jejunum, and transmitted by the sympathetic nervous system. A variety of causes may be involved—rapid emptying of the stomach with increased small bowel activity, distention of the afferent loop, and a fall of blood volume (of the order of 7 per cent.), especially in patients susceptible to such changes. According to Le Quesne, there is a primary disorder of carbohydrate metabolism. Following the ingestion of glucose, there is initial transient hyperglycaemia. This suppresses further absorption of glucose, which is retained in the intestine, resulting in fluid shift from the blood to the lumen of the intestine. Both increased intestinal activity and fall of blood volume may occur.

Treatment.—Time must be allowed to elapse for the syndrome to subside naturally. Meals should be small in bulk and dry. Milk and carbohydrate meals should be avoided. Belladonna or codeine may reduce intestinal activity. Insulin or an oral hypoglycaemic agent before food frequently has a beneficial action. It is important to maintain the haemoglobin level as near 100 per cent. as possible. When the syndrome persists, especially when associated with nutritional defects, conversion of a Polya anastomosis to a Billroth I is the operation of choice.

Late Post-cibal Syndrome.—This is not a common or a serious cause of symptoms. It is due to a low blood sugar, occurs two hours after meals, and may be seen in some 5 per cent. of patients. After the initial hyperglycaemia there is a rapid fall of blood sugar to 50 mg. per cent. or so. This rapid fall is probably due to increased insulin sensitivity (Butler). The syndrome is readily corrected by taking food, especially glucose.

Bilious Vomiting.—This symptom is usually an isolated feature but may be associated with dumping. It occurs in 10 to 15 per cent. of patients and consists of vomiting of bile unmixed with food. The vomiting is intermittent rather than after every meal. In the Polya operation the cause is probably transient obstruction of the afferent loop. Following the Billroth operation it is more likely to be due to incompetence of the cardia. This symptom can be eliminated almost entirely by a retrocolic anastomosis with the shortest possible afferent loop, i.e. into the fourth part of the duodenum if this is possible. If surgery is necessary to correct the vomiting, conversion to a Billroth I should be done if there are other post-cibal symptoms. If bile vomiting occurs alone, conversion to a Roux-en-Y anastomosis is preferable (fig. 989).

Nutritional Disturbances

I. Weight Loss.—This is a well recognised feature, occurring in some 50 per cent. of patients. It is the rule after total gastrectomy, common after the Polya operation, and infrequent after the Billroth I. It is due to reduced intake of food, and diminished absorption, and is particularly liable to occur in patients with post-cibal symptoms. It is not necessarily serious, and indeed may have a beneficial effect. There is a reduced incidence of coronary disease following gastrectomy.

If severe, correction may be achieved by small frequent meals of high protein, high calorie content. Anabolic steroids—Methandienone 50 mg. daily, often helps. It is essential in these patients to exclude reactivation of previous tuberculous disease.

II. Steatorrhœa.—This has a similar incidence to weight loss, and is due to poor mixing of food and enzymes, reduced pancreatic output, and inactivation of pancreatic enzymes in the afferent loop. If it appears slowly after operation, the cause may be latent intestinal disease such as jejunal atrophy or the development of a cul-de-sac phenomenon (i.e. blind loop, fig. 1147) in the afferent loop.

III. Diarrhœa.—Most patients say that their bowel habits are much improved

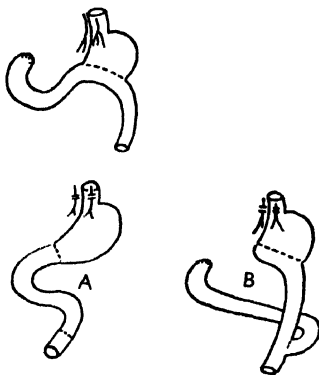


FIG. 989.—Conversion operation. A to Billroth I. B to Roux-en-Y loop. Vagotomy should be added if high acid secretion is present.

after gastrectomy, but some 5 per cent. have diarrhoea, either episodic or persistent. It may be related to intestinal hurry or to steatorrhoea.

IV. Iron-deficiency Anæmia.—This is a progressive condition after gastrectomy, and may be seen in some 40 per cent. of patients, particularly when the duodenum is by-passed. If occult blood loss is excluded, daily supplements of iron are essential (ferrous sulphate 180 mg. t.d.s.). Only very rarely is the anæmia refractory to oral iron.

V. Megaloblastic Anæmia.—Although invariable several years after total gastrectomy, it is rare following partial gastrectomy. It may occur as an isolated lesion due to gastric mucosal atrophy, or as part of a gross malabsorption defect (*vide infra*). There is evidence of reduced serum vitamin B₁₂ levels in some 25 to 50 per cent. of patients five to ten years after partial gastrectomy. Intramuscular vitamin B₁₂ (Cyanocobalamin) 100 mg. weekly is given until the blood is normal; thereafter a maintenance dose each month is necessary.

VI. Vitamin B Deficiency.—This occurs in roughly 10 per cent. of patients, and is manifest by angular stomatitis, glossitis, or peripheral neuritis. A regular supplement is the best method of prevention.

VII. Calcium Deficiency.—Reduced acidity in the proximal intestine may interfere with calcium absorption, and some 40 per cent. of patients may become deficient. Bone changes may occur in some 5 per cent. and although such changes are usually seen associated with steatorrhoea, they may occur independently.

VIII. Gross Malabsorption States.—Very rarely, in less than 1 per cent. of patients, a severe malabsorption syndrome may develop—with wasting, gross vitamin deficiencies, hypoproteinæmia, œdema, steatorrhoea, creatorrhoea, and anæmia. It is usually associated with a long afferent loop. These are examples of the 'cul-de-sac' syndrome, but some may be due to latent intestinal absorption defects brought into relief by gastrectomy. Conversion to a Billroth I is usually very effective in correcting this serious condition.

9. Intestinal Obstruction

(a) Herniation Obstruction.—The condition is more liable to occur in cases of a Polya gastrectomy with an antecolic anastomosis. Small intestine herniates through a gap between the anastomosis and the transverse mesocolon, either from right to left or from left to right. It is easily prevented by a few stitches between the anastomosis and the transverse mesocolon at the time of gastrectomy.

Clinical Features.—Usually symptoms of high intestinal obstruction commence between the third and eighteenth post-operative days, but often these symptoms soon become atypical because gastric aspiration is instituted after the first or second vomit. For the same reason colicky pains, so typical of intestinal obstruction, are often lacking; in most cases the pain is continuous, and increases in severity.

Treatment.—Even if the condition is only suspected, the abdomen should be re-opened. The hernia is usually reducible, but in 30 per cent. of cases gangrene has occurred from long delay. The gap should be closed as detailed above and gangrenous bowel resected if necessary.

(b) Bolus Obstruction.—The commonest cause of this is a piece of un-masticated orange pith which, in the absence of the pylorus is able to pass into the small intestine and lodges about two feet from the ileo-cæcal valve. Clinically, it is not unlike gall-stone obturation (p. 947) but occurs in a younger age group. *Every patient who has had the pylorus excised or by-passed must be told not to eat orange pith or dried fruits.*

10. Pulmonary Tuberculosis.—Gastrectomised persons show a slight but definite increased susceptibility to pulmonary tuberculosis, probably due to reactivation of a latent focus owing to diminished absorption of fat.

11. Carcinoma.—Following a gastrectomy, especially for a gastric ulcer, the gastric remnant is slightly more prone to develop a carcinoma than is a normal stomach. This risk appears after a latent period of fifteen to twenty years or more. It is because of such a complication following gastrectomy that the recent results of vagotomy plus a drainage procedure must be accepted with some reserve. Carcinoma may follow this operation also.

GASTRIC NEOPLASMS

Benign Tumours.—Although they constitute 3 per cent. of all gastric neoplasms benign tumours of the stomach are so over-shadowed by the frequency and gravity of gastric carcinoma that they tend to be forgotten.

Leiomyoma is the most common benign neoplasm of the stomach. Sometimes it grows mainly towards the serosal coat, in which case it is symptomless, and attracts notice only when it is large enough to constitute a painless, smooth lump in the epigastrium. It may cause a low-grade fever. More usually a leiomyoma protrudes towards the lumen of the stomach, and gives rise to melaena, mild indigestion, and epigastric pain, in that order. Hæmatemesis is, however, the most common symptom, and results from deep central ulceration of the tumour. Radiography after a barium meal reveals a space-filling lesion. Necrobiosis of the tumour has led to perforation of the serosal surface with intraperitoneal bleeding, and even to perforation of the stomach. Local excision is curative.

Neurofibroma.—A tumour arising from a nerve sheath gives rise to exactly the same symptoms as a leiomyoma from which, macroscopically, it is indistinguishable. Microscopically it requires an expert to distinguish the two conditions.

Adenomatous polypus, sometimes single, but more often multiple, is, as a rule, situated in the distal half of the stomach. Usually the symptoms are bleeding and abdominal pain. Achlorhydria is present in nearly all cases. Rarely a pedunculated adenomatous polypus of the pyloric antrum is carried into the pyloric canal, there to cause pyloric obstruction. The diagnosis rests on gastroscopy and/or X-ray. It is wise to regard multiple polypi as premalignant, and accordingly perform partial gastrectomy so as to include all the polypi; a single polypus can be excised locally.

Adenomatous Polypi and Pernicious Anæmia.—In pernicious anæmia there is severe atrophy of that portion of the gastric mucosa containing the fundal glands, and this provides a fertile bed for precarcinomatous adenomatous polypi to flourish. In about one in five cases of pernicious anæmia gastroscopy reveals adenomatous polypi. It is for this reason that patients suffering from pernicious anæmia are recommended to undergo annual gastroscopy and a barium meal.

An aberrant pancreas has been found arising in the wall of the stomach from time to time. Usually partial gastrectomy has been performed by a surgeon mistaking it for a carcinoma.

CARCINOMA OF THE STOMACH

Carcinoma of the stomach is one of the 'captains of the men of death'. In 1959, 14,076 persons died from this cause in England and Wales but, fortunately, it now appears to be decreasing. While from early adult life to senility no age is exempt, the highest incidence is between forty and sixty years of age. Three times as many males as females fall victims to this disease.

Ætiology.—There are a few definite pre-malignant conditions. These are a gastric polypus, pernicious anæmia, and possibly, but only rarely, a gastric ulcer. Apart from this the ingested carcinogens seem to be important. Examples of these include the hydrocarbons of cooking fat and tobacco juice; furthermore, substances that cause an irritative gastritis, such as neat spirits as drunk in Scandinavia, Holland, and Czechoslovakia, may increase the incidence of carcinoma. The geographical distribution of this disease is important and may prove a most profitable avenue in the study of ætiology. An interesting example of this is the differing incidence in the North and South of Iceland. Crude smoked salmon was the staple diet of the Northerners who have a much higher incidence than those in the South. Aird found a higher incidence of gastric carcinoma in people with the blood group A.

Pathology.—*Macroscopically* five types can be recognised:

Type 1.—A cauliflower-like growth with sharply defined edges. Its surface is indurated. Later it ulcerates.

Type 2.—An ulcer with an irregular indurated edge. Sometimes small superficial ulcers are present in the immediate vicinity.

Type 3.—Colloid carcinoma.

Type 4.—Leather-bottle stomach.

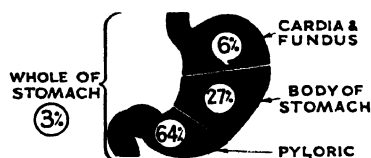


FIG. 990.—Incidence of carcinoma in various portions of the stomach.

arises within the œsophagus.

The most common site for the neoplasm is in the prepyloric region (fig. 990); when carcinoma follows pernicious anæmia however, it is more likely to be fundal and polypoid.

The Spread of Carcinoma of the Stomach.—No better example of the various modes by which carcinoma spreads can be taken than the case of the stomach.

1. *Direct Spread.*—As the growth enlarges, it tends to invade neighbouring structures. The pancreas, transverse colon, mesocolon, œsophagus, or liver may be involved.

2. *Lymphatic Spread* (fig. 996): both by emboli and permeation.

3. *Spread by the Blood-stream.*

4. *Transperitoneal Implantation.*—Carcinoma cells sometimes pass from the stomach into the peritoneal cavity. They gravitate to the pelvis, where secondary tumours palpable on rectal examination may develop. On occasions, in the female, they alight upon the ovaries, giving rise to **Krukenberg's tumours** (fig. 991), which are liable to cause diagnostic confusion. These tumours are premenopausal, because after the climacteric the ovaries atrophy and thus are unlikely to give sustenance to tumour cells. In some cases Krukenberg's tumours are not the result of transœlomic implantation, but are due to retrograde lymphatic permeation, especially from a carcinoma of the colon.

Clinical Features.—This is a very difficult disease to diagnose early, not only because of the diversity of its presentation but also because of the time-lag between the commencement of the growth and the appearance of symptoms. These may be considered as due to interference with:

(1) Nerve endings—persistent pain: no response to treatment—no periodicity.

(2) Gastric distension: fullness, inability to take a normal meal, vomiting.

(3) Gastric juices—anorexia leading to loss of weight.

(4) Blood formation—anæmia, tiredness, weakness, pallor.

In general, it may be possible to consider the presentation under the following clinical groups:

Type 5.—Carcinoma secondary to a chronic gastric ulcer. Probably about 4 per cent. of carcinomas of the stomach arise in this way.

Microscopically.—The growth is usually columnar-celled, but cubical, and even squamous-celled neoplasms arise near the œsophageal orifice. The last type probably

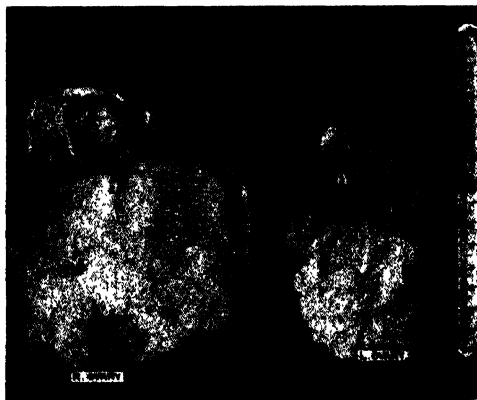


FIG. 991.—Krukenberg's tumours from a woman of thirty-seven. Scale in inches.

i. The '*new dyspepsia*' after forty—vague but *persistent* indigestion occurring in a patient who has never previously had 'stomach trouble'.

ii. *Insidious Onset*.—Especially in a man: he feels tired and weak, and the three A's may be in evidence—Anæmia, Anorexia, and Asthenia. Any patient with pernicious anæmia must be kept in regular review to watch for this. Although the majority have an iron-deficiency anæmia, a few may present with a macrocytic anæmia.

iii. The *obstructive* types—carcinoma of the cardia presents with dysphagia; carcinoma of the pylorus with fullness, belching, and then vomiting.

iv. *Lump*.—The incidental discovery of a lump in the epigastrium, no other symptoms being present, is sometimes the cause of the patient seeking advice. In about 30 per cent. of all cases a lump can be palpated.

v. *Silent*.—Carcinoma of the body of the stomach may be silent but give rise to features in other organs, such as obstructive jaundice due to secondary deposits in the liver, ascites from carcinomatosis of the peritoneum (p. 882), Krukenberg's tumours (p. 766), Trousseau's sign (phlebo-thrombosis of superficial veins of leg), or Troisier's sign (fig. 992).

One of the main obstacles to early diagnosis is that on an average it is between three and six months after the onset of symptoms that the patient visits his doctor. Even so, often a further three to six months sometimes elapse before the doctor sends the patient to hospital for gastric investigation.

INVESTIGATIONS IN A SUSPECTED CASE

If the diagnosis of this dread condition is to be made earlier, it is essential that attention is paid to the following investigations in *all* cases of indigestion.

Hæmoglobin—anæmia is present in 45 per cent. of cases.

Sedimentation rate—this is raised in 70 per cent. of cases.

Occult blood is present in the stools in 80 per cent. of cases.

Where necessary, repeated tests should be done. It is important to remember that a negative test does *not* exclude a neoplasm.

Radiology.—While a skilled radiologist attains a high degree of accuracy in the interpretation of a barium meal, small lesions are liable to escape notice, particularly if too much barium is given initially. Irregularities of the cardia or fundus are inclined to be over-looked unless the technique includes an examination in the inverted position. Flat growths may remain undetected in the early stages. Radio-diagnosis is accurate in 90 per cent. for pyloric growths, 75 per cent. for the cardia, and 60 per cent. for neoplasm of the body, but in only 47 per cent. of patients with conclusive radiological findings (filling defects) (fig. 993) is the neoplasm found to be resectable.



FIG. 992.—Troisier's sign found in the course of a routine examination for vague dyspepsia of recent origin. In this case there was a visible as well as a palpable mass of lymph nodes in the left supraclavicular fossa.

Gastroscopy undertaken by an expert gives the highest percentage of correct pre-operative diagnoses. If the tumour can be visualised, its characteristics are obvious. Its nodularity, the presence of several irregular

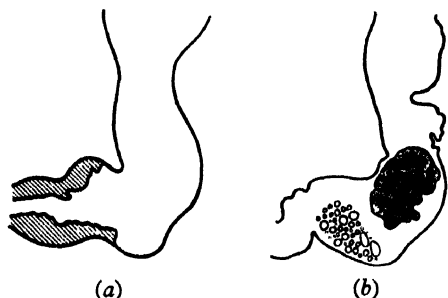
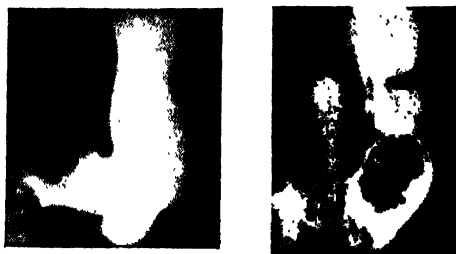


FIG. 993.—Barium-meal X-ray in carcinoma of (a) the antrum and (b) the body of the stomach.

superficial ulcers, the multi-coloured base, the immotility of the adjacent mucosa, all point to the nature of the lesion. If any doubt exists, the patient must remain at complete rest in bed (p. 746) for three weeks and then be re-gastroscooped. If there is no sign of healing the ulcer is almost certainly malignant. It must be remembered that there are two zones—the fundus and the roof of the antrum—which may not be visualised fully even by the most expert endoscopist.

Gastric Analysis.—The majority have either achlorhydria (p. 743) or hypochlorhydria, but occasionally some increase of acid is found. If there is more than 30 mEq. HCl in a post-histamine hour a carcinoma is unlikely.

Exfoliative Cytology.—Examination of cells found in the washings after gastric lavage has proved sufficiently reliable to confirm a diagnosis of carcinoma of the stomach in doubtful cases in about 70 per cent. of cases. Negative results are of no moment. Chymotrypsin lavage to soften the mucous lining has aided this method of investigation, and so has the use of tracer substances, such as tetracycline, which mark the malignant cells.

Laparotomy.—If there is still reasonable doubt after all these tests, it is wise to explore the abdomen.

COLLOID CARCINOMA

Macroscopically the stomach appears infiltrated in all its layers. Its walls are thickened and the individual layers cannot be differentiated. They are replaced by a kind of areolar tissue, the interspaces of which contain a transparent gelatinous substance. Although the gross appearance and the radiographs of colloid carcinoma resemble those of leather-bottle stomach, the microscopical picture is quite different, and very characteristic. Groups of cancer cells often line the accumulations of colloid. The tubular glands are extremely distended with this substance. Colloid carcinoma represents about 6 per cent. of the total cases of gastric carcinoma. It is this type of carcinoma which classically gives rise to the Krukenberg phenomenon (p. 766).

The treatment is the same as that for leather-bottle stomach and the prognosis is almost equally gloomy.

LEATHER-BOTTLE STOMACH (*syn.* LINITIS PLASTICA)

There is a generalised and a localised form of leather-bottle stomach.

When localised, it is the pyloric antrum that is mainly involved. The stomach wall is enormously thickened (fig. 994), and feels, as its name implies, like leather. Syphilis of the stomach may produce a similar change and a Wassermann reaction should always be done.

The enormous proliferation of fibrous tissue involves especially the submucosa, which often appears as a dense layer, mother-of-pearl in appearance. In contrast to colloid carcinoma, the mucosa, submucosa, and muscularis remain well differentiated, and, astonishingly, the whole of the mucous membrane looks and feels quite normal. Microscopically there is a tremendous overgrowth of fibrous tissue in the subserosa and submucosa, which sometimes spreads between the muscle fibres and strangulates them. The blood-vessels show evidence of endarteritis. It is often difficult or impossible to find any evidence of carcinoma, even in serial sections, but metastases are usually found in the regional lymph nodes. When distant metastases occur they are usually found in the liver or the ovary; on rare occasions bones are involved.



FIG. 995.—X-ray of a case of leather-bottle stomach.



FIG. 994.—Leather-bottle stomach, showing the enormous thickening of the stomach wall.

The symptoms are those of pyloric obstruction, but the small capacity of the stomach as revealed radiologically by a barium meal (fig. 995) makes the diagnosis tolerably certain.

Treatment.—In the localised variety, partial gastrectomy, and in the diffuse, total gastrectomy, offer hope of prolonging life, but the results, especially in the more common generalised variety, are among the worst, if not the worst, of all varieties of carcinoma of the stomach.

Boeck's sarcoid of the stomach occurs occasionally, and gives symptoms and X-ray findings identical with those of leather-bottle stomach.

TREATMENT OF CARCINOMA OF THE STOMACH

No description of the treatment of carcinoma of the stomach is comprehensible without a knowledge of its lymphatic drainage (fig. 996).

The lymphatic vessels of the stomach arise in its submucous and subperitoneal layers, and divide into four main sets that accompany corresponding blood-vessels. Each set drains both the anterior and the posterior surfaces of the stomach, and although they intercommunicate, their valves direct the lymph flow as follows :

The lymphatics of the proximal half of the stomach drain primarily into the left gastric and the splenic lymph nodes, and thence into the left, middle, and right superior pancreatic lymph nodes.

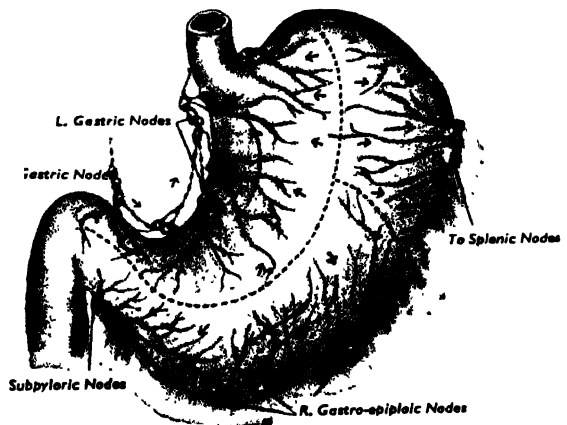


FIG. 996.—The lymphatic drainage of the stomach. (After Jamieson and Dobson.)

The lymphatics of the antrum drain into the right gastric lymph nodes superiorly and the right gastro-epiploic lymph nodes inferiorly.

The lymphatics of the pylorus drain into the right gastric (suprapyloric) superiorly and the subpyloric lymph nodes, situated around the gastro-duodenal artery, inferiorly. The efferent lymphatics from the suprapyloric lymph nodes converge on the para-aortic lymph nodes around the coeliac axis, while the efferent lymphatics from the sub-pyloric lymph nodes pass to the main superior mesenteric lymph nodes situated around the origin of the superior mesenteric artery. The lymphatic vessels related to the cardiac orifice of the stomach communicate freely with those of the œsophagus, but intercommunication between the lymphatic vessels of the pylorus with those of the duodenum is less definite.

Whether or not there is histological evidence of regional lymph node involvement makes a tremendous difference in the prognosis of *completely operable* cases of carcinoma of the stomach. It must be remembered that retrograde (or downward) spread may occur if the upper lymphatics are blocked.

Forty per cent. of patients without regional lymph node involvement survive five years or more, whereas only ten per cent. with regional lymph node involvement survive this length of time.

Prognosis.—Of recent years the extent of stomach resected has been on the increase without any coincident benefit. The important fact that has emerged is that, providing a reasonable amount of stomach is resected, it is the histology of the growth which determines the prognosis. The clinical counterpart of this is that the longer the history the better the outlook. Fifty per cent. of cases are inoperable when first seen; in the remainder exploration is worthwhile, resection being possible in half of them. Five per cent. of the initial cases, and ten per cent. of those having resection, survive for more than five years.

Operative Treatment :

Nomenclature.—A 'curative' resection consists of removing a block of tissue, including the growth, a margin of at least $1\frac{1}{2}$ inches (3.8 cm.) beyond its palpable limits with the stomach unstretched, and the related lymph nodes. The term radical gastrectomy should be reserved for a monoblock dissection fulfilling these requirements, thus bringing the term 'radical' into line with operations for carcinoma in other situations, e.g. the breast. Radical operations upon the stomach are of three varieties—(1) radical total gastrectomy, (2) radical upper gastrectomy, and (3) radical lower gastrectomy.

Incision.—Where possible an abdominal approach is to be preferred to an abdomino-thoracic (fig. 997). The latter carries an increased risk. The deciding factor for the use of an abdomino-thoracic incision is involvement of the gastro-œsophageal junction.



FIG. 997.—Abdomino-thoracic incision started as a left paramedian.

Operability.—As soon as the abdomen is opened, a firm plan, based on the extent of the growth, must be made. This decision is of profound importance and calls for considerable clinical judgement. There are three possibilities—radical surgery, palliative surgery, or the lesion may be inoperable.

Signs of Inoperability.—In general, these depend on the extent of the growth which include (1) fixation to posterior abdominal wall or involvement of the mesentery especially the origin of the superior mesenteric vessels, (2) gross local involvements of lymph nodes leading to fixity and evidence of retrograde spread downwards in the pre-aortic lymph nodes, (3) the presence of secondaries in the liver—an exception may be made in the case of a solitary resectable nodule, (4) peritoneal seedlings either locally or in the pelvis. A Krukenberg tumour does not of itself make the case inoperable.

Radical Operations

1. **Total gastrectomy**—indicated for growth involving the upper or middle two-thirds of the stomach. An upper left paramedian incision is made (fig. 997). The

operation is commenced by delivering the spleen and incising the peritoneum at its lateral border. The spleen with its hilar lymph nodes, the stomach, the splenic vessels, the tail and body of the pancreas are mobilised from left to right *en bloc*, the operation being essentially retroperitoneal. The left gastric artery and the splenic vessels are ligated retroperitoneally. The lymph nodes around the cardiac orifice are freed, and the lesser omentum is detached as far from the stomach as possible. Similarly, the lymph nodes beneath the first part of the duodenum are mobilised and the greater omentum is detached from the transverse colon. Performed in this way, the peritoneum over the tail and body of the pancreas are included in the block resection of the stomach with its lymphatic field. The cut edge of the pancreas is closed with sutures and a

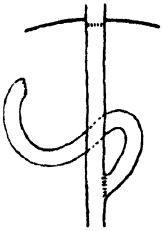


FIG. 998.—Total gastrectomy with œsophago-jejunostomy-en-Y (Roux's method).

soft rubber drain is passed down to this site, to be brought out through a separate stab incision. The stomach having been excised, the continuity of the alimentary canal can be restored in the manner shown in fig. 998; this procedure is valuable because it prevents regurgitation of bile and pancreatic juice into the œsophagus, which is so vulnerable to these digestive ferments. Even better is the Hunt method (fig. 999) which improves absorption to some extent. An œsophago-duodenostomy should never be done as an *alkaline* œsophagitis so frequently follows. If a thoraco-abdominal incision has been necessary, the diaphragm must be carefully repaired with non-absorbable sutures. Water-seal drainage of the pleural cavity is desirable.



FIG. 999.—Hunt's method of constructing a jejunal food pouch.

Post-operative Care of Total Gastrectomy.—Here nutritional disorders, e.g. weight loss, steatorrhœa, and macrocytic anæmia are the rule. The first two appear early but the anæmia takes five or six years to develop. These patients need frequent small meals of high protein, high caloric content, and regular vitamin B₁₂ supplements (100 to 200 mg. monthly).

2. Upper Radical Partial Gastrectomy.—Indicated for growth involving the cardia and upper third of the stomach. The procedure here is as for total gastrectomy, but the pyloric end of the stomach is preserved and anastomosed to the œsophagus. A pyloroplasty is usually done to prevent post-vagotomy retention (p. 749).

3. Lower Radical Partial Gastrectomy.—This operation for carcinoma of the pyloric end of the stomach involves separation of the greater omentum from the colon in its entirety, separation of the lesser omentum from the liver, freeing the subpyloric lymph nodes, and removal of the spleen and body and tail of the pancreas. The left gastric vessels and the right gastro-epiploic vessels are divided at their origin, stripping all fatty and lymphatic tissue around them towards the stomach. The operation is then completed by a Billroth I anastomosis.

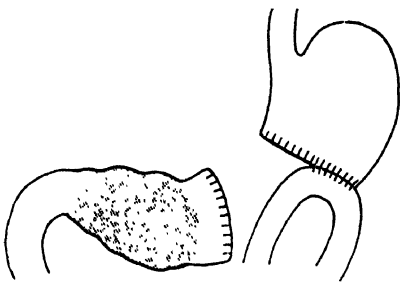


FIG. 1000.—Palliative exclusion operation for incurable pyloric carcinoma.

Palliative Operations.—These are frequently necessary to deal with or to anticipate specific symptoms such as obstructive vomiting, bleeding, or gastrogenous diarrhœa. Rapid dissemination and death may follow indiscriminate local resections and it is better to do nothing than for this to occur. The most valuable palliative operations are:

(1) **Palliative Gastro-jejunostomy.**—For irremovable pyloric carcinoma, gastro-jejunostomy should be performed some distance from the tumour, so that the advancing growth will not invade the stoma, at any rate for some time. This operation is symptomatically not so good as the—

(2) **Exclusion Operation.**—Excluding the growth from the pathway of the food is often beneficial, especially where an antral growth is fixed and inoperable and the upper part of the stomach is free. The risk of bleeding is reduced, appetite is increased, and vomiting ceases. The stomach is transected 1 inch (2.5 cm.) proximal to

the edge of the growth, the distal end is closed with non-absorbable sutures, and a gastro-jejunostomy is done (fig. 1000). If the pylorus is completely occluded, this operation is contraindicated.

(3) For *obstruction of the cardia* considerable relief can be obtained by the introduction of a plastic tube (p. 727). This is a most valuable manœuvre in those patients whose days are numbered. It is preferable to the palliative œsophago-jejunostomy (fig. 1001) which carries a definite operative mortality in these debilitated patients.

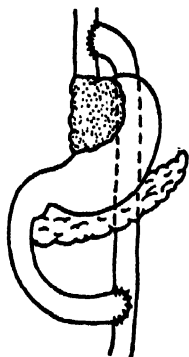


FIG. 1001.—Palliative retro-pancreatic œsophago-jejunostomy.

SARCOMA OF THE STOMACH

Sarcoma of the stomach accounts for about 1 per cent. of gastric neoplasms.

Lymphosarcoma is the most common variety. In contradistinction to lympho-sarcoma of the small bowel which is multiple, this tumour is usually solitary. It is impossible to distinguish it at operation from carcinoma of the stomach. In all inoperable cases a piece of the growth should be removed for histological examination. Although radio-sensitive, the best treatment, if circumscribed, is gastrectomy. This is a tumour in which chemotherapy may be useful.

Leiomyosarcoma occurs with equal frequency in men and women. The symptoms, clinical features, and X-ray findings are identical with those of leiomyoma (p. 765), but even more often than is the case with leiomyoma, the first symptom is a massive hæmatemesis and/or melæna. The presence of a barium-filled sinus extending into the tumour visualised by X-rays is extremely characteristic of leiomyosarcoma. Unlike a carcinoma of similar dimensions, the gastric acidity is unchanged. Usually these tumours metastasise late, and, when they do, secondary growths are often found in the liver. The treatment is partial gastrectomy. The tumour is more vascular than a carcinoma. It is stressed particularly that leiomyosarcomata should be attacked with vigour, regardless of the magnitude of the procedure, as the results are so much more gratifying than those of carcinoma of the stomach. At least 50 per cent. of the patients are alive five years after the operation.

Neurofibrosarcoma behaves precisely as a leiomyosarcoma, from which it is distinguished only by histological examination.

Fibrosarcoma, derived from the subserosa, is the rarest variety and the least malignant; it sometimes gives rise to a tumour of immense size, and in the female such a tumour is liable to be mistaken for an ovarian cyst.

DUODENAL NEOPLASMS

Neoplasms of the duodenum are exceedingly uncommon. Adenomatous polypi and adenomas have been reported from time to time as causes of severe melæna. Carcinoma of the first part of the duodenum, where ulceration is so common, is so rare that many of great experience have never seen a case. There is an apparent bar to spread of carcinoma of the pyloric end of the stomach into the duodenal mucosa, but there is no such obstacle to the spread in the subserous layer which, indeed, occurs with some frequency—hence the necessity for excising 1 inch (2.5 cm.) of the duodenum when performing radical lower partial gastrectomy. Carcinoma of the second part of the duodenum has been described, but these cases appear to arise in the ampulla of Vater (p. 846). Carcinoma of the third part of the duodenum occurs rarely, and the prognosis after local resection is favourable if the patient is seen sufficiently early to enable such an operation to be carried out.

CHAPTER 33

THE SPLEEN

Surgical Physiology.—The functions of the spleen are not wholly understood. In general they may be considered as:

1. **Destruction of effete and misshapen erythrocytes** and possibly of platelets. It is believed that during their sojourn in the spleen the lipoid coverings of ageing erythrocytes are rendered less strong. If red blood cells from a patient with hereditary spherocytosis are transfused into a normal person, they are destroyed in fifteen days. When transfused into a splenectomised person, they have a normal life span. After splenectomy the liver, bone marrow, and lymph nodes deal with effete red cells.

2. **Formation of erythrocytes and lymphocytes** during foetal life, infancy, and childhood. In case of need, this function may be resumed during adult life.

3. **Maintenance of a Reserve of Erythrocytes.**—Anoxia, caused by high altitudes, hæmorrhage, carbon monoxide poisoning, or prolonged exercise, stimulates the spleen to contract, thereby causing reserve red cells idling in its mesh-work to be conscripted into circulation as oxygen-carriers. This reaction may be useful in some animals like dogs, but the spleen is relatively too small to be of use in man (containing about 1 per cent. of the blood volume).

4. **Regulation of iron metabolism and iron storage.**

5. **Phagocytosis of foreign substances** by the reticulo-endothelial cells of the spleen is well illustrated by the high concentration of opaque substance in the spleen in patients who have received an injection of thorium. This function is also evident in the lipid dystrophies, when phagocytosis of abnormal lipoids is the main cause of splenic enlargement.

6. **Protection against Certain Toxins.**—It would appear that the spleen plays a part in the formation of antibodies. There is increasing evidence that splenectomised children are less able to combat severe infection than those with a normal spleen and they require close observation so that immediate and energetic steps can be taken in cases of pyogenic infection.

7. **Hormone Regulation of the Marrow.**—There is some evidence that the spleen secretes a hormone that inhibits the production of thrombocytes and leucocytes by the bone marrow. Dameshek considers excess of this hormone to be the cause of thrombocytopænic purpura and splenic neutropænia.

PHYSIOLOGICAL EFFECTS OF SPLENECTOMY

1. **Splenunculi** (accessory spleens), if present, **hypertrophy**.

2. **Bone Marrow Changes its Character.**—Within six months red marrow replaces yellow marrow in many of the long bones (Short). This accounts for fleeting bone pains 'like rheumatism' that are sometimes a matter of serious concern to the patient.

3. **Changes in the Blood.**—*Initial changes:*

- | | | |
|---|---|---|
| <ul style="list-style-type: none"> (a) Leucocytosis (b) Erythropenia (c) Diminution in hæmoglobin (d) Increased platelet count. (e) Increased coagulability. | } | <p>These changes reach their zenith between the second week and the second month after splenectomy.</p> |
|---|---|---|

A characteristic morphological change occurs in some of the erythrocytes, viz. the presence within them of Howell-Jolly bodies. These are rounded structures usually occurring singly, and placed eccentrically, that stain in the same way as nuclei. They are thought to be fragmented nuclei.

After two months lymphocytosis is present invariably. After many months there is moderate eosinophilia, and the mast cells increase in number.

William Dameshek, Contemporary. Haematologist, Mount Sinai Hospital, New York, U.S.A.

Arthur Rendle Short, 1880-1953. Professor of Surgery, University of Bristol.

William Henry Howell, 1860-1946. Professor of Physiology, Johns Hopkins University Medical School, Baltimore.

Justin Jolly, 1870-1953. Professor of Histophysiology, Collège de France, Paris.

4. **Undue Excretion of Iron by the Kidneys.**—Therefore the patient should take a preparation of iron.

SPLENUNCULI (syn. ACCESSORY SPLEENS)

Instead of a spleen, some fishes are provided with islets of splenic tissue scattered through the coelom. In man, accessory spleens, in common with the main organ, are derived from the dorsal mesogastrium. One or more are present in 25 per cent. of children (Jolly), but they are to be found in only 11 per cent. of adults (Adami). Obviously, then, as maturity is reached accessory spleens tend to disappear. Their usual locations are as follows:

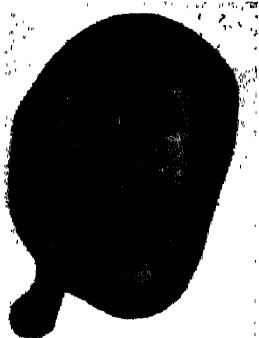


FIG. 1002.—Spleen with a splenunculus.

1. In contact with the spleen (fig. 1002), usually near the hilum; more than 50 per cent. are found in this situation.

2. Related to the splenic vessels and behind the tail and body of the pancreas—about 30 per cent.

3. The remainder are found in the splenic ligaments, the greater omentum, mesentery, mesocolon, and abutting on the left ovary or testis (carried thither during descent of the gonad).

When splenectomy has been performed for conditions that are known to be cured by removal of the spleen, return of symptoms after an interval of freedom is the result of hypertrophy of (usually) one accessory spleen. Occasionally a morsel of the spleen left attached to the pedicle during splenectomy continues to grow, and may be said to form a new spleen. Therefore, not only must the whole of the enlarged spleen be removed, but, except in cases of rupture, the sites of an accessory spleen

should be examined meticulously and any splenunculi present excised.

Splenosis is a rare condition, and nothing to do with splenunculi. Fragments of splenic tissue, spilled at the time of bygone rupture of the spleen, are found disseminated widely over the peritoneum, having thrived in situations other than those to be expected on developmental grounds. Sometimes there are scores of them. Splenosis does not give rise to symptoms, and is found only at laparotomy or necropsy. It is liable to be confused with endometriosis of the peritoneum, carcinomatosis or multiple angiomas.

SPECIAL METHODS OF INVESTIGATING THE SPLEEN

1. **Radiography.**—In plain films of the upper abdomen the spleen is visible in whole or part, especially when the stomach and the splenic flexure contain gas, and the patient is not obese. These findings, repeated at intervals, are good evidence that the spleen is unruptured (p. 776).

2. **Splenic Puncture.**—Provided the following stipulations are adhered to rigorously, splenic puncture can prove a most valuable diagnostic aid.

(a) Only definitely palpable spleens should be punctured.

(b) No patient with a hæmorrhagic tendency should undergo splenic puncture.

(c) The puncture should not be performed in the presence of infective splenomegaly, or of a tender spleen.

(d) Unconscious patients and children must never undergo splenic puncture.

Technique.—An ordinary lumbar puncture needle equipped with a guard to regulate the depth of the thrust should be employed. With the patient lying on his back, the site of puncture (fig. 1003) is infiltrated with local anæsthetic. The puncture must be performed during full inspiration, with the guard set to the required depth. Strong aspiration with the syringe is performed for several seconds. Before the needle is withdrawn the negative pressure must be released. After splenic puncture the patient is kept in bed for six hours, as a precaution against hæmorrhage.

3. **Splenoportography** can be carried out under general anæsthesia prior to laparotomy, or under local anæsthesia. The needle is inserted as above. After withdrawal of the stilette of the lumbar puncture needle, blood drips from the lumen; only after this has been demonstrated is the dye-filled syringe connected, and 30–50 ml. of hypaque injected rapidly. Apnœa, voluntary or controlled, must be

maintained during insertion of the needle and throughout the injection of the dye. If this stipulation is not adhered to rigorously, a laceration of the spleen as the spleen moves with the diaphragm, and possibly serious hæmorrhage, may result. Splenoportography permits visualisation of the spleen and the portal venous system (p. 812). The first exposure is made towards the end of the injection, and three or four exposures follow subsequently.

CONGENITAL ABNORMALITIES OF THE SPLEEN

Congenital absence of the spleen is exceedingly rare, and when it occurs it is nearly always associated with a gross congenital cardiac abnormality (usually a septal defect) and also the presence of accessory lobes to the lungs, and sometimes lobulation of the liver.

Movable Spleen (*syn.* Wandering Spleen).—The spleen, well supported by its two ligaments (the gastrosplenic and lienorenal), and resting on a third (the costocolic), which acts as a shelf, is the one abdominal organ that maintains its correct anatomical position in general visceroptosis. Movable spleen is a congenital abnormality that occurs more often in women. The condition is of great rarity, and the diagnosis is seldom made correctly.

Torsion of the splenic pedicle sometimes complicates the wandering organ. The torsion may be acute or chronic, acute cases presenting the symptoms of an intra-abdominal catastrophe. Chronic torsion may result in atrophy of the spleen, and after a period of indefinite abdominal discomfort all symptoms abate.

RUPTURE OF THE SPLEEN

Usually a ruptured spleen occurs as the result of an industrial or traffic accident of the crushing or run-over type. Blows on the abdomen or the left lower thorax and falls on to a projecting object are other sources of the violence. When rupture of the spleen occurs as a result of a traffic accident, associated injuries are commonly present. A number of cases of spontaneous rupture of a normal spleen have been reported, but in all probability in some of these there has been an accident that has been forgotten or suppressed by the patient.

Cases of ruptured spleen may be divided into three groups :

1. **The Patient Succumbs Rapidly, Never Rallying from the Initial Shock.**—This type is comparatively rare in temperate climates ; tearing of the splenic vessels and complete avulsion of the spleen from its pedicle¹ give rise to the symptoms which characterise this group. In countries where malaria is rife, splenic rupture is often rapidly fatal, and advantage was once taken of this knowledge by murderers in the Orient, who achieved their end by digging the victim beneath the left ribs with an implement known as a larang (fig. 1004).

2. **Initial shock ; recovery from shock ; signs of a ruptured spleen** is the usual type seen in surgical practice. After the initial shock has passed off, there are signs which point to an intra-abdominal disaster, and by correlating these signs it is often possible to arrive at a correct pre-operative diagnosis.

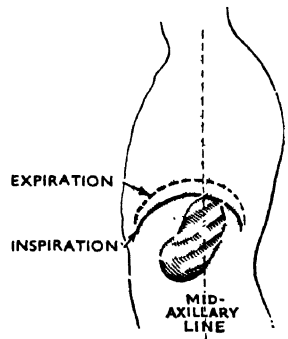


FIG. 1003.—The puncture is made in the 9th intercostal space in the mid-axillary line during full inspiration.

¹ Favoured by adhesion of the spleen to the diaphragm.



FIG. 1004. — A larang, which, being interpreted, means 'forbidden'. (After J. Johnston Abraham.)

General signs of internal hæmorrhage are inconstant. Perhaps the most helpful are increasing pallor, a rising pulse-rate, sighing respiration, and restlessness.

Local Signs.—(a) Abdominal rigidity is present in more than 50 per cent. of cases ; it is most pronounced in the left upper quadrant.

(b) Local tenderness is found constantly.

(c) Shifting dullness in the flanks is often present.

Ballance's sign is positive in about 25 per cent. of cases. There is a dull note in both flanks, but on the right side it can be made to shift, whereas on the left it is constant. The interpretation is that there is blood in the peritoneal cavity, but the blood in the neighbourhood of the lacerated spleen has coagulated.

(d) Abdominal distension commences about three hours after the accident, and is due to intestinal paresis, an early stage of paralytic ileus.

(e) Kehr's sign is pain referred to the left shoulder. There may be hyperæsthesia in this area. This sign is present very often, especially if sought a quarter of an hour after elevation of the foot of the bed, and is due to blood in contact with the under-surface of the diaphragm, the pain being mediated through afferent fibres in the phrenic nerve.

(f) Rectal examination frequently reveals tenderness and sometimes a soft swelling, due to blood or clot in the rectovesical pouch.

3. The Delayed Type of Case.—After the initial shock has passed off, the symptoms of a *serious* intra-abdominal catastrophe are postponed for a variable period up to fifteen days, or even more. As a rule it is only a matter of minutes to an hour or so, during which time the patient often appears to have recovered from the blow. Thus a Rugby footballer has continued to play after a short rest, only to collapse later from internal hæmorrhage.

Considerable delay of serious intraperitoneal bleeding is explained in one of the following ways :

(a) The greater omentum, performing its well-known constabulary duties, shuts off that portion of the general peritoneal cavity in the immediate vicinity of the bleeding (fig. 1124 (c), (p. 884)).

(b) A subcapsular hæmatoma forms and later bursts.

(c) Blood-clot temporarily sealing the rent becomes digested by escaping ferments from the lacerated tail of the pancreas. What may happen is that a patient with a suspected rupture of the spleen is taken into hospital; the symptoms abate, and in due course he is allowed up. Suddenly he collapses, perhaps in the lavatory while straining at stool. At other times fresh hæmorrhage is heralded by a rising pulse-rate, increasing pallor, advancing to air-hunger and collapse. Such disasters can, and should, be prevented by careful re-examination and radiography.

Radiography.—A normal, well-outlined spleen is a reliable negative sign, but radiographs should be repeated if the decision not to operate *pro tem* is taken. Indeed, every patient with a left-sided thoracic injury, especially where a lower rib is fractured, should be kept under radiological observation. When there is delayed rupture, or when the symptoms and signs are atypical, radiography often can help considerably in diagnosis. In decreasing frequency, the radiological signs of ruptured spleen are : (1) Obliteration of the splenic outline ; (2) Obliteration of the psoas shadow ; (3) Indentation of the left side of the stomach air-bubble ; (4) Fracture of one or more lower ribs

on the left side (present in 27 per cent. of cases); (5) Elevation of the left side of the diaphragm; (6) The presence of free fluid between gas-filled intestinal coils.

Treatment of Rupture of the Spleen.—Immediate laparotomy and splenectomy is the only reliable course. Blood is evacuated and, after injury to other viscera has been excluded, the abdomen closed completely. When the organ is damaged by a stab wound or missile penetrating the left pleural cavity, access to the spleen is best obtained by excising the thoracic and diaphragmatic wounds, and enlarging the opening in the diaphragm.

When circumstances permit, blood transfusions should be given as required. In the absence of stored blood, auto-transfusion is indicated.

Auto-transfusion.—Extravasated blood is collected from the peritoneal cavity by means of a sterile suction apparatus. After mixing it with 3·8 per cent. sodium citrate solution, 2 ounces (60 ml.) in 1 pint of blood (568 ml.), it is strained through several layers of sterile gauze and gravitated into a vein. We have carried out the procedure on numerous occasions, and never with regret.

Post-operative Complications.

Acute Dilatation of the Stomach.—This preventable complication should not be allowed to occur. Routine gastric aspiration for at least twenty-four hours post-operatively has banished it.

Hæmatemesis occasionally occurs due to transient congestion of the gastric mucosa following ligation of the vasa brevia veins.

Left basal atelectasis, sometimes accompanied by left pleural effusion, is particularly common, and is due to bruising of the diaphragm at the time of the accident, or to trauma during splenectomy. Breathing exercises and forced coughing go a long way in preventing atelectasis.

Paralytic ileus (p. 949) is rather common.

Peritoneal effusion or wound disruption may be secondary to concomitant injury of the tail of the pancreas. If there is any question of damage to the tail of the pancreas at operation, the area must be drained. It is wise, in any case, to use non-absorbable sutures to repair the abdominal wall.

'Rheumatism' due to bone marrow activity (p. 773).

Persistent hiccough is the result of irritation of branches of the left phrenic nerve on the under-surface of the left side of the diaphragm.

RUPTURE OF A MALARIAL SPLEEN

As has been mentioned, in tropical countries this is a frequent catastrophe. The delayed type of rupture (following a trivial injury) is also very common, and the patient is admitted with a perisplenic hæmatoma (fig. 1005). If splenectomy can be performed before the hæmatoma bursts into the general peritoneal cavity, the prognosis is less grave.

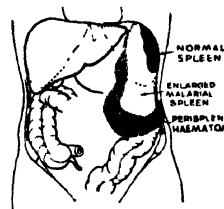


FIG. 1005.—Normal spleen, malarial spleen, perisplenic hæmatoma. Note that the splenic contour is lost when the capsule has ruptured.

The operation is considerably more difficult than in the case of a ruptured normal spleen. Surgeons with tropical experience have surmounted these difficulties by ligating the splenic vessels as they run along the superior border of the body of the pancreas (fig. 1006), before disturbing the hæmatoma (Andreasen).

ANEURYSM OF THE SPLENIC ARTERY

Perhaps due to its tortuosity or to its liability to become arteriosclerotic (frequently arteriosclerosis affects the splenic artery and no other), the splenic artery is more

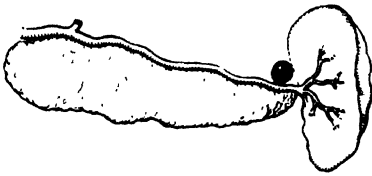


FIG. 1006.—Usual position of a splenic aneurysm. (After O. E. Owen.)

frequently the site of an aneurysm (fig. 1006) than any other artery in the abdomen, the abdominal aorta excepted.

As a rule the aneurysm is symptomless unless it ruptures. Exceptionally it gives rise to upper abdominal pain, in which event the condition has been diagnosed pre-operatively: (a) By a bruit over the left hypochondrium; (b) By radiography, concentric calcification being present in the walls of many splenic aneurysms of some standing. An unruptured splenic aneurysm

may be discovered at operation, usually upon the stomach.

Rupture of the aneurysm occurs somewhat more often in women than in men, and it is noteworthy that approximately 25 per cent. of the female patients are pregnant, and usually the pregnancy is advanced (six to eight months). In most instances rupture occurs into the peritoneal cavity, and the symptoms are precisely those of delayed rupture of the spleen (p. 776), although in relevant cases, ruptured ectopic gestation will receive prior diagnostic consideration. During the stage of perisplenic hæmatoma (as in spontaneous rupture of the spleen), bursting often occurs in two stages and either or both the diagnostic signs set out above are frequently present. Hæmorrhage into the stomach or the retroperitoneum has been recorded.

Treatment.—So dangerous is an aneurysm of the splenic artery that unless the patient is very old or infirm, excision of the sac should be advised in all cases. Rupture of the aneurysm demands urgent laparotomy and blood transfusion. The lesser sac must be opened and both the proximal and distal portions of the splenic artery are ligated. Proximal ligation alone is useless, for the distal end bleeds furiously. Splenectomy, although often performed, is not obligatory unless it is obvious that the arterial ligation has deprived the spleen of all its blood supply. Rupture of an aneurysm of the splenic artery carries a high mortality.

INFARCTION OF THE SPLEEN (Fig. 1007)

The patient, who is often suffering from subacute infective endocarditis or auricular fibrillation, is seized with agonising pain in the left hypochondrium. Symptoms usually mimic those of a perforated gastric ulcer, but the heart lesion helps the clinician to elucidate the diagnosis. When the embolus is aseptic the symptoms pass off in a few days. Infarct of the spleen has become a problem of air travel. At a height of over 10,000 feet patients with sickle-cell anæmia (a disease confined to Negroes) frequently sustain a splenic infarct.

In bacterial endocarditis, the infection may be maintained by an infected infarct or infarcts of the spleen. In a few instances splenectomy has proved to be the deciding factor in curing the patient of a condition that is almost assuredly fatal.

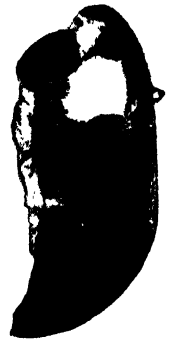


FIG. 1007.—Infarct of the spleen.

ENLARGEMENT OF THE SPLEEN

The spleen is a meeting-place of medicine and surgery. The following is a useful table of the causes of enlargement of the organ:

1. Infective	{	<i>Bacterial</i> —	{	Typhoid and Paratyphoid.
				Typhus.
				Anthrax.
				Tuberculosis.†
				Septicæmia.
	{	<i>Spirochætal</i> <i>Viral</i> —		Abscess of the spleen.‡
				Weil's disease.
				Syphilis.
				Psittacosis.
				{
	Egyptian splenomegaly.†			
	Kala-azar.			
	{	<i>Parasitic</i> —	Hydatid cyst.*	

3. *Hydatid Cyst of the Spleen*.—Cases of hydatid cyst have been reported. A cyst of the spleen should be treated by splenectomy.

HÆMOLYTIC ANÆMIA

This disorder, which is due to a defect in the erythrocytes, presents in two forms: (1) congenital (hereditary spherocytosis) and (2) acquired.

1. **Hereditary Spherocytosis** (*syn.* Congenital Hæmolytic Anæmia; Acholuric Familial Jaundice).—Because of the existence of cases demonstrating neither anæmia nor jaundice, the designation hereditary spherocytosis is to be preferred. The defect can be transmitted by either parent as a Mendelian¹ dominant, with the result that males and females are affected equally.

Acholuric jaundice is jaundice without bile in the urine.² Spherocytosis

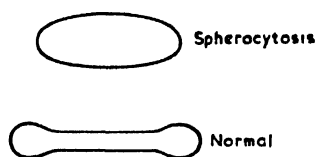


FIG. 1009. — Diagrammatic representation of normal (biconcave) and biconvex red cells of spherocytosis.

is a term that gives a clue to the underlying pathology. The erythrocytes are smaller than normal, and instead of being biconcave, are biconvex. Not all of them are so shaped, but a proportion are, according to the severity of the condition. Biconvex (spheroidal) red cells, or any red cells with an inadequate cell envelope, are destroyed by the spleen.³ Whereas the life of a normal erythrocyte is about one hundred

days, that of a red cell in a case of hereditary spherocytosis is about fourteen days. The spleen is enlarged because it is constantly destroying excessive numbers of red cells.

Clinical Features.—Once the disease manifests itself, spontaneous remissions are almost unknown. As a rule the patient is pale and has jaundice, which varies in intensity and at its height is of a daffodil hue. Careful enquiry must be made into similar disease in other members of the family. In established cases lassitude and undue fatigue are seldom absent, but they vary with the amount of hæmolysis that is proceeding.

Sometimes the patient is born jaundiced, or becomes so early in life. In certain families the disease is characterised by severe crises of red blood-cell destruction; thus, with the onset of a crisis, an erythrocyte count may fall from $4\frac{1}{2}$ millions to $1\frac{1}{2}$ millions in less than a week. Such crises are characterised by the sudden onset of pyrexia, abdominal pain, nausea, vomiting, and extreme pallor, followed by increased jaundice. These crises may be so severe as to cause death in infancy or childhood. They may be precipitated by acute infections. More usually the jaundice, although variable, is very mild, and may not appear until adolescent or even adult life. In adult cases there is often a history of attacks of gall-stone colic; indeed, 68 per cent. of untreated patients over the age of ten years have pigment stones in the

¹ In 1865 Mendel described 'dominant' and 'recessive' traits in hybrids. His work passed unnoticed for thirty-five years.

² Although there is excessive breakdown of red cells with transformation of liberated hæmoglobin to bilirubin, the bilirubin compound so produced is excreted by the liver and not by the kidneys.

³ 'The spleen dislikes, distrusts, and devours spheroidal red cells' (A. Rendle Short).

gall-bladder. Every child with gall-stones should be investigated for evidence of hereditary spherocytosis.

On examination the spleen is large, and in thin subjects it can be palpated easily. Sometimes the liver is also palpable. Chronic ulcers of the legs are a common occurrence in adult sufferers.

Hæmatological Investigations.

The Fragility Test.—Increased fragility of erythrocytes characterises this disease. Normally, erythrocytes begin to hæmolyse in 0.47 per cent. saline solution. In this condition hæmolysis occurs in 0.6 per cent., or even in stronger solutions.

The Reticulocyte Count.—To compensate for the loss of erythrocytes by hæmolysis, the bone marrow discharges into the circulation immature red cells, which differ from the adult cells by possessing a reticulum. This cannot be seen in the usual blood films, but can be demonstrated readily by vital stains. Crises are associated with reticulocytopenia, and hypoplasia of the erythroid element of the bone marrow. After a crisis the reticulocyte count is increased very much.

Fæcal urobilinogen is increased, as most of the urobilinogen is excreted by this route. Although technically difficult, this measurement is the best means of determining the extent of hæmolysis in cases of hereditary spherocytosis.

Use of Radioactive Chromium.—Labelling of the patient's own red cells with ^{51}Cr followed by daily scanning over the spleen will show the degree of red cell sequestration by the spleen. If splenic radioactivity is high, splenectomy will be of value.

Treatment.—All patients who have hereditary spherocytosis should be treated by splenectomy. Not, of course, that the spleen is at fault. The trouble lies with the red cells, but it is the spleen which will not let them survive sufficiently long to be of much service. After splenectomy, the spheroidal cells have a normal life span.

In the great majority of cases splenectomy can be undertaken as an elective operation during a remission. In juvenile cases the age at which operation is recommended has been decreasing. If it is not imperative before, between the third and fourth years seems the optimum time, i.e. before gall-stones have had time to form.

At operation (splenectomy) the gall-bladder should be palpated for gall-stones, and if they are present cholecystectomy can be undertaken then or at a later date, depending on the patient's fitness.

Following splenectomy, the patient soon recovers from the anæmia, which does not return. The jaundice disappears, but the tendency to hæmolysis persists; it has been demonstrated twenty-five years after removal of the spleen. Ulcers of the leg due to this disease heal rapidly. In short (ruptured spleen excepted), in no other condition is splenectomy more triumphant. Owing to the absence of adhesions, the operation can be undertaken easily and expeditiously, which in part accounts for the very low operative mortality.

When a patient is so anæmic as to render blood transfusion advisable, more than ordinary precautions are required. Coombs' test (p. 74) must always be performed—in hereditary spherocytosis it is negative (cf. acquired hæmolytic anæmia). While correctly-matched blood is usually well tolerated and the transfused red cells survive in patients whose disease is in remission, transfusion increases the amount of hæmolysis during a crisis. In such circumstances whole blood should be eschewed and a cautious transfusion of packed red cells substituted. As a rule packed red cells are accepted. Should a reaction occur, however, the risks of splenectomy are probably less than risking a severe hæmolytic reaction induced by unacceptable red cells.

below the costal margin. Pronounced splenic enlargement makes the diagnosis of thrombocytopenic purpura highly improbable.

The counting of platelets is technically difficult, and the reading is vitiated by profound hæmorrhage, which renders the count relatively high. The count is best made after spreading the blood upon a cover slip spread previously with brilliant cresyl blue. The stained platelets can be identified easily.

Sternal Puncture.—The bone marrow shows megalokaryocytosis¹ with absent platelet budding.

The behaviour of the disease in adults is dissimilar from that in children.

In Children.—After one acute episode consisting of cutaneous purpura, often accompanied by epistaxis and bleeding from the oral mucous membrane, the disease undergoes a spontaneous cure in 75 per cent. of cases.

In Adults.—Although the initial attack is seldom as severe as in children, the disease tends to become cyclic, and often the relapses are of increasing severity, uterine or gastro-intestinal hæmorrhages being much in evidence. This difference has an important bearing on treatment.

Treatment :

In Children.—As intracranial hæmorrhage is exceptional, and for reasons given above, a conservative policy can be followed and, if necessary, continued for three months. No active treatment is given if the symptoms are mild, but should the bleeding be severe or persistent, in addition to maintaining a safe hæmoglobin level, a course of ACTH or cortisone often effectively controls the hæmorrhagic manifestations. Such a course lasts two or three weeks. Splenectomy is advised in (1) severe cases ; (2) when relapses occur ; (3) especially in girls approaching the menarche. At least two days' steroid therapy should be given prior to operation. The results of splenectomy are usually satisfactory, in spite of the fact that after an initial rise, the low platelet count persists. If, after splenectomy, relapses occur, an overlooked accessory spleen should be suspected.

In Adults.—Steroid therapy, if necessary combined with blood transfusion, should be employed in preparation for splenectomy.

Blood transfusion improves the hæmostatic state for a few hours without necessarily increasing the platelet count. The volume of any one transfusion should not exceed 1 litre, because massive and rapid transfusions often aggravate the thrombocytopenia and the bleeding tendency. For the same reasons repeated transfusions are also contraindicated. Fresh blood is essential, and vein-to-vein transfusion with siliconed syringes and tubes is undoubtedly the best method of blood transfusion in this instance. Some centres hold stocks of platelet concentrates, and transfusion of platelets, repeated as necessary, is a procedure of great promise.

Cortisone Therapy.—Unless the patient has already lost much blood, cortisone therapy frequently controls the hæmorrhage and renders blood transfusion unnecessary. Its mode of action appears to be that of making the capillaries less permeable,

¹ Megalokaryocytes (*syn.* megakaryocytes), the giant cells of bone marrow, give origin to blood-platelets.

it also results in a reduction of the time of clot retraction, but without a change in the platelet count.

Initially 300 mg. of cortisone is given by mouth in divided doses during the first twenty-four hours, 200 mg. during the second twenty-four hours and 100 to 150 mg. daily thereafter for two or three weeks. In preparing the patient for operation the dose of cortisone is decreased in the immediate five days prior to the operation, substituting an equivalent amount of ACTH. No cortisone is given two days pre-operatively, but it is given immediately post-operatively, and continued for about a week in decreasing doses.

Splenectomy.—In slightly more than half the cases the bleeding tendency is controlled by the above measures, the operation can be converted from an emergency procedure to one of election which, being unhurried, permits a thorough search for accessory spleens.

In the remainder, operation must be undertaken with as little delay as possible. Following incision into and through the parietes there is profuse and sometimes frightening bleeding. This must be ignored until the splenic artery is reached and clamped. After waiting for twenty minutes or more, clotting will proceed more or less normally and the operation can be completed.

Splenectomy in primary thrombocytopænic purpura is followed by good results in over 80 per cent. of cases, in spite of little change in the platelet count after an initial rise lasting about a week.

Treatment of Secondary Purpura.—Obviously, if the cause is known and can be removed, considerable faith can be placed in conservative measures. Cortisone and ACTH are effective in producing a remission in most instances. Up to a few years ago it was thought that splenectomy was contraindicated in secondary purpura, but a number of cases of cure by splenectomy have been reported. Particularly encouraging have been cases where the purpura is due to splenic tuberculosis and in rare examples of Boeck's sarcoid. If the bleeding is so profuse or prolonged as to tax the resources of the blood bank, splenectomy should not be delayed (Lowdon). In most instances, however, with steroid therapy it is safe to wait for a remission. Half of the cases are benefited by splenectomy.

MEDITERRANEAN ANÆMIA (*syn.* COOLEY'S ANÆMIA; THALASSEMIA¹)

Cooley's anæmia is a hereditary disease due to faulty synthesis of hæmoglobin.

Originally thought to be confined to those who dwelt near the Mediterranean Sea, or who had a Mediterranean heritage, it is now known that this is only partially true, for scattered reports from various parts of the world show that the disease occurs from time to time in families who have no such heritage; for instance, a number of cases have been reported in natives of Thailand. The condition is inherited. In a severely affected infant both parents show characteristic blood changes. While the mild form is asymptomatic and can be detected only by an examination of blood, severe forms should be suspected by the following characteristics:

Clinical Features.—The patient is of short stature and the facies are reminiscent of a congenital syphilitic; the skull is large and bossed, and the bridge of the nose is depressed (fig. 275, p. 235). In addition the maxillæ are prominent, also the upper front teeth. There is a mongoloid slant of the eyes. The complexion is muddy; the scleræ have an icteric tinge. Especially in a young child, the abdomen is prominent because of an enlarged spleen and liver. In patients with moderate anæmia these signs are correspondingly less, and in mild cases all are absent.

¹ Thalassæmia—Greek, Thalassa = Sea. (Because it occurs in persons of Mediterranean stock.)

Andrew Gilchrist Ross Lowdon, 1911–1960. Professor of Surgery, University of Newcastle-upon-Tyne.
Thomas Benson Cooley, 1871–1946. Professor of Paediatrics, Wayne University College of Medicine, Detroit.

Blood Examination.—Increased resistance of red cells to hæmolysis in hypotonic saline solution is *the* characteristic finding. It is always present in Mediterranean anæmia, irrespective of the severity or mildness of the disease. In the more severe forms, not only is there a diminished number of erythrocytes and a low hæmoglobin level, but nucleated red cells and other immature blood cells abound.

Radiography of the skeleton shows early rarefaction of all bones and late sclerosis of tubular bones.

Treatment.—In selected cases splenectomy reduces the number of transfusions required.

PORPHYRIA, WITH SPECIAL REFERENCE TO THE ABDOMINAL CRISES OF THE DISEASE

Porphyria is an hereditary error of katabolism of hæmoglobin in which porphyrinuria occurs. The abdominal crises, which are characterised by violent intestinal colic with constipation, are liable to be precipitated by the administration of barbiturates, to which these patients have an idiosyncrasy. The patient is anæmic, frequently suffers from photosensitivity, and in advanced stages of the disease neurological or mental symptoms (from damage to the brain) are often present. On examination the spleen will be found to be enlarged. On a number of occasions the splenic enlargement, which is usually well marked, has been overlooked and *the abdomen has been opened on the diagnosis of intestinal or appendicular colic*, with negative findings. Another manifestation of acute porphyria is spasmodic abdominal pain followed by jaundice. This is due to spasm of the common bile-duct and Oddi's sphincter.

Two methods of establishing the diagnosis are available :

The urine is sometimes normal in colour. Usually it is orange (which is often dismissed as 'concentrated'). If the specimen is left exposed to daylight for a few hours it develops a port-wine colour, particularly near the surface, where it is exposed to the air. There are several conclusive laboratory tests for porphyrinuria.

Radiography of the abdomen. Serial X-ray films show areas of intestinal spasm causing short segments of gaseous dilatation of the small and large intestine, especially of the cæcum.

Treatment.—Often there is a striking decrease in the serum sodium level and the patient is improved considerably by infusion of normal saline solution with careful control of electrolytic balance. To relieve the abdominal pain pethidine is the best drug. If a sedative is required, paraldehyde should be given. Splenectomy is not of value.

ENDEMIC HEPATO-SPLENOMEGALY (*syn.* EGYPTIAN SPLENOMEGALY)

Although especially common amongst the fellaheen (peasants) who dwell in the delta of the Nile, this disease is by no means confined to Egypt. It is prevalent in many parts of Africa, and is met with in Asia and South America, particularly Venezuela.

Ætiology.—The consensus of opinion is that in 75 per cent. of cases the disease is due to infestation by the *Schistosoma mansoni*, and the remainder by *Schistosoma hæmatobium*. In some parts of the world chronic malaria appears to be the cause, e.g. Bengal Splenomegaly (Basu).

Pathology.—A diffuse periportal fibrosis of the liver (fig. 1011) is the first manifestation. Bilharzia ova in various stages of degeneration can be found in the portal vein and its tributaries. Next, enlargement of the spleen occurs, due, it is believed, to bilharzial toxins and the products of disintegrated worms and ova, all of which are filtered by the spleen, causing active hyperæmia and hyperplasia of its reticulo-endothelial elements. The splenic capsule becomes thickened, due to perisplenitis, and the splenic vessels are often tortuous and sclerotic. Diminished splenic contractility causes stagnation of blood in the spleen, and results in its further enlargement. About this time the enlarged liver commences to contract, and the degree of enlargement of the spleen is roughly proportional to the degree of fibrosis of the liver (Halawani). Advanced cases are accompanied by ascites.

Ruggero Oddi, 1845-1906. Surgeon and Anatomist, Rome.

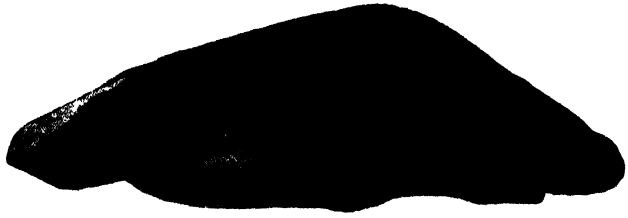
Sir Patrick Manson, 1844-1922. Practised in Hong Kong; later Physician to the Dreadnought Hospital, Greenwich.

Ajit Kumar Basu, Contemporary. Director-Professor, Department of Surgery, Institute of Post-Graduate Medical Education and Research, Calcutta, India.

Theodor Maximilian Bilharz, 1826-1862. Professor of Zoology, Cairo.

Ahmed Halawani, Contemporary. Director of the Institute of Tropical Medicine, Cairo.

FIG. 1011.—Periportal fibrosis of the liver in Egyptian splenomegaly. (Dr. Halawani, Cairo.)



Clinical Features.—Egyptian splenomegaly is encountered at all ages from ten to seventy years, frequently reaching its zenith in the fourth decade. Ninety per cent. of the patients are males.

1st Stage.—There is gradual enlargement of the spleen, hypochromic anæmia, eosinophilia, leucopenia and a relative lymphocytosis. The liver becomes moderately enlarged and is tender in 70 per cent. of cases. During this stage there is little or no disturbance of general health.



FIG. 1012.—Egyptian splenomegaly. Successful splenectomy was performed. (Owen Richards, F.R.C.S.)

2nd Stage.—The spleen becomes further enlarged, extending to the pelvic brim (fig. 1012). The liver gradually shrinks. The major symptoms at this time are :

Splenic pain or 'heaviness'	90 per cent.
Weakness	78 per cent.
Spasmodic abdominal pain	75 per cent.
Diarrhœa (often blood-stained)	60 per cent.
Hæmaturia	38 per cent.
Hæmatemesis	4 per cent.

The patient suffers from lassitude and may fall a victim to intercurrent infection.

3rd Stage.—The liver atrophies more and more. Ascites supervenes. The superficial abdominal veins sometimes become dilated and tortuous. The general health deteriorates, and usually the patient dies in six to twelve months from the appearance of ascites. As a rule it takes eight to twelve years to pass from the first to the final stage.

Laboratory Findings.

The urine and the fæces are examined for ova.

Liver function tests reveal a varying degree of hepatic impairment. A hypochromic anæmia is always present.

Treatment.—Many early cases respond to the general treatment of *bilharzia mansoni* detailed on p. 1003. In more established cases no substantial reduction in the size of the spleen results from a six-weeks' course of this treatment. It is in the latter class that, after careful pre-operative preparation, splenectomy with pre- and post-operative blood transfusions is undertaken under penicillin cover, and is often curative. The consensus of opinion in Egypt is that splenectomy should be performed in cases where there is no evidence of ascites, liver damage or severe renal insufficiency.

SPLENECTOMY

The common indications for splenectomy in this country are trauma, either following an accident or at operation (e.g. gastrectomy), thrombocytopenic purpura, acholuric jaundice, or preparatory to spleno-renal anastomosis.

Other indications for splenectomy are tabulated on pp. 778 and 779.

Technique of Splenectomy.—Most surgeons use a left paramedian or left subcostal incision. For large spleens adherent to the diaphragm a thoraco-abdominal incision may be necessary. Before operation, however, the passage of a naso-gastric tube enables the stomach to be emptied. This facilitates the operation and prevents the post-operative complications of dilatation of the stomach and paralytic ileus.

If the operation is for *traumatic rupture*, a quick mobilisation is necessary. The hand is passed round the concave margin, the lienorenal ligament (fig. 1015) divided largely by blunt dissection and the spleen rotated medially into the incision. A large

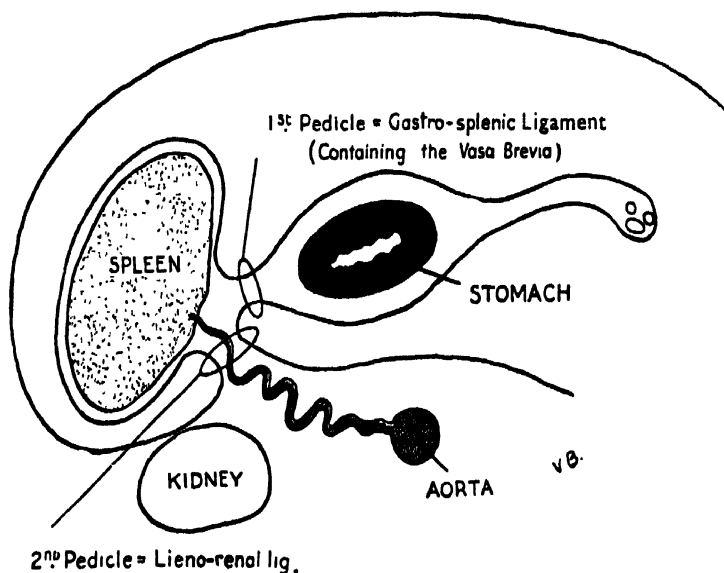


FIG. 1015.—From the surgical standpoint the spleen may be said to have two pedicles—the gastro-splenic ligament and the lienorenal ligament. The splenic artery and vein lie in the latter.

pack is inserted and the short gastric vessels and those in the pedicle are ligated and divided. It is important to separate the tail of the pancreas from the vessels in the hilum before ligation.

For *hypersplenism and other conditions* requiring splenectomy.—The first step is to open the gastro-splenic ligament and divide the short gastric vessels (fig. 1015). The splenic artery at the superior border of the pancreas is then under-run with silk and ligated. The posterior surface of the spleen is exposed, the lienorenal ligament divided by long curved scissors, and the spleen rotated medially, together with the tail and body of the pancreas. The pancreas is separated from the hilum and the vessels dissected out, ligated, and divided. Careful search must be made for accessory spleens. It is wise to drain the wound in case damage to the pancreatic tail has occurred.

Unexplained abdominal pain and fever in the post-operative period may herald thrombosis of the portal vein, when anticoagulants and antibiotics must be given.

CHAPTER 34

THE LIVER

Surgical Anatomy.—The dual afferent blood supply, consisting of the hepatic artery and the portal vein, singles out the liver from all other organs. That the portal vein has no valves is a basic fact in the understanding of portal hypertension. Both the portal and the hepatic veins have well-defined coats of unstriated muscle which together with the hepatic artery control the blood flow through the liver. In shock, active constriction of the hepatic veins takes place through reflex action. This produces congestion and some degree of stagnant anoxia that, if severe and prolonged, results in hepatic centrilobular degeneration.

The blood in the sinusoids is derived from two sources:

1. The portal system conveys between 60 and 80 per cent. of the afferent blood to the liver, together with the products of digestion from the alimentary canal and the internal secretions, insulin and glucagon, from the pancreas. No less than one-fifth of the portal blood comes from the spleen.

2. The hepatic artery transmits oxygen to the liver and, by a delicate pressure arrangement with the hepatic vein, determines the blood flow through the liver lobules. The pressure relationships of the hepatic artery, the portal vein and the hepatic vein testify further as to the unique nature of the circulation through the liver (fig. 1016). During exercise the blood flow through the liver, which is normally about 20 per cent. of the cardiac output, falls sharply in favour of other vital organs and the muscles.

Surgical Lobes of the Liver (fig. 1017):

The right and left branches of the hepatic artery do not supply the two main anatomical lobes as such. Their territory has a boundary running along the gall-bladder bed

and thence upwards over the anterior surface to the inferior vena cava, down this vessel to return to the porta hepatis.

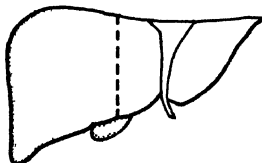


FIG. 1017.—Surgical lobes of the liver.

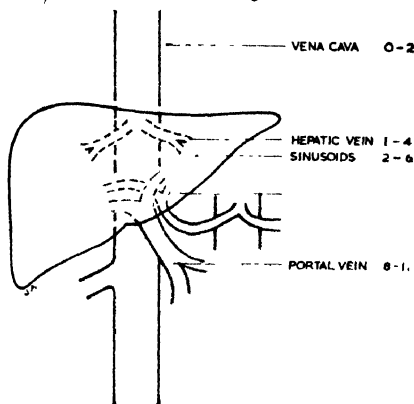


FIG. 1016.—Pressure (in mm. Hg.) in various components of the hepatic circulation.

Functions of the Liver:

1. The secretion of bile.
2. The formation of albumin, fibrinogen, and prothrombin.
3. Storage and metabolism of carbohydrates, including the conversion of monosaccharides (e.g. dextrose) into glycogen, and vice versa.
4. Formation of phospholipids and cholesterol, synthesis of fatty acids from carbohydrate, and other steps in fat metabolism.
5. Deamination of amino-acids with formation of urea. Removal of ammonia from portal blood.
6. Heat production.
7. Reticulo-endothelial activities.
8. Storage of vitamin B₁₂ (cyanocobalamin) and vitamin A.
9. Iron and copper storage.
10. Destruction of bacteria. Bacteria, especially Gram-positive cocci, have been

found repeatedly in samples of portal blood taken at laparotomy for non-infected conditions. It is therefore believed that the liver destroys many bacteria that gain entrance to the body through the portal tributaries.

11. Detoxication of drugs and hormones, e.g. short-acting barbiturates; oestrogens.

Unlike other organs, the liver possesses a remarkable ability to replace lost tissue rapidly and completely by compensatory cellular hypertrophy and hyperplasia. This permits wide resection for a localised hepatic neoplasm, and removal of large portions of doubtfully viable liver tissue in cases of severe trauma.

LIVER INSUFFICIENCY—HEPATIC COMA

The most prominent features are due to the failure of the detoxicating functions of the liver, so that these toxic substances reach other parts of the body. Liver failure in a patient who already has a diseased liver may be precipitated by intercurrent infection, a surgical operation (including paracentesis), the administration of a general anaesthetic, or even narcotics.

Clinical Features.—Fœtor hepatis, a sweetish musty odour, is frequently noticeable; sometimes it is so strong as to fill a room. Lethargy and disorientation are prodromal signs of hepatic coma; convulsions occasionally occur. Jaundice is usually present.

Treatment:

Adequate fluid and electrolyte balance must be obtained. If the patient can swallow, a high carbohydrate diet should be maintained. If the patient is unconscious, this may be given by a naso-gastric tube, but intravenous administration of glucose is sometimes necessary. Frequently potassium needs supplementation.

Hæmodialysis will help to remove toxic products from the blood and the results of the use of an artificial kidney or perfusion through a pig's liver are encouraging.

Prognosis.—This depends entirely on the functional capacity of the liver. When coma is due to liver-cell failure the mortality is very high.

PORTA-SYSTEMIC ENCEPHALOPATHY

This syndrome occurs when much blood is by-passing the liver, as in cirrhosis or when a porta-systemic venous shunt has been created. It is due to the passage of protein breakdown products, of which ammonia is one, from the alimentary canal into the systemic circulation. These products are normally metabolised in the liver, and the syndrome rarely occurs unless the liver function is impaired. It thus tends to be precipitated by a large protein meal or an upper gastro-intestinal hæmorrhage. In individual patients it is often difficult to determine to what extent the symptoms are due to this cause or to liver-cell damage. In patients who have had symptoms for some years, organic changes have been found in the cerebral cortex or in the spinal cord.

Clinical Features.—It is characterised by disorientation and a flapping tremor of the outstretched hands. Cogwheel rigidity of the limbs may be elicited, and the patient may pass rapidly into deep coma. Sometimes he may recover equally rapidly when the source of the toxic products is removed from the alimentary canal.

Treatment.—1. The same general steps must be taken as have been described for hepatic coma.

2. If there has been bleeding into the alimentary tract, the blood must be removed by all means available, especially by colonic lavage. Neomycin should be given by mouth or by gastric tube to prevent enzymic activity of the intestinal flora.

3. If the patient is likely to remain unconscious for any length of time, tracheostomy reduces the risk of inhalation of saliva or gastric contents.

4. If repeated attacks occur, sub-total colectomy or a colon by-pass operation may be advisable in order to remove the source of the toxic products.

Prognosis.—Many patients with cirrhosis who pass into coma as a result of hæmorrhage into the gastro-intestinal tract recover with appropriate treatment, and later operative measures may be undertaken to reduce the risk of further bleeding.

SPECIAL METHODS OF INVESTIGATING THE LIVER

Liver Function Tests.—Owing to its manifold functions, there is no single test by which the liver can be stated to be functioning normally. Therefore several tests are usually undertaken in each patient, and in some instances the individual test must be repeated. No less than 80 per cent. of the liver can be out of action without affecting individual tests, and in patients with cirrhosis they may all be within normal limits. Among a large number of tests available the following are the most useful:

1. **Serum Bilirubin Estimation.**—A value of conjugated bilirubin exceeding 0.2 mg./100 ml. is one of the most valuable indices of either stasis within the biliary tree or hepato-cellular damage, but it is of no value in distinguishing the one from the other.

2. **Serum alkaline phosphatase** (normal value 3 to 13 King-Armstrong (K-A) or 1.5-4 Bodansky units). Values below 30 K-A favour hepatogenous jaundice; those above 35, obstructive jaundice.

3. **Estimation of serum albumin** is a good general test of liver function, as it is the only site of production. A level below 2.0 G. per 100 ml. indicates that liver function is greatly impaired and, if an operation is essential, only the minimum should be done. Above 3.0 G. per 100 ml. is satisfactory.

4. **Serum Transaminase.**—This is an enzyme present in liver cells and heart muscle. Increased values (over 100 units) are found in hepato-cellular disease but not in obstructive jaundice.

5. **Plasma prothrombin Index**, if *low*, is an indication for pre-operative vitamin K therapy. Where there is little or no response to vitamin K, extensive hepato-cellular damage is almost certain. Owing to large reserves a satisfactory response does not exclude considerable liver damage.

6. **Bromsulphalein Test.**—The value of this test is diminished in the presence of jaundice, not only because of the technical difficulty of estimating the dye in the presence of bile pigments but also, more importantly, because either intra- or extra-hepatic biliary obstruction may cause increased retention of the dye irrespective of hepato-cellular damage. In the absence of jaundice, it is a most sensitive test of even slight derangement of hepatic function. After an intravenous injection of the dye, not more than 30 per cent. of it should be present in the serum at the end of an hour. An increase in this amount is indicative of liver damage.

7. **Scanning of Radio-active Isotopes.**—This may be useful in localising lesions in the liver. Rose Bengal, labelled with ^{131}I , is used as it is a gamma-ray emitter and is taken up by the liver cells, so that lesions which do not contain functioning liver cells, such as abscesses, cysts, metastatic and most primary tumours appear as defects on the hepatic photoscan. Radio-active gold or iodinated human albumin are taken up by the reticulo-endothelial cells and may be used in a similar way.

Liver biopsy has a limited field of usefulness. It should never be attempted until the presence or absence of a bleeding tendency on the part of the patient has been ascertained by all accepted tests, and if present, remedied. The puncture is carried out on the right side or in the epigastrium. The Menghini needle (fig. 1018) is the best. Sections are cut from the cylindrical specimen obtained. Hæmorrhage or biliary peritonitis are rare complications.

Angiography by means of coeliac axis or superior mesenteric arteriograms is occasionally valuable in detecting changes in the vascularity of the liver, either due to vascular obstruction or to an avascular lesion such as a cyst or abscess, or to a vascular tumour. In cases of portal hypertension films in the venous phase of superior mesenteric angiography are useful if the spleen has been removed and splenic injection is thus impossible.

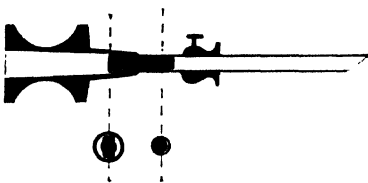


FIG. 1018.—Menghini 'one-second' liver biopsy needle. The nail in the cannula allows fluid to pass but prevents the biopsy specimen from being fragmented by violent aspiration into the syringe. (*From Gastroenterology.*)

INJURIES

Rupture of the liver, when extensive, is an extremely grave accident; moreover, in a considerable proportion of cases it is associated with other serious injuries, and consequently the mortality is high. The violence that produces this injury is usually

of a crushing type or from the steering wheel of a car in collision. Tears in the liver are sometimes found at necropsy in subjects who have died from other injuries; small tears, therefore, probably occur occasionally, but do not give rise to serious symptoms. Rupture of the right lobe is much more common than of the left, because it is larger and less mobile; usually the tear is on the anterior or the superior surface of the organ. Very often a rib, or ribs, on the right side are fractured. Four clinical types are encountered:

1. There are neither special symptoms nor signs of a ruptured liver. They are those of a hæmoperitoneum, and cannot be distinguished from a ruptured spleen.
2. There are signs of a hæmoperitoneum with localising signs of pain, tenderness, and rigidity in the right upper quadrant.
3. Occasionally, but less commonly than in the case of a ruptured spleen, the symptoms and signs of serious intra-abdominal hæmorrhage are delayed for hours, or even days.
4. A large subcapsular hæmatoma gives rise to a palpable, tender, enlarged liver.

Treatment.—In the first three types a blood transfusion is of cardinal importance, and in all cases laparotomy must be undertaken.

Operation.—As soon as the peritoneum is opened, hæmorrhage from the liver can be controlled by passing a finger into the epiploic foramen (of Winslow) and compressing the hepatic artery and portal vein between the finger and thumb. If the tear extends into the superior surface of the liver, it can be rendered more accessible by dividing the falciform ligament. Adequate access to the injured area is essential and the surgeon must not hesitate to extend the incision into the thorax if the rupture involves the posterior aspect of the liver. Many ruptures of the liver are fissured and do not require excision of tissue; on the other hand, pulped liver and semi-detached portions of doubtful viability must be removed. Failure to debride non-viable liver tissue favours infection and secondary hæmorrhage, a combination which too often culminates in liver failure. When the tear is fissured it is repaired by sutures inserted

parallel to the wound (fig. 1019) and then coapting stitches are placed so that they underrun the mattress sutures. A round-bodied needle and thick catgut should be used and the sutures may be tied over pads of absorbable gauze. When, after debridement, there is loss of substance, the laceration should be packed with strips of Oxycel or suitably shaped pieces of Gelfoam. Two or three superficial sutures are passed and tied loosely to keep the packing in place. In extenuating circumstances where speed is essential, the rent can be packed with a roll of ordinary sterile gauze. If this method is used, post-operative antibiotic therapy must be continued until the wound is healed and the gauze must not be disturbed for three or four days, when it is removed little by little by gentle traction. Adequate drainage must be provided in all cases, because some leakage of bile occurs from torn ductules. The drainage should be maintained till all discharge of bile ceases.

Wounds of the liver occur as a result of gunshot injuries and stab wounds. When, as is commonly the case, the wound of entrance is in the right lower thorax, the thoraco-abdominal approach, excising the wound of entrance, is advisable. In some cases the wound of the liver is comparatively small, and no serious bleeding occurs; but in all cases, laparotomy must be performed to exclude damage to other organs. If the wound is a large one, the principles of arresting hæmorrhage given above are employed.

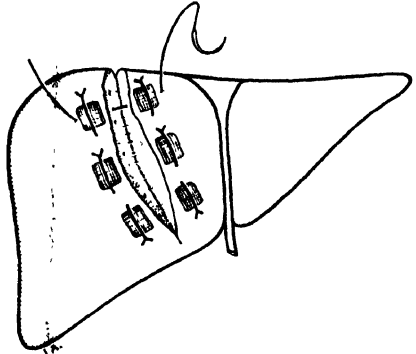


FIG. 1019.—Repair of liver tear. Mattress sutures parallel to tear then under-run with coapting stitches.

ANEURYSM OF THE HEPATIC ARTERY

Of over one hundred cases of aneurysm of the hepatic artery, only six were cured by ligation or excision, and many died as the result of attempting these measures. Arterial grafting under hypothermia is the only treatment which gives more hope of success.

In this connection, it is instructive to observe that in fifty cases of accidental ligation of the hepatic artery during cholecystectomy, the mortality was 60 per cent.

ENLARGEMENTS OF THE LIVER

<i>General enlargement</i>	With jaundice	Regular	{ Virus hepatitis. Cholangitis. ¹ Pylephlebitis. ¹ Carcinoma of head of pancreas. Carcinoma of the common bile duct.
		Irregular	{ Hepatic cirrhosis. Late secondary tumours.
	Without jaundice	Regular	{ Hepatic cirrhosis. Budd-Chiari's syndrome (p. 805). Failing heart. Leukæmia. Rickets.
		Irregular	{ Amyloid. Secondary tumours. Gummata.
<i>Localised swelling</i>			{ Riedel's lobe. Hydatid cyst. Amœbic abscess. ¹ Hepatoma.

¹ Intermittent pyrexia, sometimes rigors occur.

<i>Massive irregular enlargement</i>	{	Polycystic disease. Cholangio-hepato-adenoma. Secondary tumours.
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INFECTIVE HEPATITIS

Infective hepatitis occurs in sporadic and epidemic forms. The virus is excreted in the *fæces* and this is probably the method of spread. It is the commonest form of jaundice in children and young adults, and there is no evidence that one attack confers immunity. The incubation period is from three to five weeks.

The condition commences with anorexia, nausea and perhaps vomiting, with malaise and fever, and jaundice appears after about three days. The liver may be palpable and tender, and occasionally there is transient ascites. The fever subsides shortly after the jaundice appears. Usually the jaundice disappears after two or three weeks, but it may persist very much longer. In such cases, and if the jaundice is deep, the diagnosis from extrahepatic biliary obstruction may be difficult, and liver function tests may not give a clear distinction. If biliary obstruction cannot be ruled out, a needle biopsy may be helpful, but laparotomy for exploration of the bile ducts, and operative cholangiography should be performed if the possibility of obstruction remains.

Sometimes infective hepatitis is followed months or years later by portal cirrhosis, but the factors which cause this are unknown, as most patients recover completely. However, a few develop acute or subacute hepatic necrosis which accounts for the mortality of 3 per cent.

There is no specific treatment and no evidence that any of the antibiotics are of value, but it is regarded as important that the patient should avoid alcohol or strenuous exercise for some months after all symptoms and signs have subsided. A small proportion of patients have recurrent attacks or mild symptoms persisting for a year or more.

Serum Hepatitis.—This may follow blood transfusion, plasma infusion, and, rarely, the administration of sera, and resembles infective hepatitis except that the incubation period is about twelve weeks. Transmission by plasma has been reduced by avoiding the pooling of plasma from a large number of donors. Transmission by syringes is prevented if all syringes are disposable or are sterilised freshly for each patient.

CHOLANGITIS (*syn.* INTERMITTENT HEPATIC FEVER OF CHARCOT)

By cholangitis is meant a state of inflammation of the bile ducts, but it is the radicles of the biliary tree within the liver, as opposed to the extrahepatic ducts, that are the main site of the infection. Cholangitis can occur as the result of mucoviscidosis (p. 847) or obstruction of the common bile duct by a gall-stone or a stricture. Cholangitis is characterised by pyrexia with rigors and jaundice, and occurs as a complication of obstruction to the common bile duct (p. 836).

Suppurative cholangitis is rare, and, like pylephlebitis, is characterised by the formation of multiple liver abscesses.

PYLEPHLEBITIS (*syn.* PORTAL PYÆMIA)

Pathology.—Pylephlebitis can follow a suppurative disease in any part drained by the portal system, but usually arises as a complication of appendicitis or diverticulitis. The process commences as a thrombophlebitis of a small vein draining the infected lesion. The thrombus spreads to a larger collecting vessel, and pieces of infected thrombus break off and are swept into the liver, where they lodge and form abscesses. Abscesses so formed are usually small and multiple, in which case they are connected together, forming a 'canal and cavity' system (Turnbull). Cultures show a mixed infection in which *Esch. coli* is prominently represented. In rare instances there is localisation of the infection.

Clinical Features.—Pylephlebitis is characterised by a hectic temperature and repeated rigors are never absent. In relevant cases this symptom serves to differentiate the condition from a sub-diaphragmatic abscess, but it does not exclude it. If tested for, urobilinogen will be found in the urine in every case. Diarrhoea is a frequent accompaniment. Moderate ascites is sometimes present. The patient soon becomes slightly jaundiced, the liver is somewhat enlarged and tender and the serum bilirubin is raised.

Blood culture is positive in 50 per cent. of cases if blood is withdrawn during or immediately after a rigor. Sensitivity tests must be done on the organism so that the correct antibiotic can be given.

Prophylaxis.—The most important means of preventing pylephlebitis is to administer a broad spectrum antibiotic (penicillin and streptomycin together) in cases of fulminating appendicitis and diverticulitis.

Treatment.—As the organisms responsible are mixed, broad spectrum antibiotics such as tetracycline and chloramphenicol, according to sensitivity must be administered and continued for at least ten days after the temperature has become normal. Blood transfusion is often required. If, in a protracted case, it is considered that there is a sizeable abscess within the liver, laparotomy should be performed with a view to draining the abscess.

Prognosis.—When pylephlebitis is fully established the outlook was formerly almost hopeless, but with the use of antibiotics many cases recover. Extrahepatic portal venous obstruction is likely to develop later.

IDIOPATHIC PYOGENIC LIVER ABSCESS

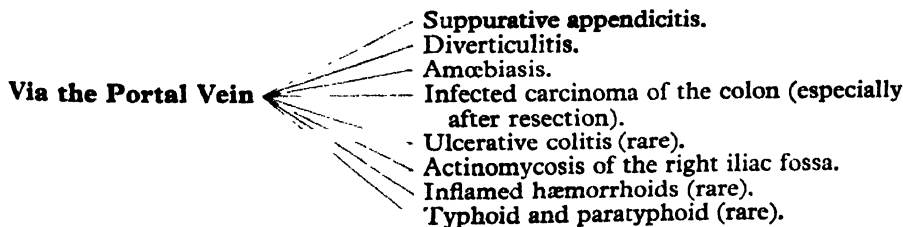
Acute abscesses are usually multiple and may be associated with biliary or abdominal disease, but often, especially in elderly patients suffering from malnutrition, no cause can be found. The clinical features and treatment are similar to those described for pylephlebitis (see above).

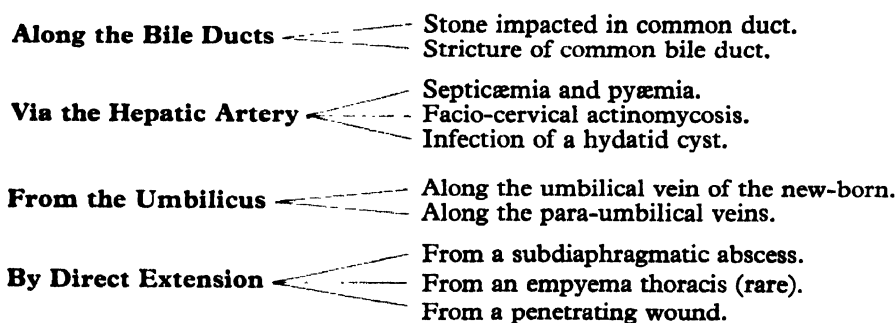
Chronic abscesses usually are single and occur mainly in elderly females. The onset is often insidious, or even silent (cryptogenic abscess). A cause for the infection may be found in the biliary passages or intestinal tract, but in half the cases no source can be discovered. The patient presents with pyrexia, abdominal discomfort, an enlarged liver, and in advanced cases hepatic coma. Leucocytosis is absent, but the serum albumin level rapidly falls, while the level of Vitamin B₁₂ rises (Butler).

The condition is fatal unless, after excluding an amoebic abscess, the abscess is drained, when the mortality is reduced to 30 per cent. Laparotomy is performed, and the abscess is discovered by aspiration. Dependent drainage is effected, after loculi have been broken down by digital manipulation. Examination of the pus will indicate a suitable antibiotic. It is a curious fact that a liver abscess is a rarity even in long-standing cases of ulcerative colitis.

In most instances the cause of a liver abscess is apparent, or can be ascertained, infection reaching the liver in one of several ways, viz:

ABSCESSES OF THE LIVER





AMŒBIC LIVER ABSCESS (*syn.* **TROPICAL ABSCESS, DYSENTERIC ABSCESS**)

Amœbic abscess of the liver is one of the terminations of amœbic hepatitis, which in turn is a complication of amœbic dysentery—not so very uncommon.

Pathology.—*Entamœbæ histolyticæ* (fig. 1020) pass from a focal lesion in the colonic wall into a radicle of the inferior mesenteric vein and via the portal vein they enter the liver to take up residence there, usually in the upper and posterior portion of the right lobe. In the liver the entamœbæ colonise and live at the expense of the liver cells, causing localised liquefaction necrosis. The amount of liver destruction is proportional to the size of the colony and the resistance of the host. In 70 per cent. of cases the abscess is solitary; in 30 per cent. more than one abscess is present. Characteristic pus from an amœbic liver abscess is chocolate-coloured, or like anchovy sauce, and consists of broken-down liver cells, leucocytes, and red blood cells; nevertheless, in an appreciable number of instances the pus is green, from being admixed with bile. In about half the cases the pus contains staphylococci, streptococci, and *Esch. coli*, as well as *E. histolytica*. In the remainder the pus is sterile, but except in long-standing cases, motile entamœbæ can be demonstrated in the last few drops of pus to be withdrawn, or from a scraping of the abscess wall at operation. Commonly perihepatitis causes the liver to become fixed to the diaphragm or the abdominal wall.

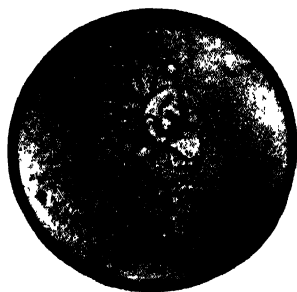


FIG. 1020.—*Entamœba histolytica*.

Course.—An amœbic abscess of the liver runs a variable course:

1. In early stages of amœbic hepatitis with abscess threatening, resolution may occur under emetine treatment, but some authorities doubt whether there is really a stage of hepatitis without abscess formation; it is more probable that treatment results in cure of a small abscess or abscesses (Milroy Paul).

2. When an abscess forms the liver enlarges, most often in an upward direction. It is at this stage that surgical intervention is called for.

3. It may become encapsulated and remain dormant for long periods.

4. Unrecognised and untreated, it may burst into (a) the right lung, (b) the peritoneal cavity, (c) the right pleural cavity, in that order, or, more

rarely, into a hollow viscus (fig. 1021). Exceptionally the abscess points subcutaneously.

Erosion into the lung and the expectoration of a quantity of chocolate-coloured sputum sometimes results in a natural cure.

Bacterial infection is a rather frequent and serious complication of amœbic abscess. While a solitary amœbic abscess is usually amenable to combined specific drug and surgical treatment, the prognosis in cases of multiple amœbic liver abscesses is extremely poor.

Clinical Features.—White males between twenty and forty years of age are usually affected. Although women suffer from amœbic dysentery equally with men, amœbic abscess of the liver is a rarity in the female sex, possibly because they drink less alcohol. As a rule, the condition develops soon after an attack of amœbic dysentery while the patient is resident in a tropical or sub-tropical country. Less frequently its appearance is delayed, sometimes for many months; exceptionally it has occurred more than thirty years after returning home from the tropics. Occasionally an amœbic abscess develops in a carrier who has not had overt dysentery; indeed, it sometimes appears in persons who have had mild diarrhœa not diagnosed as dysentery, and consequently have not had treatment for that condition.

Early Symptoms.—Anæmia, loss of weight, and an earthy complexion are often the first symptoms.

Pyrexia rising to 101° F. (38° C.) or more, at night, with profuse sweating, is usually present. Rigors occasionally occur, especially in the early stages.

Pain is constantly present in the liver area, and is occasionally referred to the right shoulder. Jarring increases the pain so that the patient learns to try to support his enlarged liver with his hands when he walks.

Tenderness and rigidity in acute cases are comparable to that of acute cholecystitis. In chronic cases tenderness may be absent.

Enlargement of the liver can often be demonstrated by clinical methods (fig. 1022), but it is not unusual for an abscess to be present in a liver which is fixed by perihepatitis, and therefore the liver cannot enlarge in a downward direction.

Basal lung signs on the corresponding side can always be elicited in acute cases of hepatic abscess.

Leucocytosis is present in nearly all cases. Polymorphonuclear cells constitute, at the most, only 75 per cent. of the total count.

Examination of the stools for amœbæ should be undertaken, but their absence does not exclude the diagnosis.

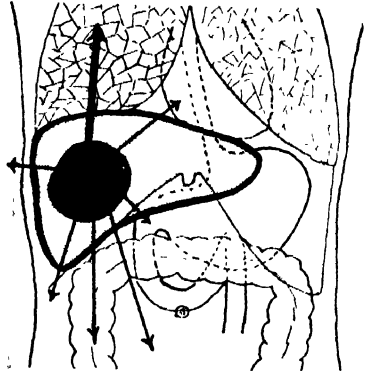


FIG. 1021.—Directions in which a tropical liver abscess may burst. (After Sir Zachary Cope, F.R.C.S.)

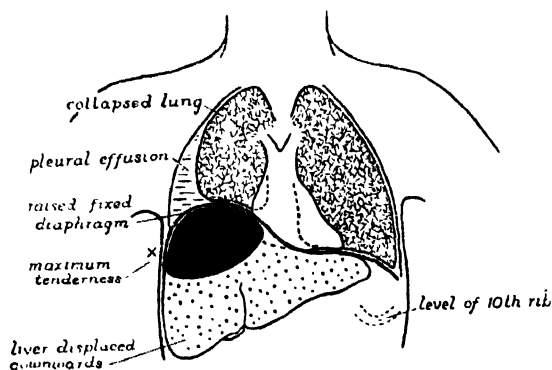


FIG. 1022.—The physical signs of a tropical liver abscess (commonest site). (After A. T. Andreasen.)

liver abscess does not give rise to symptoms; healed lesions have been found at necropsy and calcified lesions (that may have been abscesses) have been revealed radiologically.

Treatment.—When amœbic hepatitis is even suspected, chloroquine 0.6 G. daily by mouth for two days, followed by 0.3 G. daily for twelve to nineteen days, is the treatment of choice. Should there be no response after a few days, it suggests the possibility of secondary infection, and a tetracycline (achromycin or terramycin) in doses of 250 mg. four times each twenty-four hours should be given in addition. When this combination fails to produce the desired result the treatment is stopped, and intramuscular injections of emetine hydrochloride, 1 grain (60 mg.) daily for ten days is substituted. Emetine was the standard remedy for this condition for many years, and very successful it was. Its only drawback is its tendency to cause myocarditis; therefore blood-pressure readings twice daily and an electrocardiograph once weekly should be the rule in patients receiving emetine.

In cases where specific drug therapy proves successful (often it produces such obvious improvement in amœbiasis as to be diagnostic), and provided it has no toxic side-effects, the course of treatment is repeated after an interval of three weeks.

Aspiration.—When, in spite of specific drug therapy, the temperature does not settle, if the pain persists, and particularly if the presence of a suspected abscess is confirmed radiologically, aspiration should be undertaken. A secondarily infected abscess should be treated similarly in the first instance; indeed, such infection can be discovered only by bacteriological examination of the pus.

Technique.—Aspiration must be conducted in the operating theatre. A long needle is necessary, and its bore should be wide (1–2 mm.), as the pus is usually thick in consistency. The important technique of percutaneous introduction of the hollow needle (under local anæsthesia) into the abscess cavity in various locations is shown in fig. 1024. In some cases the abscess eludes the aspirating needle, in which event laparotomy must be performed, thus allowing the liver to be explored by the aspirating needle more thoroughly. At the same time laparotomy permits the exclusion of a primary carcinoma of the liver which, in coloured races, sometimes closely resembles an amœbic liver abscess in its onset and physical signs, and at times is even accompanied by a low pyrexia. In the case of an amœbic liver abscess the amount of pus aspirated averages half a pint (285 ml.) although sometimes a much greater quantity is obtained (up to 18 L. have been reported). Many surgeons with tropical experience instil 1 G. of emetine hydrochloride into the abscess cavity at the end of

Sigmoidoscopy sometimes reveals the characteristic ulcers (p. 909).

Radiography (antero-posterior and lateral positions) often reveals an elevation and fixation of the right cupola of the diaphragm (fig. 1023).

Often an absolute diagnosis is possible only on finding typical pus by exploratory aspiration.

Occasionally an amœbic



FIG. 1023.—Radiograph showing tropical liver abscess. (Major C. J. Hassett, Nairobi, Kenya.)

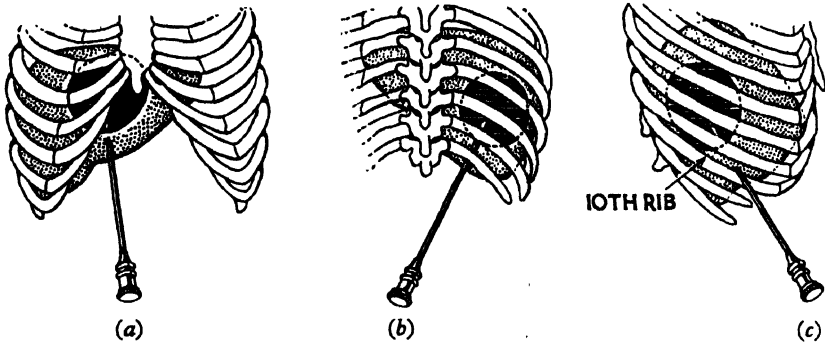


FIG. 1024.—Aspiration of an amœbic hepatic abscess: (a) in the anterior part of the liver; (b) in the posterior part of the liver; (c) located near the dome of the diaphragm.

the aspiration. Aspiration avoids secondary infection and amœbic infestation of the wound.

After-treatment.—A full course of specific drug therapy should be given after aspiration and each re-aspiration.

Operative Aspiration of a Liver Abscess.—This is necessary when an amœbic abscess eludes the needle during the course of the manoeuvres shown in fig. 1024.

Technique.—A posteriorly-situated abscess is aspirated extra-pleurally in a manner similar to the drainage of a subdiaphragmatic abscess (p. 873). When laparotomy is indicated (central and anteriorly-placed abscesses of the right lobe, and all abscesses of the left lobe), packs impregnated with a solution of erythromycin 1:1,000 are so arranged as to isolate the liver. The presence of an abscess having been ascertained by needling, a trocar and cannula is thrust into the abscess cavity, and the abscess is thoroughly emptied by syringe suction. The incision is closed completely and on no account should a drainage tube be employed. Its use leads to invasion of its track by amœbæ and in some cases a fatal spreading infection of the abdominal wall (fig. 1025). After the operation a full course of specific drug therapy should be given, and an antibiotic should be administered.

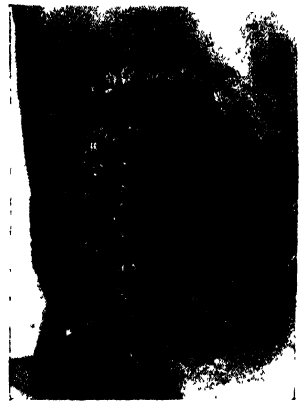


FIG. 1025. — Amœbiasis cutis following drainage of a ruptured amœbic abscess. (Prof R. Milnes Walker, Bristol.)

ACTINOMYCOSIS OF THE LIVER

Actinomycosis (p. 22) produces the well-known 'honey-comb' liver. Actinomycetes reach the organ in one of the following ways:

1. Via the portal vein from actinomycosis of the right iliac fossa—60 per cent. or more.

2. Via the hepatic artery from a more distant primary focus, e.g. facio-cervical actinomycosis—about 30 per cent.

3. From contiguous viscera, e.g. a penetrating peptic ulcer invading the liver—rare. The disease is slow to develop, and causes swelling of one or other lobe. Gradually the liver tissue is destroyed and replaced by multiple abscesses.

Treatment.—Exploration is essential, and in every case of liver abscess of doubtful origin the pus should be examined for actinomycetes. Long-acting penicillin, or other more suitable antibiotic therapy (p. 23) must be continued for some months. Usually by the time the liver is involved the prognosis is grave, but not hopeless.

TUBERCULOSIS OF THE LIVER

In patients with advanced tuberculosis, the liver is frequently fatty; presumably due to the toxicity of pulmonary tuberculosis. In 50 per cent. of necropsies upon patients who have died from tuberculosis, miliary tubercles are present in the liver.

Local tuberculosis of the liver is usually diagnosed at necropsy, but occasionally at laparotomy. There is a large mass containing necrotic material. This lesion is more common in children, and in negroes with little immunity. Less uncommon is a frank tuberculous abscess. In a great majority of cases the lesion is secondary to tuberculosis elsewhere. The symptoms are identical with those of an amoebic abscess. Tuberculosis of the liver responds well to chemotherapy (p. 23) and the timing of aspiration or drainage of such an abscess should be left to the tuberculosis specialist. That these lesions sometimes resolve can be testified by subsequent visualisation of one or more calcareous areas in the liver on radiography.

HEPATIC SYPHILIS

As far as the liver is concerned, syphilis, always an accomplished actor, can, and does, deceive the clinician, and even the operator. Gummata give rise to rounded masses in the liver. These swellings sometimes simulate closely a liver abscess. Multiple gummata of the liver give signs not unlike secondary carcinoma when examined clinically, although, when displayed to the light of day, they lack the characteristic umbilicated appearance of the latter. Penicillin treatment is frequently effective, especially when gummatous lesions predominate. It can also be employed as a therapeutic test to substantiate the diagnosis.

HYDATID DISEASE OF THE LIVER

Although the parasite can thrive in many parts of the body, in 70 per cent. of cases it does so in the liver. After ingestion, the *echinococcus granulosus* enters this organ through a radicle of the portal vein.

Source of Infection.—While dogs are the chief mediators of hydatid disease to human beings by direct contact, it is the eating of grass on which ova

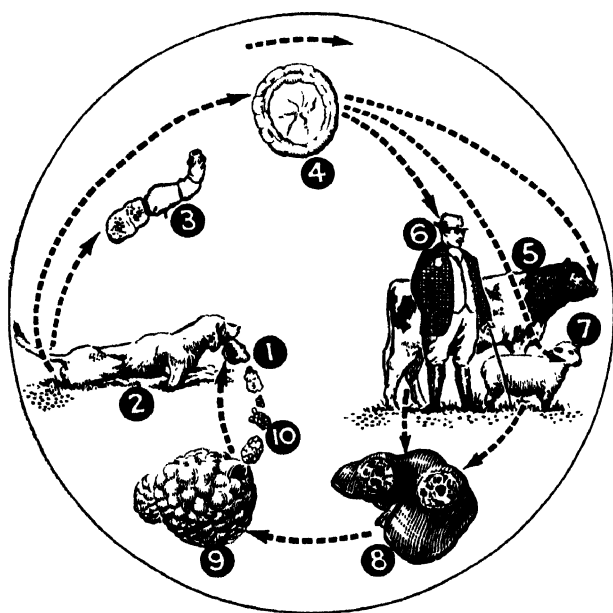


FIG. 1026.—The life-cycle of the *echinococcus granulosus*. (1) Offal infected with hydatid cysts is eaten by a dog (2). The *echinococcus granulosus* (3) develops in the dog's intestine. This parasite is made up of a head and three segments, the last of which contains about 500 ova (4). The ova are expelled from the dog's intestine on to grass, vegetables, etc. Cattle (5), human beings (6), or sheep (7) ingest the eggs. The liver (8) is the organ most frequently infested with hydatid cysts, a larval form of *echinococcus granulosus*. Such cysts (9) harbour thousands of heads of the parasite (scolices) (10). (After V. P. Fontana.)

have been deposited that is usually responsible for the infestation in animals (fig. 1026). Once in the stomach, the ova burrow through the stomach wall to enter the portal system and the liver. Dogs become infected by feeding on offal of infested sheep and, to a lesser extent, cattle. As would be expected, the disease is relatively common in the sheep-rearing districts of Australasia

and South America, while, for the same reason, in the British Isles, Wales shows the highest incidence.

In other parts of the world where the disease is common the life-cycle can be completed in other animals; thus pigs (not kept in styes) and occasionally horses can take the place of sheep and cattle, while in the frozen North (the disease is common among Red Indians and Esquimaux) the wolf → moose → wolf often maintain the cycle.

Pathology.—A hydatid cyst consists of two layers.

1. *The adventitia*, consisting of fibrous tissue, the result of reaction of the liver to the parasite, is grey in colour and blended intimately with the liver, from which it is inseparable.

2. *The laminated membrane* formed of the parasite itself is whitish and elastic, and contains the hydatid fluid. Indeed, this membrane closely resembles a child's uncoloured balloon filled with water, and unless bacterial infection has occurred it peels readily from the adventitia.

Hydatid fluid is crystal-clear; it registers a specific gravity of 1.005–1.009, contains no albumin, and when not too old, hooklets and scolices. The cyst grows very slowly.

The only living part of a hydatid cyst is a single layer of cells (germinal epithelium) lining the cyst. This secretes: (a) internally: the hydatid fluid; (b) externally: the laminated membrane (fig. 1027 (a)). The laminated membrane is of variable thickness, according to the age of the cyst, and is composed of white hyaline material. The surrounding tissues of the host react to the presence of the parasite by entombing it in fibrous tissue—the adventitia. Brood capsules within the cyst develop from the

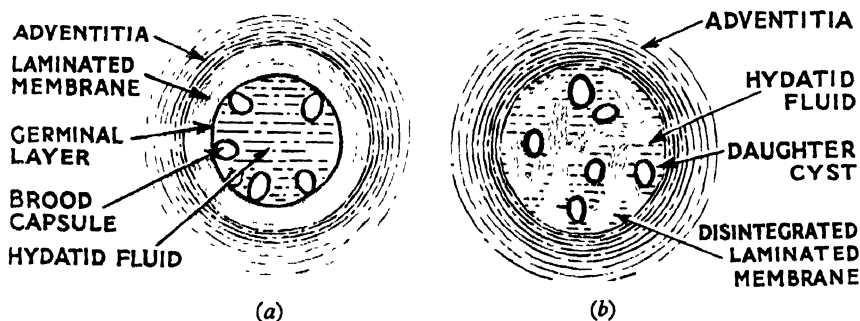


FIG. 1027.—(a) Typical hydatid cyst.

(b) Development of daughter cysts (not common).

germinal epithelium and are attached by pedicles to its innermost wall. Within the brood capsules, scolices (heads of future worms) develop. Should the laminated membrane become damaged, it disintegrates (Dew), and the brood capsules, becoming free, grow into daughter cysts (fig. 1027 (b)). In this event the mother cyst ceases to exist as such, the hydatid fluid and its content being confined by the adventitia only.

Clinical Features.—For a long time, perhaps for some years after the original infestation (which often occurs in childhood), a hydatid cyst remains symptomless (fig. 1028). In the course of time a visible and palpable swelling in the upper abdomen is discovered. The size which a hydatid may attain without causing serious disturbance to health, would seem to be limited only by the capacity of the peritoneal cavity.

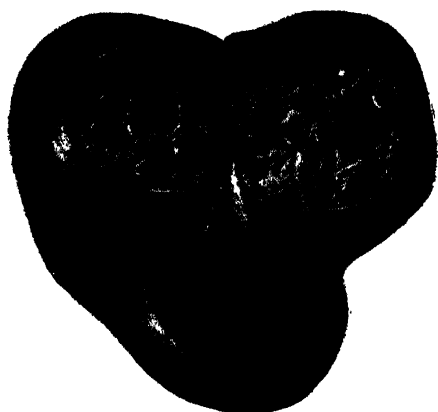


FIG. 1028.—Multiple hydatid cysts in the liver. The patient, who had never left England, died after a street accident.

The complement fixation test, although more complicated, is of greater accuracy.

A blood-count often, but not invariably, shows an eosinophilia (6 per cent. or more).

Course of the Disease.—1. Occasionally the parasite dies. The fluid is absorbed, and all that remains is an encapsuled, laminated, bile-stained membrane. In cases of very long standing the walls of the cyst may calcify.

2. Usually the cyst enlarges gradually, and becomes manifest by its size. It is at this juncture that surgical intervention is indicated.

3. Complications arise. "It is the onset of complications that makes the morbidity not much inferior to that of malignant disease" (Fortacin).

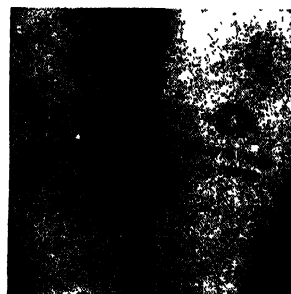
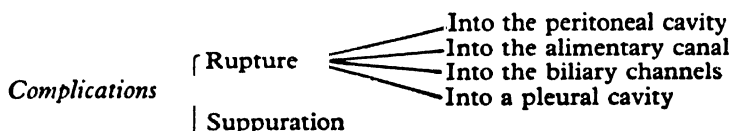


FIG. 1029.—Casoni's test. Positive reaction.

Rupture into the peritoneum is accompanied by profound shock, and all the signs of diffuse peritonitis. As with any case of rupture of a hydatid cyst, anaphylactic phenomena, notably urticaria, are prone to occur. The treatment of intraperitoneal rupture must be immediate, and directed to combating shock and cleansing the peritoneal cavity. Even in those who survive, the ultimate prognosis is poor, for if, as is usual, the cyst contains brood capsules, however meticulous the cleansing, the disease tends to become disseminated within the peritoneum.

TREATMENT OF HYDATID CYST OF THE LIVER

The only treatment is surgical, for there is no drug which has the slightest effect upon the course of the disease.

Operation.—The cyst is exposed by an incision that gives the best access. Abdominal packs are arranged meticulously so as to isolate that portion of the liver containing the cyst from the peritoneal cavity; finally, a black pack, wrung out in 2

per cent. formalin, is tucked around the exposed liver—*black* so that daughter cysts and scolices will show up against the background; *formalin* because this antiseptic kills the parasites. The cyst is aspirated, and a suitable quantity of 10 per cent. formalin solution is injected so as to render the cyst about three-quarters full. An incision is made through the liver overlying the cyst, and the adventitia is opened. This brings the rubbery laminated membrane into view. Very gently the laminated membrane is grasped with ovum forceps and separated from the adventitia with a finger. The aim should be to separate the laminated membrane and deliver the cyst intact; usually this can be accomplished in cysts uncomplicated by the effects of past infection. In complicated cases it is sometimes necessary to remove the laminated membrane piecemeal. When the intact cyst can be enucleated, the resulting cavity in the liver can be closed completely. In less favourable circumstances it is advisable to drain the cavity, particularly when infection is present, when the contents of the cyst are bile stained, or when there is uncertainty of its complete removal. An attempt should be made to remove the adventitia in order to allow the cavity to collapse. In all cases every precaution must be taken to prevent spilling the contents of the cyst into the peritoneal cavity or the layers of the abdominal wall, otherwise dissemination is likely.

NON-PARASITIC CYSTS OF THE LIVER

A solitary traumatic cyst usually follows abdominal trauma and the symptoms are delayed weeks or months after the injury. The clinical manifestations include abdominal pain, frequently referred to the shoulder, a lump that is usually palpable, and less frequently jaundice. In most instances the cyst is located in the right lobe and contains blood and bile. Drainage cures the condition.

A localised collection of cysts is probably the result of sequestration of bile ducts during foetal life. If large, the mass may require excision.

Congenital polycystic disease of the liver is rare, and a third of the cases are associated with congenital polycystic kidneys. Slow, irregular enlargement of the whole liver becoming manifest in adult life is the only clinical feature. The patient may eventually succumb from renal or hepatic failure, or the effects of portal hypertension.

BUDD-CHIARI'S SYNDROME

This syndrome results from obstruction to the hepatic veins. Neoplastic encroachment from other organs accounts for the majority of cases. Other causes are congenital obstruction and thrombosis occurring in the veins, or veno-occlusive disease which occurs particularly in the West Indies as a result of drinking 'bush teas' (herbal tea made from *Senecio* and *Crotalaria*).

Clinical Features.—In acute cases, nausea, vomiting and severe pain, due to rapid enlargement of the liver as a result of congestion, are often followed by death from hypotensive shock.

A less sudden onset is characterised by rapidly accumulating ascites and signs of portal hypertension with a developing collateral circulation. Signs of hepatic insufficiency are usually pronounced, and early death and hepatic coma are the rule. The simultaneous development of œdema of the legs testifies to involvement of the inferior vena cava in the occlusive process.

Chronic cases closely resemble hepatic cirrhosis which, indeed, may develop. Liver function tests indicate severe hepatic parenchymal damage, and rarely does the patient survive more than a few months. A few patients who do not die from hepatic coma succumb to hæmorrhage from œsophageal varices, mesenteric infarction, or intercurrent infection. In short, the Budd-Chiari syndrome, when the occlusion is, or becomes, complete, must be looked upon as a harbinger of death.

HEPATIC CIRRHOSIS

Hepatic cirrhosis is essentially a necrosis of the liver followed by fibrosis and regeneration of liver cells around the radicles of the portal vein, and involving the whole organ.

Ætiology.—It is convenient to divide the ætiological factors into those occurring in infancy and childhood and those occurring in adult life. It will

George Budd, 1808-1882. Professor of Medicine, King's College Hospital, London. He was probably the first to describe the Budd-Chiari syndrome.
Hans Chiari, 1861-1916. Professor of Pathological Anatomy, Prague.

be understood that some of the factors appertaining in early life take years to manifest themselves as hepatic cirrhosis.

Usually there is a fine fibrosis producing slight nodularity (Laënnec's cirrhosis¹ or hob-nail liver) (fig. 1030), but coarse lobulation may result from large regeneration



FIG. 1030.—Portal cirrhosis.

nodules (fig. 1031). The commonest cause in infants is congenital atresia of the bile ducts (p. 823), irremediable by operative means. The jaundiced child soon



FIG. 1031.—Portal cirrhosis with large regeneration nodules. (Prof. R. Milnes Walker, Bristol.)

shows signs of hepatomegaly, splenomegaly, ascites, and œsophageal varices. Most survive only a few months; a few up to three or more years. In adults the condition can arise following post-operative stricture of the common bile duct, or from obstruction of the bile duct due to some other cause. Obstructive jaundice is therefore a cause common to all ages.

Causes in Infants and Children:

1. *Nutritional.* (a) Infantile cirrhosis in India occurs mainly in high-caste Hindu vegetarians. It often progresses rapidly to an early fatal termination. It is suggested that the condition is due to a susceptibility of protein-deficient children to massive necrosis of the liver during viral hepatitis. (b) Kwashiorkor in Africa is characterised by an enlarged liver and peripheral nutritional œdema. Patients with this disorder develop cirrhosis, if at all, only during early adult life. (c) Galactosæmia, patients with this inborn error of metabolism who continue to take lactose invariably develop cirrhosis.

2. *Infective.*—A history of severe jaundice in early infancy is obtainable in a number of instances. Virus hepatitis is believed to play a part in the production of cirrhosis.

3. *Cardiac Cirrhosis.*—Chronic venous congestion of the liver due to tricuspid incompetence, constrictive pericarditis, or chronic congestive failure.

4. *Congenital Hepatic Fibrosis.*—An abnormality often associated with congenital cystic kidneys. The liver shows excessive development of bile ducts and defective intrahepatic portal veins.

5. *Erythroblastosis Fœtalis.*—Isolated instances have occurred.

Causes in the Adult:

1. *Alcohol.*—Over-indulgence in alcoholic beverages is a leading cause. Nutritional deficiency is probably an accessory factor.

2. *Virus Hepatitis.*—The exact percentage of cases in which cirrhosis is an aftermath of virus hepatitis is debatable, and it varies in different localities.

3. *Infestation of the liver by schistosomiasis* undoubtedly is a principal cause of the condition in districts where schistosomiasis is rife (p. 1002).

4. *Nutritional Deficiency.*—In the tropics this appears to be a major factor. Its mode of action is as follows: some toxic agents and mild infections, harmless to the well-nourished, are liable to have a deleterious effect on the liver of protein-deficient individuals. Therefore protein deficiency is indirectly responsible for the cirrhosis.

6. *Hæmochromatosis* (Bronzed Diabetes).

7. *Hepato-lenticular degeneration* (p. 808).

¹Laënnec introduced the term from the Greek *μυρόδς* = tawny, as the nodules are orange-yellow in colour.

8. *Chemical poisons*: carbon tetrachloride, arsenic, chloroform.

9. *Unknown*.—In many instances the cause is obscure. These account for half the cases seen in Great Britain (Sherlock).

Pathology.—As a result of widespread parenchymal destruction and overgrowth of fibrous tissue, the tiny radicles of the portal and hepatic veins become compressed and distorted (fig. 1032). In some cases there may be spasm of the portal veins. Arterioles resist this compression for a longer time than veins. As a consequence the cirrhotic liver becomes dependent upon the hepatic artery for the major portion of its blood supply, and in the desperate effort to maintain the circulation through the liver in these adverse circumstances, extensive intrahepatic communications develop between branches of the hepatic artery and the portal vein, and between the tributaries of the portal and hepatic veins.

Nature's intrahepatic shunts, combined with the opening up of the better-known extrahepatic porta-systemic communications, divert a large portion of the portal blood past the hepatic parenchyma into the systemic venous system. Thus the remaining hepatic cells are deprived of their fair share of portal blood.

Clinical Features:

First Stage.—The early stages of cirrhosis are quiet and long. The liver hypertrophies. In some cases there are repeated attacks of slight jaundice with epigastric pain and vomiting. At each attack areas of the liver are destroyed and replaced by fibrous tissue, while the remainder hypertrophies.

Second Stage.—The liver commences to contract. The spleen becomes enlarged, and usually increases in size with the raised portal blood pressure of which hepatic cirrhosis is the principal cause in adults. About this time, especially in alcoholic subjects, two classical signs may appear:



FIG. 1033.—
Spider naevus.

Spider naevi¹, which are usually located around the face, neck, shoulders and the upper arms. Histologically they prove to be an overgrown end-artery with branching arterioles (fig. 1033).

Palmar erythema—the hands are warm, and the palms are bright red.

Third Stage.—Bleeding from œsophageal varices (p. 810) occurs in a high proportion of cases, and may be the first evidence of the disease. Not infrequently the superficial veins radiating from the umbilicus enlarge, forming a Caput Medusæ. Progressive ascites occurs (fig. 1034). Testicular atrophy and gynæcomasia are common in cirrhosis, no doubt due to increase of œstrogens in the blood. The failing liver is unable to neutralise these substances. In women there is a tendency to masculinisation.

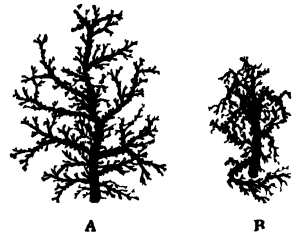


FIG. 1032.—A, Cast of branches of a normal portal venule; B, Those of the branches of a portal venule of a cirrhotic liver, showing their attenuation and distortion. (After I. S. Ravdin.)

¹ An older Miss Muffet
Decided to rough it
And lived upon whisky and gin.

Red hands and a spider
Developed outside her—
Such are the wages of sin.
(W. B. Bean.)

Fourth Stage.—Once hepatic decompensation has occurred (if the patient escapes death from torrential hæmorrhage from œsophageal varices), liver failure, like a sword of Damocles, sooner or later falls upon the sufferer from this disease.

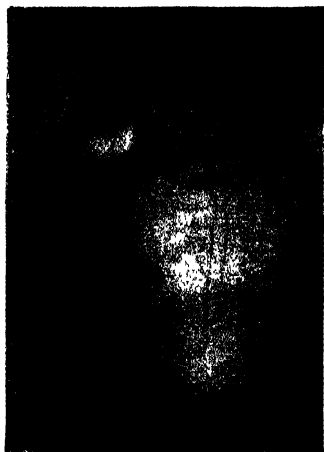


FIG. 1034.—Caput Medusæ, and striæ in cirrhosis with ascites.



FIG. 1035.—Hepatic cirrhosis with ascites.

Liver Function Tests (p. 793).

Liver biopsy (p. 794) is usually necessary only for the diagnosis of a well-compensated case in order to confirm the diagnosis and assess prognosis.

Treatment.—Medical treatment can do much for cirrhosis, but nothing for portal hypertension.

Hepato-lenticular degeneration (*syn.* Wilson's disease) is an uncommon condition that is confined to children and young adults due to an hereditary error in copper metabolism. In infants the symptoms are those of cirrhosis. In adolescents a coarse tremor is commonly an initial symptom. As the disease progresses, muscular rigidity dominates the picture. Kayser-Fleischer rings in the eyes due to peripheral corneal opacity are present. Liver function tests give varying results. Liver biopsy shows cirrhosis. Death ensues within a few years.

In cases developing during the third decade (the disease seldom manifests itself later) evidence of portal hypertension is often conspicuous and, in spite of other manifestations, the advisability of performing portacaval shunt sometimes arises.

PORTAL HYPERTENSION

Ætiology.—The obstruction can be (1) Pre-hepatic; (2) Intra-hepatic; (3) Post-hepatic.

1. **Pre-hepatic.**—About 20 per cent. of patients belong to this group, most of whom are children. The obstruction arises in one of two ways:

(a) *There is congenital absence or abnormality of the portal vein* (fig. 1036).

(b) *Thrombosis of the portal vein* due to extension of the normal obliterative process of the umbilical vein and ductus venosus (fig. 1037) sometimes associated with omphalitis of the new-born (p. 1024).

The vein becomes replaced by a mass of collateral channels which have been described as a cavernoma.

Damocles, a Greek who was feasted with a sword suspended above his head by a single hair (Greek mythology).
Samuel Alexander Kinnier Wilson, 1878–1937. Neurologist, King's College Hospital, London.
Bernhard Kayser, 1869–1937. German Ophthalmologist.
Richard Fleischer, 1848–1909. German Physician.

2. **Intrahepatic** accounts for nearly 80 per cent. of all cases. The cause is cirrhosis of the liver, in particular by areas of regeneration in cases of compensated hepatic cirrhosis.

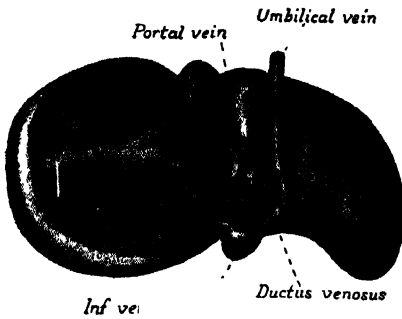


FIG. 1036.—Hilum of the liver of a full-term fetus. (After R. E. Gross.)

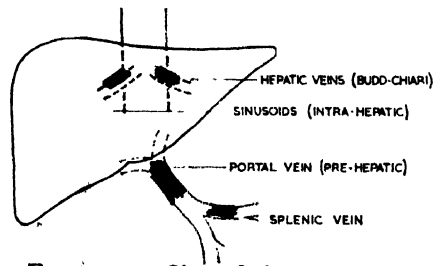


FIG. 1037.—Sites of obstruction. The blocks in the veins represent possible sites of thrombus formation. (After Sir James Learmonth.)

3. **Post-hepatic** is extremely rare. The Budd-Chiari syndrome has been discussed on p. 805. Post-hepatic obstruction is also caused by constrictive pericarditis and tricuspid valvular incompetence.

Collateral Circulation.—When there is obstruction of blood flow to or from the liver, Nature endeavours to relieve the obstruction by opening up normally insignificant anastomotic channels.

(a) *When the obstruction is pre-hepatic*, collaterals between the portal vein distal to the obstruction and the portal vein proximal to the obstruction enlarge. Thus almost unheard of venæ comitantes of the hepatic artery and of the portal vein wax proud. Depending upon the site of the obstruction, some of the collaterals between the portal and systemic venous systems, notably the œsophageal plexus, also become dilated.

ANASTOMOSES BETWEEN THE PORTAL AND SYSTEMIC VENOUS SYSTEMS

Site of Anastomosis	Portal Vessels	Systemic Vessels	Signs and Symptoms
1 Plexus around lower end of œsophagus.	Œsophageal branches of left gastric vein.	Lower systemic œsophageal veins.	Hæmatemesis or mælena.
2 Around umbilicus.	Para-umbilical veins (accompany the round ligament of the liver).	Superficial veins of the anterior abdominal wall.	Caput Medusæ.
3 Plexuses around lower third of rectum and anal canal.	Superior hæmorrhoidal vein.	Middle and inferior hæmorrhoidal veins.	Hæmorrhoids (rare).
4 Extraperitoneal surfaces of abdominal organs.	Tributaries of superior and inferior mesenteric veins.	Subdiaphragmatic and retroperitoneal veins.	Silent.

(b) *When the obstruction is intrahepatic*, anastomotic channels outside the liver between the portal and systemic systems become engorged, dilate, and so an increasing proportion of the obstructed portal venous blood by-passes the liver. The only ones which are dangerous to life are those in the submucosa of the œsophagus and upper end of the stomach. In patients with portal hypertension, hæmorrhoids, if present, are usually of the idiopathic type (p. 989).

Œsophageal Varices.—Although called 'œsophageal', it is important to realise that these varices extend well into the stomach. Œsophageal varices (fig. 1038) are the most important collaterals of the portal circulation. Sometimes, as intra-abdominal pressure rises, e.g. when straining at stool or during heavy lifting, they rupture; in other cases the overlying mucosa becomes abraded by a rough bolus or excoriated by regurgitated gastric juice. Bleeding can occur slowly or catastrophically. The presence of œsophageal varices can be demonstrated by:

(a) *Radiology* after a barium swallow (fig. 1038). In the great majority of cases œsophageal collaterals can be revealed by this method as filling defects which may extend even as far up as the pharynx.



FIG. 1038.—Barium swallow displaying filling defects due to œsophageal varices. (A. H. Hunt, F.R.C.S., London.)

(b) *Œsophagoscopy* will demonstrate them in all cases, but great care must be exercised not to abrade the overlying mucous membrane.

Clinical Features.—If the patient is known to have hepatic cirrhosis, it should be assumed that the hæmorrhage is coming from œsophageal varicose veins, although patients with cirrhosis of the liver are particularly likely to suffer also from a gastric or duodenal ulcer. In nearly all cases the spleen is palpably enlarged. Radiography and œsophagoscopy in the presence of severe hæmorrhage are apt to be inconclusive. Liver function tests, if abnormal, greatly favour hepatic cirrhosis as the cause of the hæmorrhage.

The presence of engorged œsophageal varices is a positive indication that surgical treatment should be carried out without undue delay. So often catastrophic hæmorrhage comes like a thief in the night while the patient is doing well under medical treatment. The very fact that he is doing well implies that regeneration of areas of the liver is in progress. Regeneration nearly always entails increased pressure on the intrahepatic portal network, and so the patient may have a massive hæmorrhage which might have been averted by a timely portacaval shunt.

Treatment of Massive Hæmorrhage from Œsophageal Varices:

1. **Conservative Treatment.**—Rest, sedation, and replacement of blood will carry approximately half the patients who are actively bleeding past the emergency period. Pituitrin, which causes lowering of the portal blood pressure by constricting the splanchnic arterioles, is a valuable adjunct. To obtain the maximum effect, 20 units of obstetric pituitrin diluted in 200 ml. of isotonic saline solution is given intravenously over a period of twenty minutes. For the prothrombin deficiency consequent upon liver damage, the administration of vitamin K is necessary.

2. **Tamponade** is required when the foregoing measures fail after a short trial. A small balloon tied firmly to the end of a gastric aspiration tube can be employed but the Sengstaken trilumen tube (fig. 1039) is better. The œsophageal and gastric balloons are inflated and moderate tension applied by strapping the tube to the

forehead. While the Sengstaken tube controls œsophageal hæmorrhage in every case, and is therefore a diagnostic as well as a therapeutic measure, it has its drawbacks. Chief of these is the inspiration of saliva into the lungs, which must be anticipated. In addition to respiratory difficulty, ulceration and rupture of the œsophagus may occur if the balloon is retained for more than seventy-two hours. But the greatest danger is renewed hæmorrhage when the balloon is deflated and withdrawn. Therefore, after twenty-four to forty-eight hours the balloon should be deflated, but left in place for four hours. If bleeding recommences, immediate operation is necessary.

3. Urgent Operation for Bleeding Varices.—If the liver function is good a portacaval anastomosis (p. 813) may be undertaken, provided that a patent portal vein can be demonstrated by preliminary portal venography. Otherwise the alternative is a direct operation on the œsophagus, either ligation of the varices or œsophageal transection (p. 814) through a left thoracic approach.

4. Evacuation of Blood from the Alimentary Tract.—As soon as possible the alimentary tract must be cleared of blood; otherwise encephalopathy, due to the absorption of protein metabolites, may follow. All unclotted blood must be aspirated from the stomach, and colonic wash-outs given until all blood has been evacuated.

Portal Hypertension *au Froid*

By this is meant that the patient has portal hypertension, but is not bleeding obviously or dangerously. First let us consider how pre-hepatic portal obstruction differs from intrahepatic portal obstruction.



FIG. 1040.—Enlargement of the spleen in pre-hepatic portal obstruction.

The anæmia is usually due to oozing from œsophageal varices, but sometimes hypersplenism is a factor (p. 789). As the liver is normal, or nearly so, liver function tests remain normal as the disease advances. Ascites does not occur unless there is active phlebitis in the portal system of veins.

Splenoportography (p. 774) will usually display the site of the block, but if the portal vein does not fill, the cause may lie in a reversed flow in the splenic vein. If, therefore, the presence of a patent portal vein is to

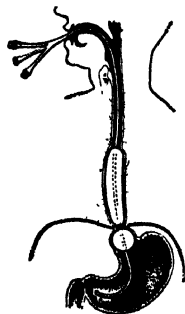


FIG. 1039.—The Sengstaken trillum tube with gastric and œsophageal balloons *in situ*.

The patient with pre-hepatic portal obstruction is nearly always young, and often a child. The most usual cause for advice being sought is either a sudden hæmorrhage or listlessness due to anæmia. On examination the liver is impalpable, but the spleen is obviously enlarged (fig. 1040).



FIG. 1041.—Venogram in case of extrahepatic obstruction.

be definitely excluded, a venogram must also be done by injection into a mesenteric vein at laparotomy (fig. 1041).

The patient with intrahepatic portal hypertension is usually an adult and suffers from hepatic cirrhosis. Enlarged porta-systemic venous communications are present long before serious œsophageal hæmorrhage occurs.



FIG. 1042.—Enlargement of the veins of the abdominal wall in a case of portal hypertension. Infra-red photograph. (A. H. Hunt, F.R.C.S., London.)



FIG. 1043.—Spleno-portograph in a case of intrahepatic portal hypertension showing dilated splenic and portal veins. (Professor R. Milnes Walker, F.R.C.S., Bristol.)

œsophageal varices can be demonstrated radiologically or seen through an œsophagoscope. Enlargement of veins of the abdominal wall radiating from the umbilicus may be present (fig. 1042). As long as hypertrophy of liver tissue is sufficient to compensate for the cellular destruction by cirrhosis, the liver function tests remain within normal limits.

Measuring the Portal Blood Pressure.—One method is to record the intrasplenic pressure, and this can be done at the same time as transplenic portovenography. In cases of intrahepatic obstruction an alternative method is to catheterise a hepatic vein. The technique is not especially difficult, and can be undertaken in any centre equipped for cardiac catheterisation. Following local anæsthesia and opening an antecubital vein as for cannulation, a cardiac catheter with only a terminal opening is passed into the vein. The catheter is advanced through the right auricle and inferior vena cava under radiological control. The tip is then insinuated into a hepatic vein until a peripheral radicle is occluded (fig. 1044). It is important that the tip of the catheter should be in a peripheral position, so as to obviate obstruction by a bifurcation of the vein, and thus falsify results. Pressure recordings are then taken. The normal portal pressure is 8 to 12 mm. Hg. In cases of established cirrhosis it may rise to 30.0 mm. Hg. The portal pressure is always high in cirrhotic patients who have œsophageal varices. The ascites of cirrhosis does not bear a direct relationship to elevated portal blood pressure, as was formerly believed.

Indication for a Porta-systemic Shunting Operation:

Hæmorrhage from œsophageal or gastric varices, and the risk of hæmorrhage when large varices are present.

Contraindications:

1. Normally a portacaval shunt should not be performed unless the patient's serum albumin¹ is more than 3 G. per 100 ml.

¹ Normal level 3.6 to 4.5 G. per 100 ml.

2. If the patient is jaundiced significantly.

Occasionally these stipulations are relaxed if further hæmorrhage is threatened.

Pre-operative treatment is directed mainly to correcting the accompanying hypochromic anæmia; blood transfusion is often required. Control of a bleeding tendency (secondary to impaired production of prothrombin) is essential and for this purpose vitamin K is given parenterally. If there is no immediate danger of renewed hæmorrhage, those in whom a porta-systemic shunting operation is indicated are often benefited greatly by a course of pre-operative medical treatment.

If ascites is present, a low sodium diet, in conjunction with diuretics, often causes absorption of ascitic fluid. If possible, paracentesis should be avoided because valuable protein is lost thereby.

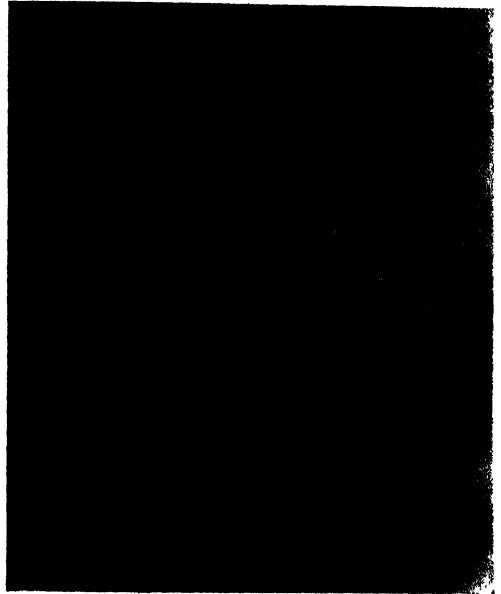


FIG. 1044.—Catheter in hepatic vein.
(Dr. Frank Ross, Bristol.)

OPERATION FOR PORTAL HYPERTENSION

An anæsthetic that allows a high concentration of oxygen to be given is important. Throughout the operation blood volume is maintained by transfusion.

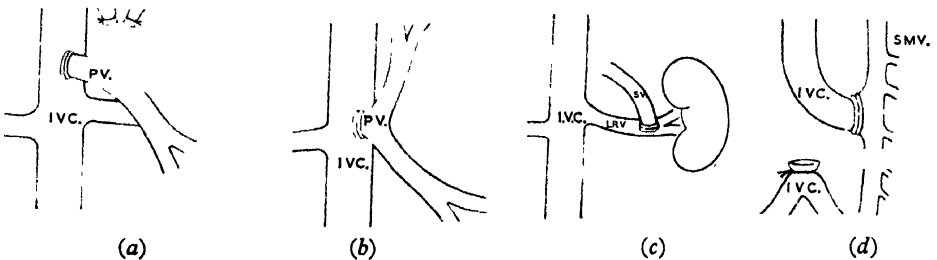


FIG. 1045.—Types of porta-systemic anastomosis.

- | | |
|--|------------------------------------|
| (a) End-to-side portacaval anastomosis. | (c) Spleno-renal anastomosis. |
| (b) Side-to-side portacaval anastomosis. | (d) Mesenterico-caval anastomosis. |

Operative Portovenography.—When the transplenic portovenographic films are inconclusive, or have not been obtainable because the spleen has been previously removed, the site of the block can be demonstrated by portovenography after the abdomen has been opened. Thirty ml. of Conray 280 are injected into a suitable vein in the gastro-hepatic omentum, or into the superior mesenteric vein. A radiograph is then taken with a portable machine during the injection of the last few ml., and with the help of the venograph thus obtained, usually the site of the block can be determined accurately.

1. **Splenectomy.**—This has no place in the treatment of portal hypertension except in the very rare cases of thrombosis confined to the splenic vein, or in combination with some other operation.

2. Portacaval Anastomosis.—Unless the portal vein is thrombosed (which renders a portacaval anastomosis impossible), a portacaval shunt is nearly always the operation of choice, provided that the liver function is adequate. The serum albumin level should be at least 3.0 G. per 100 ml., and the serum bilirubin not more than 1.0 mg. per 100 ml.

The best approach is a thoraco-abdominal incision removing part of the eighth or ninth rib. The diaphragm is incised to allow upward displacement of the liver. The peritoneum is divided along the lower border of the right lobe of the liver and the lateral border of the duodenum.

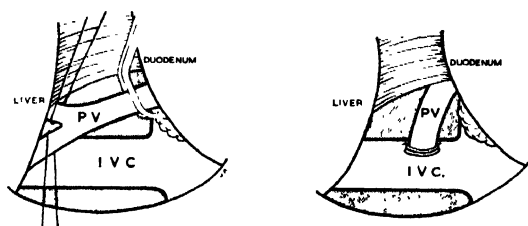


FIG. 1046.—Portacaval anastomosis.

The portal vein is exposed by an incision through the peritoneum forming the anterior wall of the epiploic foramen, and a length of about 3 cm. freed. An incision in the peritoneum over the vena cava exposes its anterior surface. The portal vein, or its two main branches, is now ligated at the hilum of the liver, a Blalock clamp placed on its lower end, and the vein divided.

After placing a side clamp on the vena cava, an incision is made in its anterior wall, and an end-to-side anastomosis made using everting fine silk sutures. The diaphragm is repaired and the wound closed with abdominal drainage.

With careful selection of cases the mortality is about 6 per cent., and recurrent bleeding from varices after this operation is most unusual.

3. Spleno-renal Anastomosis.—This may be performed if there is thrombosis of the portal vein, but it is contraindicated if the splenic vein is less than 1 cm. in diameter as shown by venography. This operation is less effective than a portacaval anastomosis in preventing further hæmorrhage, but carries a lower risk of encephalopathy. Owing to the small size of the splenic vein, this operation is rarely of value in cases of extra-hepatic obstruction in children.

The operation is performed through a left thoraco-abdominal incision. The spleen is removed but during the dissection of the hilum as great a length as possible of the vein is carefully preserved. Enough vein is separated from the pancreas to allow its apposition to the renal vein, and it is temporarily occluded with a bull-dog clamp. The renal vein is dissected out and occluded by clamps or threads and the splenic vein anastomosed by everting sutures to an opening in its upper surface. To prevent engorgement of the kidney a temporary clamp may be put on the renal artery, but it should be released after fifteen minutes to allow a fresh flow of blood to the kidney.

4. Superior Mesenterico-caval Anastomosis.—When the portal vein is thrombosed this alternative may be possible. The inferior vena cava is divided just above its lower end and ligated below. The proximal end is joined to the side of the superior mesenteric vein in the root of the mesentery.

Complications of Porta-systemic Shunting.—Any major shunt, by the very fact that portal blood is by-passing the liver, increases the liability to neuro-encephalopathy (p. 792). This may develop suddenly and tends to occur most often about the second or third day. Usually there is a quick response to a protein-free diet. In most instances a gradual return to a normal diet can be permitted; in a few, attacks of episodic stupor dictate that a supervised dietetic regimen must continue. Serial post-operative liver function tests show that diversion of the portal blood-stream from the liver does not cause greater deterioration in hepatic function than that occurring in comparable controls.

5. Alternative Procedures.—When the patient's liver function is inadequate for a porta-systemic shunt or there is no suitable patent vein, it is desirable to make an effort to interrupt the communications between the left gastric (portal system) and œsophageal veins (azygos system)—porto-azygos disconnection—so cutting off the flow of portal blood to the varices.

(a) **ŒSOPHAGEAL TRANSECTION** (Milnes Walker).—The aim of this operation is to cut off the flow of portal blood to the varices. Through a left trans-thoracic approach the lower end of the œsophagus is mobilised and the vagus nerves separated from it. The

œsophagus is transected at the level of the hiatus in the diaphragm, and resutured with continuous sutures in the mucosa and submucosa which obliterate the vessels.

(b) **PORTO-AZYGOS DISCONNECTION WITH GASTRIC TRANSECTION** (Tanner).—Through a left abdomino-thoracic approach the lower end of the œsophagus and upper 5 cm. of stomach are mobilised, dividing all vessels entering this part. The stomach is then transected 2 cm. below the cardiac orifice and resutured with two layers of continuous catgut sutures in order to obliterate the vessels.

(c) **SUTURE OF THE VARICES** after opening the lower part of the œsophagus longitudinally (Boerema, Crile).

NEOPLASMS OF THE LIVER

Benign:

Hæmangioma occurs more commonly in the liver than in any other internal organ, and usually the neoplasm is of the cavernous type. As a rule, a hæmangioma of the liver is solitary, and is found either at necropsy or incidentally at operation. Exceptionally, it becomes large enough to cause symptoms, in which event laparotomy is required to establish the diagnosis. The compressibility of the tumour makes the diagnosis unmistakable, and on no account should biopsy be performed, for the resulting hæmorrhage is terrific. It may be possible to excise the tumour with a shell of normal liver tissue around it, although this sometimes necessitates hemi-hepatectomy (p. 817).

Hepato-adenomata are composed of hepatic cells. Frequently multiple, they are distinguished with difficulty from the nodular hyperplasia of cirrhosis. They seldom grow to a large size and there is no evidence that they become malignant. Their removal is meddlesome, and unnecessary.

Cholangio-adenomata originate from the bile-ducts and form small subcapsular masses. They are found at operation or necropsy, and can simulate closely metastatic deposits, which should not be assumed without biopsy. They are harmless.

Cholangio-hepato-adenoma originates from both hepatic cells and bile-ducts, and is usually solitary. It occurs more frequently in infants than adults, and may increase in size. It should therefore be excised, if it is possible to do so.

Malignant:

Primary carcinoma of the liver is uncommon in European races, but is by no means rare in African natives, Malaysians, Chinese, and Japanese. In Johannesburg, among the Bantu the rate of primary carcinoma of the liver is at least fifty times that of the white population. In the majority of these patients it arises as a complication of cirrhosis. A similar ratio is not found among the coloured and white North American peoples. Two pathological varieties are described:

1. **Hepatoma** arises in the liver cells. There are great variations in the malignancy of this tumour, which may be multicentric in origin, particularly when it occurs in a cirrhotic liver. At one end of the scale is a relatively benign, firm, slowly growing tumour (fig. 1047), and at the other a rapidly growing, soft neoplasm prone to undergo necrosis, which soon metastasises within the liver, or to the lymph nodes in the hilum of the liver, mediastinum or neck.



FIG. 1047.—Hepatoma resected from the liver successfully.

Norman Cecil Tanner, Contemporary. Surgeon, Charing Cross Hospital, London.

de Boerema, Contemporary. Professor of Surgery, Wilhelmina Gasthuis, Amsterdam, Holland.

George Crile, Jr., Contemporary. Head of the Department of General Surgery, The Cleveland Clinic, Cleveland, Ohio, U.S.A.

2. **Cholangioma** arises in the intrahepatic bile-ducts and is columnar-celled. It grows to a large size and, metastasising within the liver, is rapidly fatal. This variety of primary carcinoma of the liver is five times less common than the foregoing.

Clinical Features of Primary Carcinoma of the Liver.—In white races the patient is usually over forty years; in coloured people the patient is frequently under forty. The usual train of events is anorexia, rapid loss of weight, and asthenia.

On examination an enlarged liver is found and often a localised mass, viz. —————→



Less frequently the patient presents on account of a swelling he has discovered in the upper abdomen: the swelling may, or may not, be tender, depending on the rate of growth. Especially in coloured people, neoplastic proliferation is sometimes so rapid as to cause a low pyrexia, in which event an amœbic abscess will be considered as a possible diagnosis. Every effort is made to seek a possible primary growth, bearing in mind that, at any rate in white races, secondary carcinoma of the liver is immeasurably more common than primary carcinoma.

Ascites is present in about 40 per cent. of patients at the time of the first examination; diagnostic aspiration often reveals blood-stained ascitic fluid. Such ascites resists therapy, and re-accumulates rapidly after paracentesis.

SECONDARY NEOPLASMS OF THE LIVER

Secondary Carcinoma.—As is well known, the liver is a favourite site for carcinomatous metastases.



FIG. 1048.—Secondary carcinoma of the liver.

Characteristically, secondary growths in the liver, owing to degeneration of cells in the centre, are umbilicated. As a rule, carcinomatous deposits in the liver are multiple (fig. 1061). Occasionally at laparotomy a secondary growth in the liver is found to be apparently solitary and accessible. In such circumstances, provided that the primary growth can be or has been resected, excision of that part of the liver containing the neoplasm sometimes results in a long-lasting survival.

Secondary carcinoid (argentaffin) tumours (p. 916) are rare; where they occur they often grow to a large size.

Secondary melanoma occurs particularly when the primary growth is in the eye. Fifteen years or more have elapsed between the removal of the primary growth and the appearance of secondary deposits (p. 483).

Secondary Sarcoma.—The liver is not an uncommon site for secondary deposits in cases of sarcoma, but the lungs show a much higher incidence.

Resection of a Benign Tumour of the Liver.—In order to conserve blood, the hepatic artery and portal vein can be clamped lightly at the porta hepatis, and released every five minutes. Another very important point is that branches of the hepatic

artery and the portal vein ramify through the liver in the portal canals: each of these canals is surrounded by a prolongation of Glisson's capsule which, when encountered with a blunt instrument, gives a sense of resistance. Thus, if liver tissue is dissected by employing a fine-pointed hæmostat using gentle strokes very slowly (Heneage Ogilvie), it is possible to divide liver tissue, but spare the vessels, which can be clamped and ligated, or coagulated with diathermy, before division. The resection completed, the resulting raw surfaces of the cut liver are dealt with in the same manner as described for rupture of the liver (p. 794).

Hemi-hepatectomy.—A number of cases of successful removal of the surgical right lobe of the liver for primary and secondary tumours or for carcinoma of the gall-bladder have been reported. Removal of the surgical left lobe (fig. 1017) is less frequently necessary and left (anatomical) lobectomy usually suffices (Rodney Smith).

Right Hemi-hepatectomy.—After operability has been determined through a right paramedian incision, extension into a right thoraco-abdominal is achieved through the eighth interspace. The liver is mobilised and rotated forwards and upwards into the chest to expose the porta hepatis. Working from below upwards, the cystic artery and duct, the right hepatic duct, right hepatic artery, and right branch of the portal vein are successively divided between ligatures. The inferior vena cava is then exposed by mobilising the duodenum and head of pancreas and drawing them over to the left (Kocher's manœuvre). It is then possible to expose, ligate, and divide the inconstant minor hepatic veins running from the right lobe into the cava, up as high as the site of entry of the right hepatic vein; great care must be taken not to obstruct the venous return in the vena cava. The liver is then divided from in front and above between the surgical lobes (fig. 1017) by diathermy. The right hepatic vein is then exposed and divided and also the right-sided branches of the middle hepatic vein as they are encountered. The raw surface of the left lobe is covered with the falciform ligament and it is wise to drain the common bile-duct.



FIG. 1049.—Right hemi-hepatectomy almost completed; the hepatic veins remain to be ligated (diagrammatic). I.V.C. = Inferior Vena Cava, C.B.D. = Common Bile Duct, P.V. = Portal Vein, H.A. = Hepatic Artery, L.T. = Ligamentum Teres. (After C. Rob and R. Smith.)

Francis Glisson, 1597–1677. *Regius Professor of Physic, Cambridge.*
 Sir Heneage Ogilvie, *Contemporary.* *Consulting Surgeon, Guy's Hospital, London.*
 Rodney Smith, *Contemporary.* *Surgeon, St. George's Hospital, London.*
 Theodor Kocher, 1841–1917. *Professor of Clinical Surgery, University of Berne, Switzerland.*

CHAPTER 35

THE GALL-BLADDER AND BILE DUCTS

SURGICAL ANATOMY AND PHYSIOLOGY

Surgical Anatomy

Embryology.—During the fourth week of foetal life the hepatic diverticulum arises from the ventral wall of the foregut near its opening into the yolk sac. As this diverticulum elongates into a stalk to form the choledochus, a lateral bud is given off, viz., which is destined to become the gall-bladder and cystic duct. By the sixth week the embryonic hepatic duct is sending out many branches which join up with the canaliculi attached to the liver cells. At the twelfth week the liver is excreting bile into the primitive bile ducts and the gall-bladder which, during very early foetal life, are hollow. As is usual with embryonic tubular structures, hyperplasia obliterates the lumina of this ductal system. By the fifth month recanalisation has restored patency, and the flow of bile recommences. During early foetal life the gall-bladder is entirely intrahepatic.



Congenital abnormalities of the gall-bladder and bile ducts are described on p. 822.

The adult gall-bladder is pear-shaped, and 7.5 to 12.5 cm. long, with a capacity of about 50 ml., but capable of fifty-fold distension. For purposes of description, the gall-bladder is divided into a fundus (which can be palpated when the gall-bladder is distended), a body, and a neck which terminates in the narrow infundibulum (fig. 1050). The somewhat angulated distal part of the neck forms a pouch, called Hartmann's pouch; this is a common site for a solitary gall-stone to lodge. Hartmann's pouch, because it overlaps and partially hides the cystic duct, is the best guide to that structure. The muscle fibres in the walls of the gall-bladder are arranged in a criss-cross manner, being particularly well developed in the neck. The mucous mem-

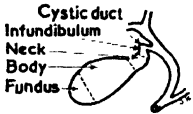


Fig. 1050.—The anatomical subdivisions. (After J. D. Rose.)

brane is thin, contains no glands; instead there are indentations of the mucosa that sink into the muscle coat; these are the crypts of Luschka.



The cystic duct is usually 2.5 cm. in length, but because of its S-shaped course its length is reduced to 1.5 cm. That half duct nearest the gall-bladder often contains the spiral valve of Heister. Internal projections of circular muscle fibres account for the valve, which makes the passage of calculi, as well as a probe, more difficult. The lower half of the duct is bereft of a valve and a probe or catheter passes easily.

The common hepatic duct is usually less than 2.5 cm. long, and is formed by the union of the right and left hepatic ducts: it is not exceptional for the right hepatic duct to be duplicated.

The common bile duct, which is 7.5 to 10 cm. long, is formed by the junction of the cystic and the common hepatic ducts. It is divided into four parts (fig. 1051): 1. *The supra-duodenal portion*, about 2.5 cm. long, runs in the free edge of the lesser omentum. 2. *The retroduodenal portion*. 3. *The infraduodenal portion* lying in a groove, but at times in a tunnel in the posterior surface of the pancreas. The right edge of the inferior vena cava lies not far distant behind it. 4. *The intra-duodenal portion* passes obliquely through the wall of the second part of the duodenum to open on the summit of the duodenal



FIG. 1051.—The four divisions of the common bile duct.

papilla, which is surrounded by the sphincter of Oddi. Near its opening it is often joined by the main duct of the pancreas (duct of Wirsung), but the presence of an ampulla of Vater¹ (an ampulla indicates a dilatation), contrary to anatomical teaching, is uncommon (Sterling). The arrangements of the exit are shown in fig. 1052.

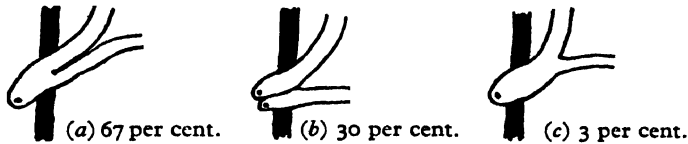


FIG. 1052.—(a) Common orifice occupying one-third to two-thirds of the length of the papilla. (b) Two orifices surrounded by the same sphincter. (c) An ampulla present.

The arterial supply and its aberrations are described on p. 824.

Lymphatics.—The lymph vessels of the gall-bladder (subserosal and submucous), drain into the *cystic lymph node of Lund* (the sentinel lymph node), which lies in the fork created by the junction of the cystic and common bile ducts. Efferent vessels from this lymph node go to the hilum of the liver, and to the coeliac lymph nodes. The subserosal lymphatic vessels of the gall-bladder also connect with the subcapsular lymph channels of the liver, and this accounts for the frequent spread of carcinoma of the gall-bladder to the right lobe of the liver.

Surgical Physiology

Bile, as it leaves the liver, is composed of 97 per cent. water, 1 to 2 per cent. of bile salts, and 1 per cent. of pigments, cholesterol, and fatty acids. The liver excretes bile at a constant rate, estimated at 40 ml. per hour. Except when active gastroduodenal digestion is in progress, the sphincter of Oddi contracts and consequently bile leaving the liver passes into the gall-bladder. The healthy gall-bladder has several functions:

1. **Concentration of Bile.**—By the absorption of water, sodium chloride, and sodium bicarbonate by the mucous membrane of the gall-bladder into the blood-stream and to a lesser extent into the lymphatics, the hepatic bile which entered the gall-bladder becomes concentrated from 5 to 10 times, with a corresponding increase in the proportion of bile salts, bile pigments, cholesterol, and calcium it contains.

2. **Reservoir for Bile.**—Bile is stored in the gall-bladder until the gall-bladder is full, when the sphincter of Oddi partially relaxes and the overflow trickles into the duodenum. Contraction of the gall-bladder is believed to be stimulated by *cholecystokinin*, a hormone secreted and absorbed into the blood-stream when acid chyme enters the duodenum. When the gall-bladder contracts, synchronous relaxation of the sphincter of Oddi is brought about and the contents of the gall-bladder are discharged rapidly into the duodenum. On the other hand, when certain foods are ingested, notably fats and egg-yolk, their physical presence in the duodenum evokes relaxation of the sphincter of Oddi and strong contractions of the gall-bladder. Loss of the gall-bladder is compensated for by dilatation of the extrahepatic ductal system. This serves its purpose sufficiently well to provoke no symptoms.

3. **Changing the Reaction of the Bile.**—The bile excreted by the liver is alkaline (pH 8.2), whereas that in the gall-bladder is acid (pH 7.0 to 7.6). This change is brought about by the selective absorption of salts by the epithelium.

4. **Cholesterol excretion** by the gall-bladder has not been proved, but pathological evidence of the gall-bladder epithelium choked with crystalline cholesterol (p. 833) is suggestive that the gall-bladder may add cholesterol to the bile.

5. **Secretion of Mucin.**—About 20 ml. is secreted each twenty-four hours.

INVESTIGATIONS

Cholecystography is essential in the diagnosis of gall-bladder disease. It is not used in acute cholecystitis, and it is valueless if the patient is jaun-

¹ So universally is the term 'ampulla of Vater' employed to indicate the region of the stoma of the common bile duct and the duct of Wirsung that it would be confusing to disturb this convenient designation.

Ruggiero Oddi, 1846–1906. Surgeon and Anatomist, Rome.

Johann Wirsung, Prosecutor at Padua, was murdered when entering his house at night in 1643.

Abraham Vater, 1684–1751. Professor of Anatomy and Botany, Wittenberg.

Julian Alexander Sterling, Contemporary. Senior Attending Surgeon, Albert Einstein Medical Center, Philadelphia.

Federick Bates Lund, 1866–1950. Surgeon, Boston City Hospital, Boston, Mass., U.S.A.

diced with a plasma bilirubin level of over 3 mg. per cent., for the poorly functioning hepatic cells are unable to excrete the medium in sufficient concentration to permit shadows to be cast on the radiographic films. Cholecystography is also contraindicated when the renal function is impaired.

Oral cholecystography (Graham-Cole test) remains the most satisfactory method of visualising the gall-bladder (figs. 1053, 1054, and 1055):



FIG. 1053.—A normal cholecystograph.



FIG. 1054.—Same after a fatty meal.



FIG. 1055.—Non-opaque stones in the gall-bladder rendered visible by the medium during cholecystography.

Telepaque (Bayer Products Ltd.) is the contrast medium employed most commonly in the British Commonwealth and North America, but Biloptin and Solubiloptin (Schering (Germany)) and Orabilix (France) are also used. On the evening before the radiological examination, the patient partakes of a light supper of non-fatty food between 9 and 10 p.m. After this the patient swallows the six uncrushed tablets, one at a time, and drinks water in moderation until bed-time. On the following morning nothing to eat or drink is allowed until the radiological examination has been made. After two or three films have been exposed, the patient is given a meal of eggs and bacon, bread and butter, and some cream in tea or coffee. Alternatively 'Prosparol', a 50 per cent. emulsion of arachis oil in a dose of two to four tablespoonfuls is equally effective in stimulating gall-bladder contraction. One hour later another film is taken, in order to ascertain if the gall-bladder contracts after this fatty stimulus. By giving Biloptin in the evening and Solubiloptin on the next morning, followed three hours later by radiography, it is hoped to visualise the bile ducts as well as the gall-bladder.

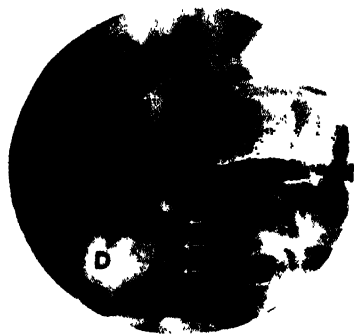


FIG. 1056.—Stones in the common bile duct visualised after intravenous cholangiography. Note the opaque medium in the duodenum (D), revealing that bile is entering the duodenum freely. (Dr. M. Israelski, Leamington Spa.)

Intravenous cholangiography (Biligradin¹) permits radiological visualisation of the intra- and extrahepatic bile ducts (fig. 1056).

The contents of the ampoule (20 ml.) must be injected very slowly, taking at least three minutes. Biligradin appears in the bile in a few minutes, achieving a concentration 30 to 100 times that of the blood, thus permitting radiological visualisation of the bile passages: here lies its peculiar and inestimable value. For the gall-bladder, it is inferior to oral cholecystography; nevertheless, it is indicated for that purpose when the patient is liable to vomit, or when absorption from the alimentary tract is impaired by diarrhoea. Intravenous cholecystography possesses another clear advantage when early confirmation or exclusion of acute gall-bladder disease is required urgently for purposes of differential diagnosis.

¹ Iodipamide, known in the U.S.A. as cholografin.

Safety Precautions.—It has given rise to severe reactions, the leading symptoms of which are feeble or imperceptible pulse, stertorous respirations, cyanosis, and perhaps incontinence of urine. A test dose is usually given first, from a supplementary small ampoule which is supplied with each package by the manufacturers. The contents of this are injected into a vein the day before (or, in urgent cases, an hour before) the examination, in order to ascertain if any reaction is likely to occur. The treatment of a reaction is to administer nikethamide, antihistamine drugs, and oxygen.

The best method of visualising the bile ducts is to give an oral contrast medium in the evening, followed by an intravenous contrast medium in the morning, and to employ tomography¹ as a routine. This allows demonstration of the ducts free from overlying gas shadows (Carstairs).

Per-operative Cholangiography.—A cassette tunnel is placed beneath the patient (fig. 1071) at a level that will include the entire biliary tract. The cystic duct is opened and a polythene or ureteric catheter is passed through into the common bile duct for 3 cm. (A baby's umbilical catheter is most suitable for this purpose.) A ligature tied round the cystic duct and catheter prevents leakage. The catheter is filled with normal saline prior to insertion (bile should be aspirated after insertion), so that there are no air bubbles present which would have a similar X-ray appearance to a radiolucent gall-stone. Instruments and packs which might obscure the radiograph are removed, and then two injections are given of 5 ml. of 30 per cent. Hypaque or Urografin (sodium diatrizoate or diatrizoate salts). An X-ray exposure follows each 5 ml.; respiration being interrupted to obviate any movement which would blur the picture. A normal cholangiograph (fig. 1058) is sufficient evidence that exploration of the common bile duct is unnecessary. When the medium fails to pass into the duodenum, and especially if the ducts concerned are dilated or if a filling defect is visualised (fig. 1058), choledochotomy and exploration of the interior of the common bile duct is the first

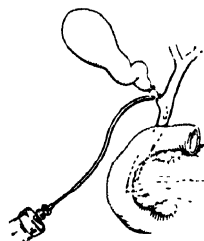


FIG. 1057.—Per-operation cholangiography. Method of introducing the medium.



FIG. 1058.—Typical per-operation cholangiographs. From left to right: (I) Normal cholangiograph. (II) Spasm of the sphincter of Oddi. Dilatation of the common bile duct. No medium enters the duodenum. (III) Showing filling defect due to a calculus at the lower end of the common bile duct (inset, the calculus, actual size).

C.B.D. = common bile duct; W. = duct of Wirsung; D. = medium in the duodenum.

consideration. In about 20 per cent. of cases the medium flows along the duct of Wirsung. This is usually physiological and not pathological.

Per-operative Confirmatory Cholangiography.—Surgeons who rely upon purely clinical indications for choledochotomy to look for stones (p. 835), may perform

¹ Tomography = a method of placing one given plane into sharp focus while blurring others.

a cholangiogram via a catheter or the T-drainage tube (p. 839), before closing the abdomen to make sure that all stones have been removed, and that there is no obstruction to the flow of bile into the duodenum.

Post-operative Cholangiography is performed some ten to fourteen days after choledochotomy via the T-tube. The absence of stones and a normal flow of bile into the duodenum indicates that the T-tube can be removed (p. 839).

Transhepatic cholangiography is described on p. 845.

CONGENITAL ABNORMALITIES OF THE GALL-BLADDER AND BILE DUCTS

A congenital anomaly of the biliary tract is found in 10 per cent. of necropsies.

Absence of the Gall-bladder.—Occasionally the gall-bladder is absent in man. Stones have been found in the common bile duct in nearly 50 per cent. of cases.

The Phrygian cap (fig. 1059(a)) is the commonest abnormality of the gall-bladder. It is present in 2 to 6 per cent. of cholecystographies, and unless the abnormality is known, it will be mistaken for a pathological deformity of the organ. The probable

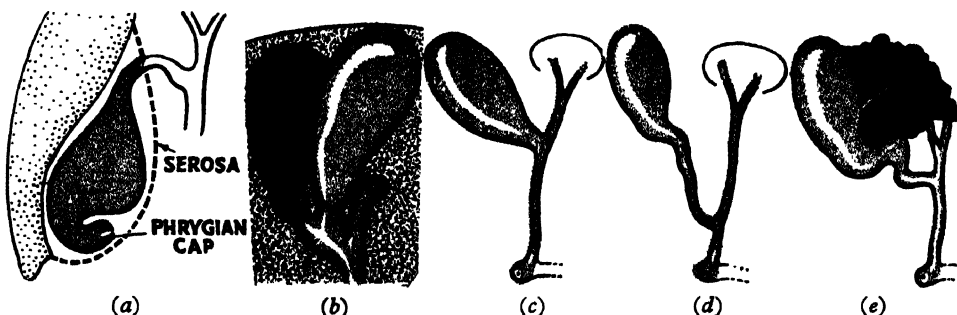


FIG. 1059.—Some anatomical anomalies of the gall-bladder and bile passages.

cause is failure of the caudal portion of the embryological diverticulum to become tubular. Gall-stones are more frequent than in normally formed gall-bladders.

Floating Gall-bladder.—Normally between one-quarter to one-third of the circumference of the gall-bladder lies in a shallow bed on the under-surface of the liver. Occasionally the gall-bladder has a mesentery, which simplifies cholecystectomy, but renders the organ liable to undergo torsion (p. 825).

Double Gall-bladder.—On rare occasions, due to bifurcation of the cystic bud, the gall-bladder is double. One of the twins may be intrahepatic (fig. 1059(b)). Most cases have been diagnosed at operation. Cholecystography often fails to be diagnostic, because the shadows of the two gall-bladders are superimposed. Since both the gall-bladders are diseased, double cholecystectomy should be performed.

Absence of the Cystic Duct.—The gall-bladder opens directly into the common bile duct (fig. 1059(c)). Injury of the common duct is particularly liable to occur when cholecystectomy is performed in a patient with this abnormality. The only way of avoiding the accident is to divide the neck of the gall-bladder and accurately to close the opening in the common duct with sutures.

Abnormally Long Cystic Duct.—Sometimes the cystic duct opens into the common duct near the pancreas (fig. 1059(d)). A particular misfortune of this type of abnormality is that a portion of the cystic duct may be left behind during cholecystectomy, and as a consequence symptoms return, or are unrelieved.

An accessory cholecystohepatic duct may open into the gall-bladder (fig. 1059(e)) and cause leakage after cholecystectomy (p. 841). In the very rare anomaly where the right hepatic duct terminates in the gall-bladder, cholecystostomy or partial cholecystectomy should be performed, so as to preserve the anomalous duct and maintain a free exit for its contents along the anatomical channels.

The Phrygian cap refers to hats worn by peoples of Phrygia, an ancient country of Asia Minor. The cap was rather like a *liberté cap* of the French Revolution.

CONGENITAL ATRESIA OF THE BILE DUCTS

Ætiology.—The probable explanation of many of the varieties is that recanalisation fails to occur in fetal life (p. 818).

Pathology.—One of the anomalies shown in fig. 1060 is present. The extrahepatic bile ducts may have no lumen, or a tiny lumen blocked with epithelial debris, and

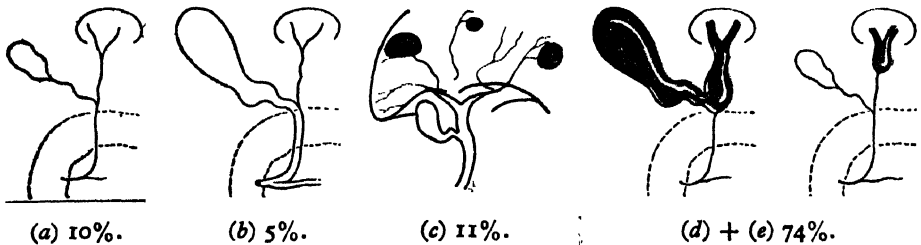


FIG. 1060.—Types of congenital obstruction of the bile ducts.

perhaps biliary sand. In (c), a few have lumina connected with scattered bile-filled cysts.

At necropsy the liver, which is greatly enlarged and stained dark green, exhibits varying degrees of biliary cirrhosis proportional to the time the patient has survived. It is a surprising fact that a few infants possessing extrahepatic ducts with no lumina whatever have lived to be eight to ten months old.

Clinical Features.—Sometimes a slight icteric tinge is present at birth; more usually jaundice appears within two or three days thereof; occasionally it is delayed for up to several weeks. Unless the atresia can be remedied the jaundice becomes deeper and deeper; the urine is bile-laden and dark brown. The first meconium is often normal, i.e. greenish-black in colour; it is said to be formed during the fourth month of foetal life. Thereafter the stools are white or clay-coloured; after two weeks they may become faintly yellow, as in cases of profound jaundice a small amount of bile pigments is excreted by the intestines. Gradually the liver enlarges, and on palpation feels unduly hard. Nutrition is fairly well maintained, especially if the baby is given feeds containing but little fat. Unrelieved, death usually results in three to six months.

Differential Diagnosis.—The condition has been confused with (a) erythroblastosis foetalis, which can be eliminated by an examination of the blood; (b) congenital spherocytosis, which can also be eliminated by appropriate tests (p. 781); (c) jaundice of hæmolytic sepsis; (d) congenital syphilis; (e) homologous serum hepatitis (serum jaundice), which can occur in patients who have had blood-transfusion or plasma infusion, and (f) neonatal hepatitis. The last, which is now more recognised, is the most diagnostically difficult to differentiate from atresia. In neonatal hepatitis the baby is likely to be more ill than is the case in atresia; also the stools are not without pigment. Liver-function tests need to be supported by the findings of a liver biopsy.

Treatment.—Pre-operative measures include the correction of dehydration, if present, and one or two small blood-transfusions. Vitamin K₁ is always necessary to combat prothrombin deficiency in patients with obstructive jaundice. A tetracycline should be administered before and after operation.

Operation.—The anæsthetic of choice is open ether. As in all cases of obstructive jaundice, the use of muscle relaxants must be forbidden. A midline incision affords adequate exposure, though the enlarged liver impedes dissection of the minute ducts.

Operative cholangiography can be employed to ascertain the site of the stricture when this is not apparent. In (a) and (b), even when the ducts appear to be fibrous cords, it is always well worth while ascertaining whether a tiny lumen is present in the main duct. After introducing a needle into the gall-bladder, or a duct, syringing sometimes has proved successful in freeing obstructed ducts of inspissated material. With this exception, the operative measure should be as follows:

Gall-bladder present. Cystic duct communicates with main ducts. Atresia lower end of common bile duct.	Cholecystojejunostomy + jejuno-jejunostomy (fig. 1109).
Gall-bladder functionless or absent. Atresia lower end of common bile duct.	Choledochoduodenostomy (fig. 1082).
Common hepatic duct alone present, and contains bile.	Hepaticodochojejunostomy + jejunojejunostomy.

In suitable cases the first of the procedures listed would appear to be ideal. Unfortunately late stenosis of the cystic duct often mars the final outcome. It should therefore be performed only when the cystic duct is greatly distended.

When the atresia belongs to types (b), (c) and (e), it is conceded generally that the case is hopeless. Sterling has had one success in a case of type (c) by inserting silver tubes into the liver substance through the hilum, and after mobilising the gall-bladder anastomosing the open fundus to the liver around these tubes. Tubes were also inserted into the right lobe and brought out through the abdominal wall.

Congenital choledochus cyst is due to a specific weakness in a part, or the whole, of the wall of the common bile duct, and is classified accordingly as cystic dilatation, diverticulum, and choledochoceles. Contrary to expectation, only occasionally does the distension involve the cystic and hepatic ducts and the gall-bladder is never distended, viz.:

This is a rare condition affecting females four times as commonly as males, Japanese being more prone to the condition than other races. Unlike congenital atresia of the bile ducts, the symptoms and signs seldom become manifest before the age of six months (in only 50 per cent. of cases do symptoms appear before the age of twenty years). A choledochus cyst may contain as much as 1 to 2 litres. The cyst wall shows inflammatory changes and an absence of lining epithelium.

Clinical Features.—There are attacks of jaundice of the obstructive type. Between the attacks the patient's colour is normal and the general health good. Usually the attacks are accompanied by upper abdominal pain and pyrexia due to infection. In 90 per cent. of cases a swelling is detected in the upper abdomen. Untreated, the condition ultimately proves fatal, due to ascending cholangitis, biliary cirrhosis, or diffuse peritonitis following rupture of the cyst.

Treatment.—For cystic dilatation, choledochocystojejunostomy, and for diverticulum and choledochocoele excision of the cyst, is recommended.



Aberrations in the Arterial Supply of the Gall-bladder.—The cystic artery is a branch of the right hepatic artery. Usually the cystic artery is given off *behind* the common hepatic duct, and then runs along the cystic duct to the gall-bladder (fig. 1061 A). Occasionally there is an accessory cystic artery that arises from the gastro-duodenal artery (fig. 1061 B). In some instances (25 out of 161 cases, Flint) the right hepatic artery and/or the cystic artery cross in front of the common bile duct and the cystic duct (fig. 1061 C), in which case the right hepatic artery is in an extremely vulnerable position. Should it be ligated, necrosis of much of the right lobe of the liver is likely to ensue. The right hepatic artery in its anterior position may compress the common bile duct. The most dangerous anomaly of all is when the main trunk of the hepatic artery takes a tortuous course in front of the origin of the cystic duct (fig. 1061 D). There is a high incidence of

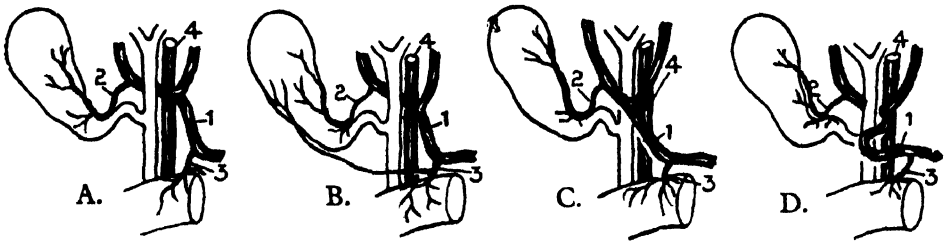


FIG. 1061.—A, Usual arrangement of the arterial supply to the gall-bladder; B, C, and D, variants. 1. Hepatic artery. 2. Cystic artery. 3. Gastro-duodenal artery. 4. Portal vein.

fatal infarction in accidental ligation of the common hepatic artery, but chances of recovery in this and right hepatic ligation are improved by giving penicillin and streptomycin.

INJURIES

1. **Traumatic rupture of the gall-bladder or the bile passages** is a rare injury, usually the result of a run-over accident. The physical signs are identical with those of rupture of the small intestine, with one notable addition—unmistakable jaundice, which appears within two or three hours of the accident in 65 per cent. of cases. When the abdomen is opened, bile is found within the peritoneal cavity.

Treatment of the various lesions is depicted in fig. 1062, but when there is a small hole in the gall-bladder (such as can occur from a penetrating wound), suture of the rent with drainage of the peritoneal cavity is indicated.

2. Injuries of the Bile Ducts incurred during Operation (p. 841).

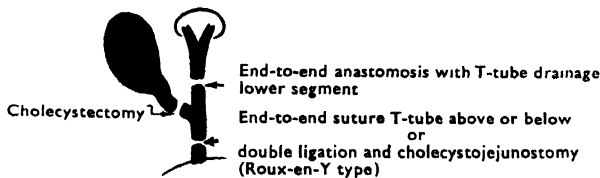


FIG. 1062.—Methods of treating early complete tears of the biliary tract.

TORSION OF THE GALL-BLADDER

Torsion can occur in those rare instances where the gall-bladder has a comparatively long mesentery—the so-called 'floating' gall-bladder. Usually the patient is a female over sixty years of age. There is a sudden agonising pain, with vomiting and shock. If the gall-bladder becomes gangrenous or ruptures, the pain passes off instantaneously, but signs of diffuse peritonitis soon follow. Cholecystectomy is the treatment.

GALL-STONES (Cholelithiasis)

Gall-stones vary in composition. Sometimes they consist entirely of cholesterol, or of calcium bilirubinate. More often they are composed of alternating layers of cholesterol and calcium bilirubinate, or cholesterol and calcium carbonate and phosphate. Protein is another constituent.

Etiology.—Certain factors are likely to be responsible for the formation of gall-stones. These are: (1) Metabolic; (2) Infective; (3) Bile stasis.

(1) *The metabolic factors* are concerned with the solubility of cholesterol in bile salts. The normal ratio of bile salts to cholesterol is 25:1, and the critical level for precipitation of cholesterol is 13:1. The concentration of bile-salt may be diminished by

dietary factors such as feeding sucrose and avitaminosis A. Gall-bladder infection causes a local fall through bile-salt reabsorption. As regards cholesterol, a high biliary level rarely occurs (only experimentally by massive cholesterol feeding and injection). A high serum cholesterol level (hypercholesteræmia) is not such a significant factor as was formerly believed.

Bile pigment stones commonly follow the *excessive hæmolysis* that occurs in types of hæmolytic anæmia, e.g. acholuric jaundice and malaria.

(2) *Infection*.—Organisms are frequently found in gall-stones (streptococci, coliforms, typhoid, actinomyces), and have long been blamed as the cause. They reach the gall-bladder via the blood-stream, from a focus of infection elsewhere (tonsils, bowel, dental tartar, and caries) or from the bowel via the lymphatics, and cause an exudation from, and an exfoliation of, the mucosal cells to act as a nidus on which the stone forms. There is some truth in Moynihan's aphorism that 'a gall-stone is a tombstone erected to the memory of the organism within it'. *Infestation* with a round worm (*Ascaris*) is a cause of stones being formed around ova in the common bile duct.

(3) *Bile Stasis*.—Without some kind of stasis, stones would not grow to the sizes commonly found. They would be voided while still in a particulate or gravel form. Stasis is usual during pregnancy and this, rather than hypercholesteræmia, may explain the increased incidence of stones in multipara. Also in the gall-bladder, the incoming bile tends to form layers owing to differences in specific gravity. At the interface between layers, the solubility of cholesterol is disturbed.

Stone Growth.—Stones begin to form as minute amorphous spheres (microspheruliths), which aggregate to form larger spheres. With cholesterol there is a gradual change in the stone from an amorphous to a radiating crystalline form.

Types of Gall-stone.

(1) **A cholesterol stone** is usually solitary (the cholesterol 'solitaire'). It is oval or rounded in shape, up to 1.25 cm. in diameter, or larger (fig. 1063). It

FIG. 1063.—Solitary gall-stone from a gall-bladder (actual size).


is light in weight as well as in colour. In its absolutely pure state it is very pale yellow in colour and, like tallow, it is feebly translucent. More often bile pigments are deposited within it. On section its crystalline interior will be found to radiate

Cholesterol stones, which make up some 6 per cent. of all gall-stones, are believed to be formed primarily in aseptic static bile (in a 'stasis' gall-bladder). They tend to reside in Hartmann's pouch. A brood of mixed (secondary or inflammatory) gall-stones (see below) may be formed later, or a mixed covering of pigment and cholesterol may form around the primary cholesterol stone, viz.

This is known as a combination stone.

(2) **Pigment stones** (calcium bilirubinate) comprise about 12 per cent. of the total. Black in colour, sometimes with an iridescent lustre, they are frequently found in the common bile duct, and tend to be the commonest type of gall-stone in Koreans and in the agricultural population of Japan. They range in shape and size from irregular, mulberry or coral-like concretions of about 5 mm. down to sludge particles. Often they are simply soft putty-like masses. On section all pigment stones seem to be amorphous

(3) **'Mixed' stones** constitute the majority (80 per cent.) of gall-stones

found at operation. Nearly always multiple, by mutual pressure or by friction one against the other, they become faceted. Dozens or hundreds of such stones are often present, frequently the gall-bladder being packed to capacity. On section each is found to be laminated—→  The central nucleus may contain epithelial debris and bacteria, which suggests that the cause is inflammatory. Whatever the composition of the central core, alternating layers of cholesterol and calcium carbonate and/or calcium bilirubinate are deposited upon it.

Calcium Carbonate in the Gall-bladder.—‘Lime-water’ bile is revealed in a plain radiograph (fig. 1064) more clearly than if the gall-bladder has been visualised by cholecystography. The opacity is the result of the gall-bladder becoming filled with a mixture of calcium carbonate and calcium phosphate, usually the consistency of toothpaste. The condition always arises in conjunction with chronic infected cholecystitis and cystic duct obstruction, but organisms are rarely grown from the emulsion.



FIG. 1064.—Plain radiograph showing a gall-bladder filled with a thick emulsion of calcium salts. Gall-stones are present also.

Incidence of Gall-stones.—A fat, fertile, flatulent female of fifty is the classical sufferer from gall-stones. Useful as is this clinical memorandum, it should be tempered with the knowledge that cholelithiasis occurs in both sexes, quite often at a much earlier age—even in childhood—and is more common in old age. Stones occur in 20 per cent. of women during the child-bearing period, and in 20 per cent. of *men* and women in old age.

THE EFFECTS AND COMPLICATIONS OF GALL-STONES

Gall-stones are usually found in the gall-bladder, but in 20 per cent. of cases stones will also be present in the bile ducts. Nearly always it is just one stone amongst many which is responsible for the patient's suffering. The effects and complications can be summarised as follows:

1. In the gall-bladder—Silent stones.

- Flatulent Dyspepsia.
- Gall-stone Colic.
- Mucocoele.

- Acute cholecystitis {
 - perforation→peritonitis.
 - gangrene.
 - penetration→fistula.
 - empyema.
- Chronic cholecystitis→acute cholecystitis.
- Carcinoma.

2. In the bile ducts

- Obstructive jaundice. Liver failure. White bile.
- Acute or recurrent pancreatitis.
- Cholangitis.

3. In the intestine

- Acute intestinal obstruction (fig. 928).

Silent Gall-stones.—It is possible for a calculus or calculi to be present in the gall-bladder and give rise to no symptoms during a long lifetime. At least 8 per

cent. of necropsy subjects of over fifty years of age have gall-stones in the gall-bladder that have not contributed to the cause of death. As 10 to 20 per cent. of gall-stones are radiopaque, they may be discovered accidentally on X-ray for another condition, e.g. by an X-ray to determine whether a rib is fractured. Cholecystectomy should be advised on account of the likelihood of complications, particularly when a cholecystogram reveals a non-functioning gall-bladder, or the stones are large and obviously fill a gall-bladder devoid of function. A follow-up of 112 patients with symptomless gall-stones in the gall-bladder found on routine examination at the Mayo Clinic showed that 51 developed symptoms within a few years.

Flatulent Dyspepsia.—Apart from gall-stone colic, stones in the gall-bladder typically give rise to a reflex flatulent dyspepsia. The symptoms include a feeling of fullness after food, belching, and heartburn—all worse after a large meal or one containing much fat. A cholecystogram may reveal a non-functioning, a poorly-functioning, or a normally-functioning gall-bladder, both as to its ability to concentrate the medium, and to empty. Patients with functioning gall-bladders always require a thorough investigation of other organs. Especially is it necessary to rule out œsophageal hiatus hernia (p. 715) and, as far as possible, chronic pancreatitis (p. 854), before cholecystectomy is performed. The dyspepsia can continue for a long time—perhaps for years—until one day a comparatively small stone enters the cystic duct (colic) or a larger one occludes the neck of the gall-bladder (mucocele or acute cholecystitis).

Gall-stone Colic.—Suddenly the patient experiences excruciating pain across the upper part of the abdomen.¹ The pain may shoot to the back or between the shoulder-blades. In severe cases it ‘doubles her up’, and she rolls in agony on the floor. The attack, which lasts for upwards of two hours and is usually accompanied by vomiting and retching, often passes off nearly as suddenly as it came. Colic usually occurs at night, as in the horizontal position small calculi tend to congregate at the neck of the gall-bladder.

Occasionally the acute pain lasts longer, and the patient abandons hope. Sir Walter Scott, after an attack lasting many hours, ‘called his family, and bidding them farewell, turned his face to the wall to await death’. Nevertheless, next morning he was up betimes and in fair spirits.

Physical Signs.—It is impossible to examine the abdomen satisfactorily during an attack of colic, but soon after the attack rigidity and tenderness can usually be elicited in the right hypochondrium. Exceptionally palpation reveals a tender, enlarged gall-bladder.

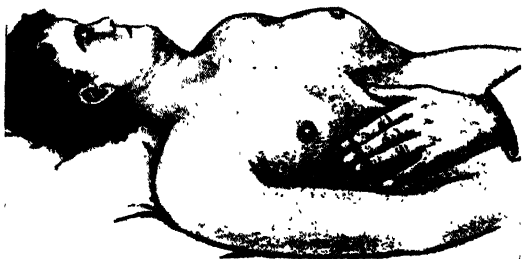


FIG. 1065.—Murphy's sign (Moynihan's method).

Murphy's Sign.²—If continuous gentle pressure is exerted over the right hypochondrium (fig. 1065) while the patient takes a deep breath, there is a ‘catch in the breath’ just before the zenith of the inspiration.

¹ The biliary apparatus is bilaterally innervated, and pain may be under the right costal margin and left costal margin. In 4 per cent. of cases it is under the left costal margin alone.

² This sign is also named after Bernard Naunyn, 1839–1925, Professor of Medicine, Strasbourg, who described it in 1890, thirteen years before Murphy.

The Mayo Clinic, Rochester, Minnesota, was founded by the Mayos—father and sons—in 1889. Sir Walter Scott, 1771–1832. Scottish poet, and master of the romantic historical novel. John B. Murphy, 1867–1916. Professor of Surgery, North-western University, Chicago.

Jaundice follows an attack of gall-stone colic in 18 per cent. of cases, and mostly is due to a stone obstructing the common bile duct.

Radiography.—Because the function of the liver is depressed, cholecystography should not be undertaken for at least three or four days after an attack of gall-stone colic, and only then if the patient is free from jaundice. On the other hand, there is no objection to having a plain radiograph taken of the upper abdomen. In 10 to 20 per cent. of cases gall-stones are radiopaque (fig. 1066).



FIG. 1066.—Plain radiograph showing stones in the gall-bladder and one in the cystic duct.

Treatment:

(a) **Of Gall-stone Colic.**—Morphine is undesirable as it causes spasm of the sphincter of Oddi (p. 853). Pethidine,² though widely used, also causes spasm. It is essential to give an antispasmodic drug (e.g. atropin or Phenergan (promethazine hydrochloride)) before or with the analgesic drug. In most cases heat somewhat relieves the pain, and in old-standing cases a brown pigmentation of the skin over the right hypochondrium tells of frequent hot applications.

(b) **Subsequent Treatment.**—Once a patient has had gall-stone colic, no question arises as to the treatment that should be recommended: it is *cholecystectomy*, unless the patient is very old or otherwise enfeebled, when *cholecystostomy* can be substituted. In either case it is essential to examine the common bile duct by all means available for the presence of additional calculi and, if such are present, to remove them (pp. 819, 833).

Mucocele of Gall-bladder.—A mucocele develops as the result of obstruction of the neck of a sterile gall-bladder by a single stone; the contained bile is absorbed and replaced by mucus secreted by the gall-bladder. The organ is thin-walled and translucent and may contain a pint of mucus. The treatment is cholecystectomy.

CHOLECYSTITIS

Ætiology.—Cultures from excised gall-bladders and their contents show:

Contents.—Bacteria present in 35 per cent. of cases. *Wall.*—Bacteria present in 65 per cent. of cases. The commonest infecting organisms are *Esch. coli*, *streptococci*, *Salmonella paratyphi B*, and *staphylococci*, in that order.

ACUTE CHOLECYSTITIS

Acute Obstructive Cholecystitis.—If a stone impacts in Hartmann's pouch or the cystic duct (fig. 1067), the gall-bladder, especially if already the seat of chronic cholecystitis, becomes intensely inflamed with its mucous membrane swollen, and the wall thickened. Occasionally gangrene occurs in places. The subsequent events are:

1. When a certain degree of distension of the gall-bladder has been

²Pethidine is known in Canada as demerol, and in the U.S.A. as demerol hydrochloride. Should the patient be taking a mono-amine oxidase-inhibitor drug for depression, pethidine is contraindicated as collapse can occur from a severe fall of blood pressure. Morphine must be also used with great care.

reached, the mucous membrane tends to be lifted away from the sides of the stone and as a consequence the stone may slip back into the body of the gall-bladder, viz. —————→

and any mucoïd (from a mucocele—see above) or muco-purulent contents of the gall-bladder escape by way of the cystic duct.

2. Less frequently the impaction persists and an empyema (or pyocele) of the gall-bladder results (p. 832).



3. On rare occasions the distended, inflamed gall-bladder perforates. Doubtless the infrequency of perforation is due to the thickened walls of an organ that has long been the seat of chronic cholecystitis.

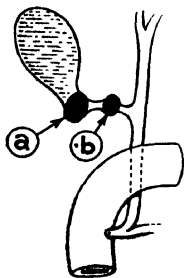


FIG. 1067.—The sites of impaction of a gall-stone in acute obstructive cholecystitis: (a) In Hartmann's pouch. (b) In the cystic duct.

Perforation of the Gall-bladder.—The site of perforation is either at the fundus, which is farthest away from the blood-supply, or, less commonly, at the neck from pressure necrosis of an impacted calculus. The sequelæ are:

(a) **Local Abscess.**—On account of the present, and probably past, attacks of cholecystitis, there are adhesions between the gall-bladder, the greater omentum, and the parietal peritoneum. Consequently, when an infected, obstructed gall-bladder perforates the usual outcome is a local abscess.

(b) **Perforation into the general peritoneal cavity** is uncommon, and the patient is usually a man. Diffuse peritonitis supervenes readily and rapidly, and the mortality is about 50 per cent. (Ellis). This catastrophe occurs in only 0.5 per cent. of cases undergoing conservative treatment for acute cholecystitis.

Internal Fistulæ of the Gall-bladder are discussed on p. 841.

Acute non-obstructive cholecystitis is less frequent than the obstructive variety. All grades of acute inflammation, from catarrhal to gangrenous cholecystitis, occur. In fulminating cases *Cl. welchii* may be implicated. Typhoid and paratyphoid cholecystitis can occur. Perforation is exceptional, save in acute typhoid cholecystitis.

Clinical Features of Acute Cholecystitis.—The onset is sudden, and pain—agonising in the obstructive variety, more gradual in the non-obstructive—is located mainly in the right hypochondrium. Severe nausea and vomiting are features in the early stages. Pyrexia, sometimes to 101° F. (38° C.) or more, is usual; occasionally fulminating cases are ushered in by rigors.

On examination tenderness and rigidity are found in the right hypochondrium. If the patient can be persuaded to relax, a mass consisting of the inflamed gall-bladder with adherent greater omentum attached may be felt, particularly in obstructive cases. In mucocele of the gall-bladder pyrexia is absent and a piriform swelling can be palpated in the right hypochondrium.

Differential Diagnosis.—The differential diagnosis between high retrocæcal acute appendicitis and acute cholecystitis and a leaking duodenal ulcer is often difficult, and when there is doubt, conservative treatment (see below) is not advised. Acute pancreatitis must be excluded by all means possible (p. 849). It is also necessary to eliminate acute pyelonephritis, and other infections of the right kidney. In relevant cases an electrocardiograph should be taken to differentiate acute cholecystitis from coronary thrombosis.

Boas's sign, if positive, is sometimes most helpful in distinguishing acute cholecystitis from other conditions. There is an area of hyperæsthesia between the ninth and the eleventh ribs posteriorly on the right side.

Radiography.—A plain radiograph in an adult is usually negative. In children the calculi are usually opaque (Forshall). The rare presence of gas¹ in the gall-bladder is due to infection with *Cl. welchii* and operation should be performed without delay. Oral cholecystography is usually carried out when the acute phase is subsiding. A non-filling gall-bladder supports the diagnosis of cholelithiasis.

Treatment:

(a) **Conservative Treatment followed by Cholecystectomy.**—Experience shows that in more than 90 per cent. of cases the symptoms of acute cholecystitis subside with conservative measures. Non-operative treatment is based upon four principles:

1. *Rest to the inflamed gall-bladder* and the biliary and pancreatic systems by gastric aspiration for three to five days. Nothing is given by mouth. Fluid and electrolyte balance are maintained by continuous intravenous dextrose-saline solution, with the usual precautions (p. 90).

2. *Sedation.*—The recommendations are given on p. 852.

3. *An anticholinergic drug* is given to reduce gastric and pancreatic secretion, and therefore loss of electrolytes by aspiration, and to relax the sphincter of Oddi. Propantheline is probably the most satisfactory for this purpose.

4. *Antibiotics* of the tetracycline group reach the interior of the gall-bladder *via* the blood-stream even in the presence of occlusion of the cystic duct. In the initial stages doses of 1 to 1.5 G. b.d. are given in the intravenous infusion, though this method is liable to precipitate thrombophlebitis (drip-arm). Later these drugs may be given intramuscularly, but oral administration should be commenced as soon as the patient is permitted to take fluids by mouth.

5. *Subsequent Management.*—When the temperature, pulse, and other physical signs show that the inflammation is subsiding (usually by the third day), after the bowels have been emptied, e.g. by a suppository, the gastric aspiration tube is removed, and, commencing with flavoured dextrose drinks, fat-free clear fluids are given for one day, and a soft fat-free diet on the next. A full fat-free diet is then given, and the patient encouraged to walk. No fats should be given during convalescence. Eight to ten weeks after the acute symptoms have subsided *cholecystectomy* is carried out.

Conservative treatment is not advised (a) when there is uncertainty about the diagnosis, e.g. when early high retrocaecal appendicitis or a leaking duodenal ulcer cannot be excluded, (b) in typhoidal cholecystitis, because of the frequency of perforation in these cases.

Conservative treatment must be abandoned: If the pain and tenderness spreads across the abdomen and the pulse-rate rises, cholecystectomy should be undertaken forthwith. In the very ill and the elderly patient it

¹ The causes of air (or gas) in the bile ducts or the gall-bladder are: (i) following operative choledcho-duodenostomy (p. 840), (ii) following cholecyst-duodenostomy, or a stone perforating into the duodenum (p. 841), and (iii) rarely, the presence of gas-forming organisms.

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may be advisable to limit the operation to cholecystostomy¹—a dependable safe operative procedure (p. 835).

When a gall-bladder perforates into the general peritoneal cavity, urgent drainage of the peritoneal cavity and the gall-bladder is imperative; after such measures, performed promptly, 60 per cent. of the patients recover. When operation is delayed the mortality approaches 100 per cent.

(b) **Routine Early Operation.**—Some surgeons advocate urgent operation as a routine measure in cases of acute cholecystitis. Provided the operation is undertaken within forty-eight hours of the onset of the attack and excellent operating facilities are available, good results are obtained, but no better than those that accrue from the delayed method described above, which is associated with fewer operative injuries to the main ducts.

It is not proposed here to enter into a controversy on the merits and demerits of early and delayed operation for acute cholecystitis, but to state categorically that the most dangerous period for operation is between the seventh and fourteenth days of the attack. Should operation become imperative during this period, when the ducts are often obscured by œdema and immature fibrous tissue and the liver function is at a low ebb, it is prudent to perform a simple cholecystostomy (p. 835) rather than a difficult cholecystectomy.

Empyema of the Gall-bladder.—This is a curious condition in which an inflamed, obstructed gall-bladder becomes filled with creamy 'pus'. This 'pus' is really a cream of calcium carbonate, cholesterol crystals, and epithelial cells, and is usually sterile on culture. The treatment is cholecystectomy.

Chronic Cholecystitis.—The gall-bladder wall is thickened, with loss of the normal mucous membrane pattern. Stones may or may not be present. Many of these gall-bladders are examples of the cholecystoses (below). The patients may present with biliary dyspepsia and be diagnosed by cholecystography, or they develop acute cholecystitis.

Typhoid Gall-bladder.—Typhoid carriers 'harbour' living *Salmonella typhi*² in their gall-bladders and their gall-stones. The organisms are discharged into the alimentary tract from time to time, and are present in the fæces. Cholecystectomy in such cases may rid the patient of the infection, and the community of a potential danger. The patient must be 'barrier nursed', and given a course of ampicillin. In conjunction with the Public Health Authorities, the patient is kept in an isolation hospital until it has been proved over a sufficiently long period that the fæces are *Salmonella typhi* free.

THE CHOLECYSTOSES

(Cholesterosis, Polyposis, Adenomyomatosis, Cholecystitis Glandularis Proliferans)

This is a not uncommon group of conditions affecting the gall-bladder in which there are chronic inflammatory changes with hyperplasia of all the tissue elements. Endocrine, metabolic, and dyskinetic factors (p. 843) have been suggested as the cause of the hyperplasia. Malignant change does not occur.

¹ Elective cholecystectomy is still indicated in due course, in suitable patients. Cholecystostomy may relieve all symptoms, but some patients continue to suffer, and they may have an intermittent mucopurulent discharge from the drainage wound.

² 'Typhoid Mary', a cook-general who passed *Salmonella typhi* in her fæces and urine, was responsible for nearly a score of epidemics of typhoid in and around New York City.

Cholesterosis (*syn.* **Strawberry Gall-bladder**).—In the fresh state the interior of the gall-bladder looks something like a strawberry; the yellow specks (submucous aggregations of cholesterol crystals and cholesterol esters) correspond to the seeds (fig. 1068). The presence of macrophages stuffed with the esters (foam cells) indicates that the condition is inflammatory in nature and not, as previously thought, an expression of hypercholesteræmia and cholesterol excretion. Cholesterosis *per se* is symptomless, and cannot be diagnosed by cholecystography. Symptoms, if any, are generally due to associated gall-stones, cholecystitis, or incompetence of the pyloric sphincter. Cholesterosis is often accompanied by one or more cholesterol stones, supposedly derived from the deposits in the mucous membrane.



FIG. 1069.—Cholesterol polyposis
(D. J. Oakland, F.R.C.S., Hereford.)

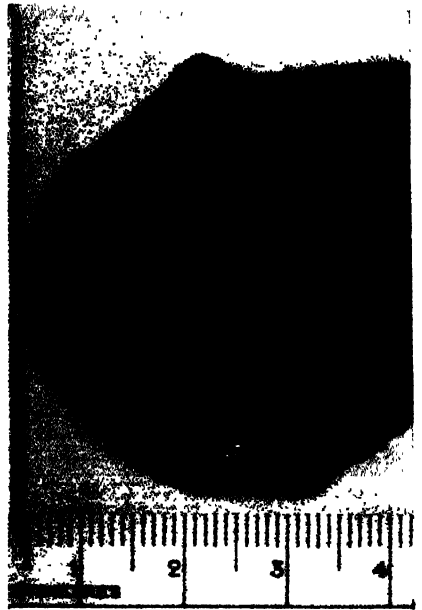


FIG. 1068.—Strawberry gall-bladder.

bladder. The shadows, which are adjacent to the wall of the gall-bladder, remain constant in position and in relation to one another in all films of the series. Histologically, cholesterol polyposis is similar to the cholesterol-laden projections of the strawberry gall-bladder, but the lesions are much less numerous and are relatively gigantic. The treatment is cholecystectomy.

Cholecystitis glandularis proliferans (polyp, adenomyomatosis, and intramural diverticulosis). Fig. 1070 summarises the varieties of this condition. A polyp of the mucous membrane is fleshy and granulomatous. All layers of the gall-bladder wall may be thickened, but sometimes an incomplete septum forms which separates the hyperplastic from the normal. Intraparietal 'mixed' calculi may be present which can be complicated by an intramural and, later, an extramural abscess. *Diverticulosis of the gall-bladder* is usually manifest as black pigment stones impacted in the out-pouchings of the normal lacunæ of Luschka. Diverticulosis of the gall-bladder may be demonstrated by cholecystography, especially after the gall-bladder contracts after a fatty meal. The treatment is cholecystectomy.

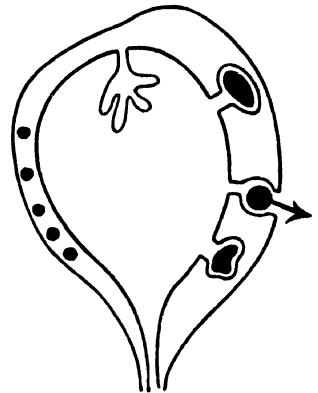


FIG. 1070.—Types of cholecystitis glandularis proliferans (polypus, intramural or diverticular stones, and fistula).

CHOLECYSTECTOMY AND CHOLECYSTOSTOMY

I. Preparation for Cholangiography (p. 821).—Provision is made for per-operative cholan-

giography by placing the patient on an operating table equipped with a radiolucent top, so that X-ray exposures may be made without disturbance (fig. 1071). If the table is not so equipped, a cassette holder and film is placed beneath the patient at the appropriate level. Some tables are designed for the use of an X-ray image-intensifier and television control so that the surgeon has an instant view of the cholangiogram (fig. 1072).

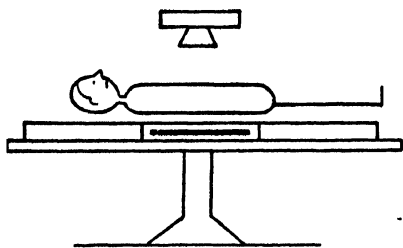


FIG. 1071.—Per-operative cholangiography using a radiolucent table-top.

when ductal stones are obviously present, or when the gall-bladder is filled with one or two large calculi and there are no clinical indications for choledochotomy (see below).

2. Laparotomy.—A right paramedian incision is satisfactory in most cases. It has the advantage of extensibility and is specially useful in those cases with a narrow subcostal angle. In stout patients, with a wide subcostal angle, a Kocher's incision may be used (fig. 1073). After the peritoneum has been opened, the gall-bladder is inspected and palpated.

N.B.—It is essential before commencing a standard cholecystectomy to examine the other abdominal organs. By so doing the writer has discovered an early carcinoma of the stomach, and on two occasions one in the colon. Naturally resection of the growth takes precedence to cholecystectomy, although in favourable cases this may also be possible.

3. Cholecystectomy.—The area is now isolated with packs. If the gall-bladder is greatly distended, it is aspirated through the fundus by means of a trochar and cannula attached to a suction apparatus.

The fundus is then grasped with sponge-holding forceps, thereby sealing the puncture. These forceps, and another pair applied to the neck of the gall-bladder, serve as excellent tractors. Rotation of the liver by a hand passed between the diaphragm and the upper surface, aided by traction on the gall-bladder, is often a great help.

Then the very important dissection to display the junction of the cystic, the common hepatic, and the common bile ducts is commenced.¹

During the course of this dissection the cystic artery is found, and its relation to the common hepatic artery verified (p. 825). The artery is then divided between ligatures at least 5 mm. away from the hepatic duct (fig. 1074). Cholangiography (p. 820) is performed at this stage. The cystic duct is then ligated close to the common bile duct. Forceps

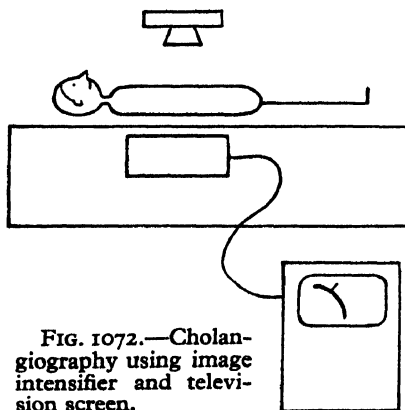


FIG. 1072.—Cholangiography using image intensifier and television screen.

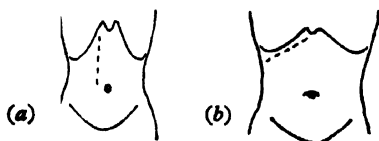


FIG. 1073.—Incisions suitable for displaying the gall-bladder: (a) Paramedian; (b) Kocher's.

¹ 'It is the left hand of my assistant that does all the work' (Moynihan). By splaying the fingers to depress the stomach and duodenum, he displays the biliary ducts, aided also by retraction of the quadrate lobe of the liver.

re applied to the gall-bladder side and the cystic duct is divided (fig. 1075). From below, upwards, the gall-bladder is dissected from its bed with scissors or a diathermy knife, dividing the peritoneum on the gall-bladder, so as to leave a flap on either side or possible suturing over the bed (fig. 1075). The gall-bladder bed is rendered dry



FIG. 1074.



FIG. 1075.



FIG. 1076.

FIG. 1074.—The junction of the cystic, common hepatic, and common bile ducts is displayed and the cystic artery is divided. FIG. 1075.—Division of the peritoneal covering of the gall-bladder. FIG. 1076.—Coagulation of the bed of the gall-bladder.

by coagulation with a diathermy button, and, if feasible, covered with peritoneum (fig. 1076). The gall-bladder having been removed and hæmostasis assured, the abdominal wall is closed with drainage of the operation site by means of a strip of corrugated rubber inserted via separate 'stab' wound.

A cholecystohepatic duct (fig. 1059), is present in about 0.5 per cent. of cases, and is usually the size and colour of No. 60 thread. Its possible presence must be remembered when separating the gall-bladder from its bed, and, if recognised in time, it can be ligated. If avulsed from the liver, a persistent ooze of bile occurs from the ruptured end, which should be sutured.

Indications for Choledochotomy.—If cholangiography is not used to reveal ductal stones or other pathology, the indications for exploratory choledochotomy after the gall-bladder has been removed are: (1) Stones can be felt in the ducts. (2) Jaundice or a history of jaundice, or rigors. (3) A dilated common bile duct (10 mm. diameter or more). This last indication can be misleading, as dilation occurs in old age, and it always remains after a stone which may long since have passed into the duodenum. Choledochotomy is described on p. 838.

Cholecystostomy.—The gall-bladder is displayed through a suitable incision, and the fundus isolated by packs. Two stay sutures are inserted on either side of the fundus, in order to steady the organ, the fluid contents of which are aspirated. The fundus is opened and stones are removed from the interior by Desjardins' forceps, aided, always, by a finger milking up a stone or stones from Hartmann's pouch. Minute calculi are often dislodged by strips of dry gauze passed into the interior. A 6-mm. drainage tube is passed into the gall-bladder, and there retained by a transfixion stitch. The opening in the gall-bladder is closed about the tube. The tube is brought through a portion of greater omentum, which is anchored to the gall-bladder by the original stay sutures. The tube is then brought to the surface through a separate stab incision. The abdominal incision is closed, and the tube is joined by a glass connection to more tubing, which leads to a sterile polythene bag. In seven to ten days' time Hypaque injected down the tube may be seen on X-ray to flow easily into

common bile duct and duodenum if no obstruction exists, in which case removal of tube will be followed by closure of the biliary fistula within a week.

THE DISEASED GALL-BLADDER IN RELATION TO OTHER DISORDERS

The Cardiac Link.—When the heart is the seat of a proclaimed or a latent disorder, vagal reflexes from a diseased gall-bladder may bring about decreased coronary blood flow, arrhythmia or heart-block; or, in other words, the diseased gall-bladder ‘triggers’ myocardial ischaemia; the ‘cholecystic heart’. Accordingly, with both heart and gall-bladder disease, the symptoms of the cardiac disorder may be mitigated, or even completely subjugated, by cholecystectomy. Electrocardiographic abnormalities without other evidence of cardiac disease often revert to normal following removal of a diseased gall-bladder. Likewise, patients with pseudo-angina pectoris have benefited. Sufferers from gall-stones or cholecystitis should be advised to undergo cholecystectomy, not only because of possible improvement of the cardiac condition, but to avoid an operation at a later date, when circumstances may be less favourable.

Saint’s Triad.—(i) Gall-stones, (ii) Diverticulosis of the colon, and (iii) Hiatus hernia frequently coexist. Possibly obesity is the common factor. It is important to find out which lesion is the cause of the patient’s dyspeptic symptoms.

STONE IN THE COMMON BILE DUCT

The bile ducts, as opposed to the gall-bladder, having little or no muscle in their walls are unable to expel stones. When the gall-bladder is chronically inflamed, especially if it is full of stones, the flushing action on the common bile duct by contractions of this little reservoir is feeble; when the gall-bladder is fibrotic, it is nil. In such circumstances, and after cholecystectomy, there are slender chances of any but a tiny calculus being swept into the duodenum by a flow of bile during a period of relaxation of Oddi’s sphincter.

Clinical Features.—It is possible for a gall-stone to be present in the common bile duct for months or even years without disabling the patient; conversely, serious symptoms are liable to supervene at any time. Usually there is a long history of flatulent dyspepsia, not infrequently punctuated by episodes of biliary colic.

Pain.—There is no fundamental difference between the biliary colic described already (p. 828) and that occasioned by a stone becoming impacted in the common bile duct. In the latter instance the pain is likely to be prolonged, severe, and difficult to relieve, and is followed by jaundice.

Jaundice.—Within forty-eight hours the pain is followed by jaundice, the tinge of which ranges from pale lemon to bright orange, and varies from day to day. The skin commences to itch, sometimes intolerably. Elevation of temperature is usually slight, although there may be one or two ‘spikes’ towards the end of a series of attacks of biliary colic (see ‘impaction of a stone’, below). The patient loses weight.

Abdominal Examination.—Usually, soon after the attack of colic, tenderness can be elicited in the epigastrium. As a rule the gall-bladder is impalpable.

Courvoisier’s Law states that if in a jaundiced patient the gall-bladder is palpably enlarged, it is probably *not* a case of stone impacted in the common bile duct, because previous cholecystitis has made the gall-bladder fibrotic. Unless the gall-bladder is very fibrotic a more rational explanation is that, in cases of carcinoma of the pancreas the rise in biliary pressure increases unrelentingly, whereas in calculus obstruction the biliary pressure is intermittent (Smith).

Sometimes it happens that after an attack of biliary colic, followed by fleeting jaundice, the patient remains well for many months. More often than not, after a varying interval another similar attack occurs, and it is sometimes presumed that another gall-stone has entered the common bile duct. But this is not necessarily the case; it is likely that the same stone has again caused obstruction, perhaps a little farther down the duct.

Impaction of a stone can occur in any part of the common bile duct (fig. 1077); severe biliary colic follows and jaundice deepens. Fluctuating jaundice, recurrent pain and intermittent fever with rigors (Charcot's biliary triad) are due to a stone which causes oedema of varying intensity in the adjacent wall of the common duct. The fever and rigors are due to ascending cholangitis¹ (p. 796).

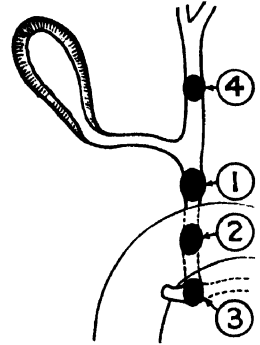
FIG. 1077.—1. Stone impacted in the supraduodenal portion of the common bile duct (commonest site).

2. Stone impacted in the retroduodenal portion of the common bile duct (second most common site).

3. Stone impacted in the ampulla of Vater (about 10 per cent. of cases).

4. Stone impacted in the common hepatic duct (very rare).

Note that the gall-bladder is usually thick-walled and contracted because of chronic cholecystitis.



Atypical Cases:

1. **Jaundice from Pressure of a Stone on the Duct from Without.**—Jaundice can arise from a large calculus in Hartmann's pouch pressing upon the common bile duct.

2. **Jaundice is the First Symptom.**—In about 3 per cent. of individuals jaundice is the first and only symptom.

3. **Stone in the Common Bile Duct without Jaundice.**—A stone or stones may lie dormant in the common bile duct, giving rise only to vague indigestion and perhaps an occasional tinge of jaundice. Intravenous cholangiography is valuable in demonstrating these unsuspected calculi.

Differential Diagnosis.—Calculous biliary obstruction is the most frequent cause of jaundice encountered in surgical practice (fig. 1078). It

is important to differentiate a stone in the common duct from virus hepatitis (p. 796) and carcinoma of the ampulla of Vater (p. 862). Blood is almost always present in the stools in carcinoma of the ampulla, while virus hepatitis results in abnormal liver function tests.

Laboratory investigations are described on p. 793.

Complications.—If the obstruction is not relieved, one of four (three dangerous) sequelæ ensues:

1. **Liver Function becomes increasingly impaired.**—When liver function becomes seriously depressed, as evinced by the finding at operation

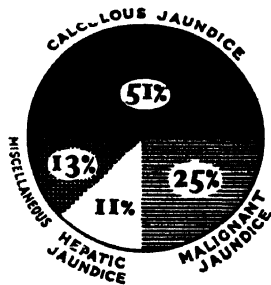


FIG. 1078.—The relative frequency of conditions causing jaundice met with in surgical practice. (After Sir John Bruce.)

¹ In Britain the two common causes for rigors are urinary and biliary obstruction with infection. In equatorial regions, malaria is the commonest cause.

of 'white bile' in the bile passages above the site of an impacted calculus the outlook is serious, but not hopeless, as a brisk recovery often takes place

'White Bile'.—The phenomenon occurs following obstruction of the common bile duct or the common hepatic duct, when the gall-bladder is absent or not functioning. The ducts then contain a secretion mainly of mucus derived from the gland lining the ducts, to which has been added a minute amount of cholesterol, and either only traces of bile salts, or none. The term 'white bile' is a misnomer, since it is not bile derived from the liver but mucus, for the liver fails to excrete due to cellular damage or mounting pressure within the bile ducts; also it is not white but opalescent.

2. Suppurative Cholangitis Supervenes.—This dangerous complication, causing liver failure and death, is, as a result of timely surgical intervention and antibiotic therapy, now comparatively infrequent (p. 796).

3. The gall-stone ulcerates through the wall of the common bile duct and diffuse peritonitis supervenes. This form of bile peritonitis, occurring in a subject with grossly impaired liver function and infected bile, requires immediate operation. The perforation may be minute and difficult to find and sometimes occurs into the retroperitoneal tissues. The treatment is to drain the common bile duct, and also the general peritoneal cavity or the retroperitoneal tissues, as the case may be.

4. On rare occasions (as subsequently revealed at necropsy) a stone in the common bile duct ulcerates into the duodenum, and a natural cure follows.

MANAGEMENT OF BILIARY OBSTRUCTION DUE TO STONE

To commence with, a high intake of dextrose is essential to build up the store of liver glycogen, and also to protect the liver.

If the Jaundice Abates.—The depth of the jaundice may diminish, the raised bilirubin level becomes constantly lower, and the stools become increasingly coloured. Such a train of events implies disimpaction of the stone, or possibly the passage of a small stone into the duodenum. In such circumstances diagnostic intravenous cholangiography can be undertaken and laparotomy performed if necessary.

If the Jaundice does not Abate.—If the jaundice is found to be increasing rather than decreasing, and particularly when any degree of jaundice is accompanied by pyrexia, operation should be planned to take place within two or three days.

Jaundiced patients tend to ooze from a cut surface. This tendency to bleed may be due to a diminution of prothrombin in the blood, resulting from failure of absorption of vitamin K₁, which is consequent upon diminution of bile salts in the alimentary canal. To raise the prothrombin level in the blood, vitamin K analogue (menaphthone B.P.) 10 mg. twice daily I.M., or vitamin K₁ (phytomenadione) 10 mg., must be given. Blood transfusion is another important ancillary. Antibiotic therapy (a tetracycline which is secreted in the bile) may also be given. *Mannitol* (p. 77) may be given intravenously to promote a diuresis and to prevent the renal failure which is a cause of mortality.

In patients who are deeply jaundiced, or who show signs of suppurative cholangitis, operation may be limited to draining the common bile duct (choledochotomy), and removing a stone or stones only if they are readily accessible. If it is impracticable to remove an impacted calculus, a biliary fistula is probable. A further operation will probably be required when the jaundice abates (p. 840).

Choledochotomy.—If a stone (or stones) is present in the common bile duct, its removal should have priority over cholecystectomy. Should the patient be unfit for cholecystectomy, or even cholecystostomy, the gall bladder should be removed on a future occasion.

Supraduodenal Choledochotomy.—Most stones in the common bile duct can be removed by this route. If, as is often the case, a stone can be felt, an attempt is made to manoeuvre it into a position midway between the entrance of the cystic duct and the superior border of the duodenum.

Direct Choledocholithotomy.—When the stone can be manipulated into the above desired position, it is steadied between the finger and thumb. The peritoneum overlying the duct is incised and dissected from the duct, which is opened longitudinally directly on to the stone, enabling it to be removed by a malleable scoop or Desjardins' gallstone forceps. The interior of the duct is then explored upwards and downwards with the scoop for further stones¹.

Indirect Choledocholithotomy.—When the stone cannot be felt, or cannot be manipulated into the optimum position just described, after incising the peritoneum overlying the common bile duct, a length of the underlying structure is displayed. When there are numerous adhesions, and especially when the gall-bladder has been removed at a previous operation, it is sometimes difficult to be certain whether the structure in question is the common bile duct or the portal vein. Aspiration through a fine hypodermic needle connected to a syringe will settle this point. As soon as about 2 cm. of the common bile duct has been exposed, the duct is transfixed by two stay sutures and a longitudinal incision into the duct is made between them. Escaping bile is mopped up or removed by suction. Through this opening it may be possible to identify the stone and remove it with a scoop or forceps (fig. 1079). After removal of the stone or stones by either of the above methods the common bile duct is cleared of any mud or grit by 'milking' the duct, followed by irrigation with saline solution. The patency of the sphincter of Oddi is tested either by the passage of a sound (3 to 5 mm. diameter), or by a confirmatory cholangiogram (p. 821)¹. Usually drainage of the common bile duct is carried out by means of a T-tube (fig. 1080). The transverse limb², shortened if necessary to about two inches long, is inserted in the duct which is closed snugly about the vertical limb, using fine catgut on an atraumatic needle. The long limb is brought out through a separate stab wound and securely anchored to the skin. The bile draining from the tube is collected in a plastic bag by the side of the bed, its amount and character being noted. Around the ninth and tenth day Hypaque is injected down the tube to obtain a cholangiogram (p. 821), and if there are not filling defects in a well-outlined duct, and the dye enters the duodenum freely, the T-tube can be removed by a sharp pull. Subsequent bile drainage is minimal and does not usually persist for more than a day.

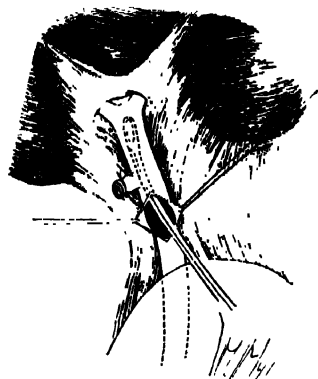


FIG. 1079.—Choledochotomy. The stone has been seized with Desjardins' forceps.

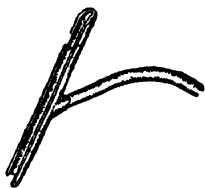


FIG. 1080.—T-tube for draining the common bile duct.

Closure of the Common Duct without a T-tube.—If this procedure of doubtful safety is attempted, it is most necessary to provide double drainage: (a) a broad piece of corrugated rubber down to the suture line through the laparotomy wound, and (b) a tube inserted into the right kidney pouch and brought out through a stab incision.

Transduodenal Choledochotomy is indicated when a stone is found to be impacted near the ampulla of Vater (fig. 1081) and it cannot be retrieved from above.

The duodenum should be mobilised by an incision through the peritoneum along the outer border of the second part. The duodenum and the contiguous head of pancreas can now be easily mobilised forward and, with the thumb

¹ It is estimated that 7 per cent. of patients with cholelithiasis have intrahepatic calculi in addition.

² T-tubes should be of latex or rubber and only used once. Plastic tubes are 'hardened' by the bile and are difficult to remove. Latex (and rubber) stimulate the fibrous adhesion of omentum to liver and colon to form a safe track. There is little reaction to a plastic tube and therefore the risk of biliary peritonitis is greater.

in front and the index and middle fingers behind, a stone in the lower part of the duct can be felt. It often facilitates the procedure if the surgeon crosses to the left side of the operating table.

The duodenum is opened in its second part by a transverse or longitudinal incision between stay sutures, and the region of the ampulla brought into the opening by traction using tissue forceps. Removal of the stone is either easy or requires the division of the duodenal papilla and the sphincter (sphincterotomy, p. 856).



FIG. 1081.—The trans-duodenal approach to a stone impacted in the ampulla of Vater.

Choledochoduodenostomy.—Acute pancreatitis may follow choledochotomy, and more especially when the ampulla of Vater is interfered with by bougies, dilators, or a scalpel. This type of pancreatitis moreover carries a definite mortality (Howard). It is for this reason that external choledochoduodenostomy has become increasingly popular. It is indicated (1) for recurrence of stone in the common bile duct, (2) where the common duct is opened for stone in association with relapsing pancreatitis, (3) when the surgeon is in doubt as to whether all stones have been removed, (4) where the duct is very dilated especially if there are no stones, (5) stricture of the lower end of the common bile duct (p. 841). The operation is contraindicated if the common duct is not 1 cm.

or more in diameter and if for some reason the duodenum cannot be mobilised easily or it is impossible to make a stoma of 3 cm. or more. The stoma must permit free entry and egress of fluid, for if it is large enough, regurgitant cholangitis does not occur and liver function is not impaired. It is always wise to make a choledochotomy incision as low as possible in the supra-duodenal portion of the common duct to remove stones. The opening is enlarged to 3 cm. or more and a transverse incision is made in the duodenum opposite this point. The two openings are approximated with catgut, with several reinforcing fine-silk sutures and a drain inserted (fig. 1082). The convalescence is usually surprisingly placid.



FIG. 1082.—Choledochoduodenostomy.

BILIARY FISTULÆ

(a) **External Biliary Fistula**—In days gone by, gall-stones and bile were sometimes discharged with the pus after a superficial abscess of the abdominal wall had been incised, or had ruptured, and even spontaneous extrusion of gall-stones through the umbilicus has been reported. Today nearly all external biliary fistulæ follow operations upon the biliary tract. When the neck of the gall-bladder or the cystic duct is occluded by a calculus (fig. 1083 A), the discharge is mucus. Should a stone be occluding the common bile duct, and cholecystostomy is performed, bile will continue to be discharged externally (fig. 1083 B). If cholecystectomy has been performed in these circumstances, again a biliary fistula (fig. 1083 C) will result. By far the most common cause of a biliary fistula is injury to the common bile duct during cholecystectomy (fig. 1083 D). A leakage after cholecystojejunostomy (fig. 1083 E) or cholecystoduodenostomy also will

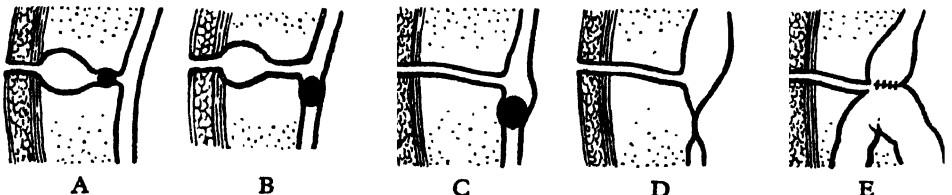


FIG. 1083.—Underlying causes of an external fistula of the gall-bladder or the bile ducts. (After J. J. Byrne.)

give rise to a fistula discharging bile. Other causes are division of an aberrant right hepatic duct or a hepaticocystic duct (p. 822).

After investigating, by retrograde cholangiography, the origin of the leak in the case of a fistula discharging bile, the continuity of the main bile duct must be restored (p. 842) or, in the case of a mucous fistula, the gall-bladder or its remains must be removed.

(b) **Internal biliary fistula** is due to a stone ulcerating through the wall of the gall-bladder or the common duct into a hollow viscus, viz. :

1. *Into the duodenum*—a large and usually single gall-stone sometimes ulcerates into the duodenum; this is the commonest internal biliary fistula. The stone may impact there causing duodenal obstruction. If smaller, it may pass on to obstruct in the lower ileum (gall-stone 'ileus', p. 947). There is evidence that such a fistula will close spontaneously. Later if symptoms persist, the gall-bladder can be removed.

2. *Into the colon*—as a rule no untoward complications ensue, but a large stone occasionally becomes impacted in the pelvic colon or the rectum.

3. *Into the stomach*—the patient may vomit the stones.

STRICTURE OF THE COMMON BILE DUCT
 < BENIGN < Post-operative 80%
 < Inflammatory 20%
 < MALIGNANT (p. 844)

Post-operative stricture concerns mainly either the common bile duct or the common hepatic duct, but in a smaller proportion of cases it is the right hepatic duct that is implicated. In almost all cases the stricture is the result of a preventable error in technique, usually during the performance of cholecystectomy. Injury at operation can occur in one of several ways:

1. **Blind plunge application** of a hæmostat to a bleeding cystic or accessory cystic artery, or to the right hepatic artery, is likely to result in damage to the common hepatic duct, viz. →

The prevention of this tragic happening is standardised. All unexpected

hæmorrhage in this region should be controlled initially by inserting the index finger of the left hand into the foramen of Winslow, and pinching the free edge of the gastro-hepatic omentum between the finger and thumb. Temporary compression of the hepatic artery in this way allows the bleeding-point to be visualised and ligated accurately.

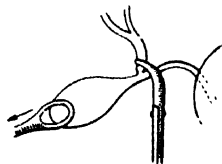
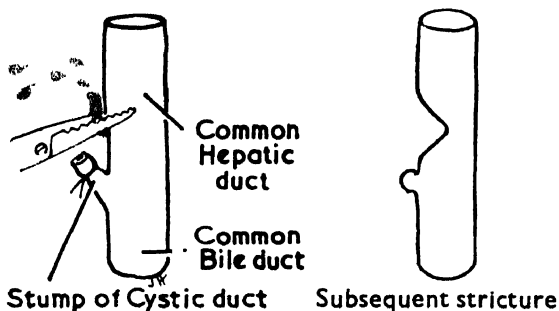
2. During cholecystectomy performed from the fundus, if too much traction is applied to the freed gall-bladder the common bile duct becomes tented, and so any forceps intended for the cystic duct grasp the angulated main channel, viz. →

3. Ignorance of the anatomical anomalies of the bile ducts (p. 822).

4. Laceration of the common bile duct while exploring it for stones or contracture of Oddi's sphincter.

5. In 3 per cent. of cases of stricture of the common bile duct, injury occurs during partial gastrectomy for a penetrating duodenal ulcer.

About 15 per cent. of injuries to the bile ducts are recognised at the time of



the operation. In 85 per cent. of cases the injury declares itself post-operatively by: (a) a profuse and persistent discharge of bile if drainage has been provided, or bile peritonitis if such drainage has not been provided; (b) by deepening obstructive jaundice. When the obstruction is incomplete, jaundice is delayed until subsequent fibrosis renders the lumen of the duct inadequate.

Inflammatory stricture may result from pooling of bile around the common bile duct, such as occurs after insecure ligation of the cystic duct during cholecystectomy or when the duct is improperly drained. Stenosis can also follow cholecystectomy or choledochostomy when the mucous membrane of the ducts is acutely inflamed ('red-hot' duct). Occasionally stricture of one or more ducts (usually the hepatic ducts) occurs as a result of what is known as stenosing cholangitis, the ætiology of which is obscure.

Treatment.—As these patients with strictures or biliary fistulæ are debilitated, efforts should be made to improve their poor condition by diet, vitamins, transfusions (including intravenous albumen), and antibiotics. Liver-function tests should be carried out before operation. Transhepatic cholangiography may be helpful (p. 845).

Various operative procedures are employed, according to the site of the stricture:

1. **Choledochoduodenostomy** (p. 840) is applicable if the duct protrudes more than 2 cm. below the porta hepatis. The duodenum can easily be rolled up after mobilisation. A wide stoma is essential, otherwise stricture of the anastomosis will occur.

2. **Hepaticojejunostomy**, using a jejunal conduit (Roux loop), is performed when choledochoduodenostomy cannot be undertaken. A mucosa-to-mucosa anastomosis is best but an effective alternative is to sew the open end of the jejunum to the liver around the opening of the duct. Any temporary post-operative drainage of the duct, and any splintage of the anastomosis, is obtained by either a transhepatic (Rodney Smith) or a jejunal tube (fig. 1084 A and B).

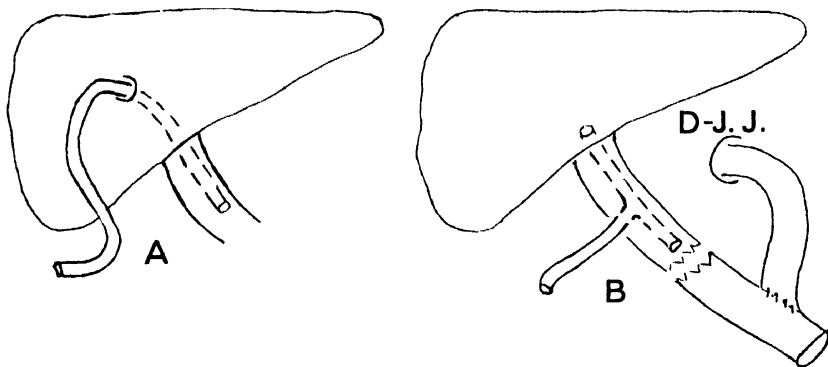


FIG. 1084.—Sketches of hepaticojejunostomy. A—Method of temporary drainage by transhepatic tube (cf. Nephrostomy, fig. 1426). B—Drainage by T-tube via jejunum. The length of the jejunal conduit in each case should be 8 to 10 inches above the Roux-Y anastomosis in order to prevent food and bacterial contamination of the hepatic ducts. D-J.J. Duodenojejunal junction.

ASIATIC CHOLANGIO-HEPATITIS

Obstruction to the common bile duct due initially to infection by liver fluke (*clonorchis sinensis*), which live in the biliary passages of a mammalian host, is frequently encountered in the Far East; in these countries it is more common than

inflammatory disease of the gall-bladder alone. The patient presents with pain, jaundice, and intermittent pyrexia. The urine contains bile. Clay-coloured stools are less frequently observed. Secondary infection of the obstructed ducts has almost always occurred by the time the patient seeks relief. Laparotomy should be undertaken as soon as the patient can be rendered fit to undergo the operation.

Operation.—The gall-bladder is frequently distended but is not often acutely inflamed. Palpation of the common bile duct reveals the presence of calculi and it is not unusual for the whole of the accessible system to feel solid with impacted stones. On opening the common bile duct, thick purulent bile, often containing adult clonorchis, gushes out and is removed by suction. The common bile duct and hepatic ducts are cleared of stones and mud by scoop and forceps, aided by irrigation with saline. Strictures frequently exist in the intrahepatic bile ducts which may render complete evacuation difficult or impossible. Choledochoduodenostomy at the initial exploratory operation gives better results than T-tube drainage, but if the duct is not very dilated transduodenal sphincterotomy may be adequate. A wide stoma allows stones to pass to the duodenum and helps to prevent a recurrence, although if intrahepatic biliary strictures are present multiple liver abscesses may form and the prognosis is then poor. Cholecystectomy should always be combined with choledochoduodenostomy or sphincterotomy, for if it is not done the accumulation of stagnant and infected bile within the gall-bladder frequently results in a secondary cholecystitis (Stock).

SYMPTOMS PERSISTING AFTER CHOLECYSTECTOMY

In 15 per cent. of all cases, cholecystectomy fails to relieve the symptoms for which the operation was performed. The term 'post-cholecystectomy syndrome' should be abandoned. A diligent search will enable the cause of symptoms to be found (Glenn). Symptoms that persist after cholecystectomy are most commonly due to lesions or disease of organs other than the biliary tract, such as hiatus hernia, duodenal ulcer, or pancreatitis. These having been eliminated, one of the following lesions of the biliary tract must be considered.

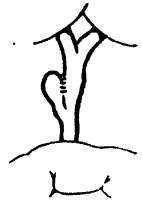


FIG. 1085.—Stump of cystic duct remaining after cholecystectomy.

1. A stone in the common bile duct escaped detection at the original operation.

2. When a comparatively long stump of a cystic duct has been left behind (fig. 1085), as frequently happens following cholecystectomy where the cystic duct joins the common bile duct lower than usual (fig. 1059(d)). This diverticulum may enlarge considerably and will harbour infection, and calculi may form therein.

3. Operative damage to the common bile duct occurred, resulting in stricture of that duct (p. 841).

4. In some patients fibrosis of the sphincter of Oddi is the cause of the symptoms, which differ from dyskinesia (see below) only in the fact that they are unrelieved by antispasmodics.

5. The cause of the original symptoms was biliary dyskinesia, which persists.

6. Hypotonia of the sphincter, which allows reflux along the ducts.

Treatment.—Cholangiography is extremely valuable in determining the cause which, if possible, is removed, e.g. a stump of the cystic duct must be excised; a stone or stones removed from the common bile duct. Dyskinesia is discussed below.

BILIARY DYSKINESIA¹

Biliary vesicular stasis is a form of dyskinesia confined to the gall-bladder. It commences early in life and causes dyspepsia. Cholecystography shows a long flask-shaped gall-bladder lying parallel to the vertebral column, but this is insufficient

¹ Dyskinesia = impairment of movement. In Britain this condition is still considered to be a doubtful clinical entity.

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Frank Glenn, Contemporary. Surgeon-in-Chief, New York Hospital, New York.

evidence; serial cholecystographs, however, demonstrate little or no evidence of active contraction and emptying of the gall-bladder. Medical treatment usually fails, and laparotomy is advised. The gall-bladder is found to be long and flaccid. The per-operation investigations must include (a) manometry to record the pressure within the gall-bladder, which in biliary vesicular stasis is less than 18 cm. of water (Rose) and (b) cholangiography, which shows that the medium runs readily into the duodenum. The treatment is right splanchnicectomy (Mallet-Guy).

Ductal Dyskinesia.—Hypertonicity of the sphincter of Oddi may follow vagotomy for duodenal ulcer; animal experiments show that the application of acid to the duodenal mucous membrane provokes spasm of the sphincter. Therefore it is always necessary to exclude peptic ulcer when spasm of Oddi's sphincter is demonstrated by intravenous cholangiography. Apart from this secondary phenomenon, cases of distension of the common bile duct and the gall-bladder without an organic lesion are due, presumably, to spasm of the sphincter of Oddi. The condition is rare. The symptoms are mainly those of minor biliary colic. Too often the condition is first thought of after cholecystectomy, which does not alleviate the symptoms. Cholecystography reveals a large gall-bladder, slow to empty, while intravenous cholangiography demonstrates obstruction at the duodenal papilla. *Antispasmodics relieve the symptoms.* In Asiatic cholangio-hepatitis (p. 842), biliary dyskinesia may be a causal factor (Harrison-Levy).

Treatment.—Prolonged medical treatment consisting of antispasmodic drugs and careful dieting sometimes renders the patient symptom-free. In resistant cases, sphincterotomy (p. 856) is indicated: cholecystojejunostomy (p. 866) also gives good results.

CARCINOMA OF THE GALL-BLADDER AND BILE DUCTS

Carcinoma of the gall-bladder and bile ducts accounts for 1 per cent.¹ of all malignant neoplasms. In nearly all the cases gall-stones are present, so this condition is more common in women than in men. It is rare under the age of forty-five.

The usual type is a scirrhous adenocarcinoma, a hard infiltrating tumour, usually starting in the fundus. If it arises near the neck of the gall-bladder a mucocele may follow from obstruction. A squamous carcinoma may follow the squamous metaplasia of the mucous membrane due to chronic irritation by gall-stones.

Malignant spread occurs early to the liver either directly or through the cystic lymph node and lymph nodes in the portal fissure. Involvement of the latter results in obstructive jaundice and, later, ascites. In many cases a past history of cholecystitis can be obtained, but pain, if present, tends to be continuous, with nausea, loss of appetite and weight. In over a third of cases a firm non-tender tumour is palpable in the gall-bladder area.

Treatment and Prognosis.—In view of the relationship with chronic cholecystitis and gall-stones, all gall-bladders on removal should be sectioned. In cases where a very early lesion is found, good results may be expected. Otherwise the prognosis is very bad (2 per cent. surviving five years). For the comparatively early definite cases, cholecystectomy with excision of adjoining liver is performed, but in many cases, especially where deep jaundice is present, no surgical treatment is possible.

Carcinoma of the major bile ducts is less common than carcinoma of the gall-bladder. Gall-stones co-exist in 38 per cent. of cases. The primary lesion is often small, but because of its strategic location, it causes symptoms and death early in the

¹ In Great Britain.

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 Pierre Mallet-Guy, *Contemporary*. Professor of Surgery, Faculté de Médecine, Lyon.
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course of its development. In 70 per cent. of cases metastases in the liver or the regional lymph nodes, or both, are found at laparotomy. The leading symptom is obstructive jaundice, which in 50 per cent. of cases is painless. Usually the liver is enlarged and the gall-bladder palpable, but sometimes, as a result of recanalisation consequent upon ulceration of the growth, the distended gall-bladder, initially palpable, becomes impalpable. Hepatic failure with coma is often a terminal event; sometimes this is preceded by cholangitis, with a high temperature and rigors. *Treatment.*—Early laparotomy affords the patient a slender chance of prolonged survival. Rarely is the growth found to be resectable. Sometimes a palliative procedure such as choledochoduodenostomy is possible, and symptomatic relief of the jaundice is afforded thereby.

Carcinoma of the Ampulla of Vater (p. 862).

Transhepatic Cholangiography (fig. 1086) may be very helpful in the differential diagnosis of jaundice due to intrahepatic and extrahepatic biliary obstruction due to carcinoma of the ducts, stenosing cholangitis, and other strictures (e.g. post traumatic, p. 841). Intravenous cholangiography and oral cholecystography (p. 820) are contraindicated in jaundice with serum bilirubin levels of above 3 mg. per cent., as bile excretion is suppressed or is not sufficient to carry through enough of the contrast medium to show on an X-ray film. The prothrombin level should be normal or raised to normal by I.M. injection of Vitamin K₁, and an intravenous test dose of 1 ml. of the medium given to exclude hypersensitivity (p. 821). Either the percutaneous (general anæsthetic) or the per-operative (laparotomy) approach is used. In the former case the needle is introduced just to the right of the xiphisternum. The needle is 15 cm. long, gauge 20, ensheathed by a flexible polypropylene tube, and it is passed into the substance of the right, and, if necessary, the left lobe of the liver. The needle is withdrawn and trial-and-error aspiration is performed until bile (usually white bile, p. 838) enters the syringe, whereupon 20 to 40 ml. of 45 per cent. Hypaque (sodium diatrizoate) is injected and an X-ray exposure made while breathing is temporarily interrupted. Up to five attempts are made to find bile before it is presumed that no dilatation of the intrahepatic bile ducts exists (as in hepatitis). A sample of the bile is always examined for bacteria—coliform organisms are often present (George).



FIG. 1086.—Transhepatic cholangiography (see text), showing a stricture of the common hepatic duct. (Miss Phyllis George, F.R.C.S., London.)

Phyllis Ann George, Contemporary. Surgeon, Royal Free Hospital, London.

CHAPTER 36

THE PANCREAS

Surgical Anatomy.—Every student is familiar with the head, neck, body, and tail of the pancreas. It is necessary to recall that from the lower and left part of the

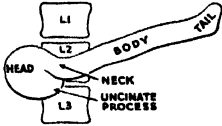


FIG. 1087. — The various parts of the pancreas and its relationship to the lumbar vertebrae. (After Rodney Smith, F.R.C.S., London.)

head projects the uncinæ process (fig. 1087), upon which rest the superior mesenteric vessels. The posterior surface of the neck is grooved deeply by these vessels, and more superiorly by the portal vein. In spite of the intimate contact of the portal vein with the pancreas, except in the case where it is bound down by inflammatory adhesions or in neoplastic infiltration, it can be separated from the pancreas with comparative ease. In so far as its blood-supply is concerned, the two embryological divisions of the pancreas retain their pristine independent nutrient vessels. Thus the head is supplied by the pancreatico-duodenal arteries while the body and the tail are nourished mainly by branches of the splenic artery. The distal part of the common bile duct lies in a deep sulcus between the second part of the duodenum and the head of the

pancreas. To examine the pancreas adequately by direct palpation, the organ must be mobilised by dividing the peritoneum (*a*) along the right margin of the duodenum and (*b*) along the lower border of the pancreas, the inferior pancreatico-duodenal artery being divided between ligatures.

The ducts of the pancreas are subject to anatomical variations, the most usual arrangement being that shown in fig. 1088 in which the duct of Santorini has a separate orifice about $\frac{1}{2}$ inch (1.25 cm.) above the duodenal papilla. This papilla is surrounded by Oddi's sphincter, known by surgeons throughout the world as the ampulla of Vater which is, in point of fact, the dilatation formed by the junction of the common bile duct and the duct of Wirsung within the base of the papilla. Spasm of the sphincter is believed to play a considerable part in the production of pancreatitis as well as persistent symptoms following cholecystectomy. It is well to reiterate that in 10 per cent. of persons the duct of Santorini is the main secretory duct of the pancreas, in which event the bulk of the secretion of the gland is ejected into the duodenum through the superior papilla.

Innervation of the Pancreas.—Apparently all the pain fibres are limited to the greater splanchnic nerves (T5–T10). These sympathetic nerves are believed to be distributed primarily to the pancreatic blood-vessels. Parasympathetic fibres reach the gland through the vagus nerves and terminate in the intrinsic pancreatic ganglia. All nerves to the pancreas pass through the celiac plexus.

Surgical Physiology.—The external secretion of the pancreas is controlled by nervous and humoral mechanisms of which the latter is the more important:

(*a*) **Nervous.**—Stimulation of the vagus evokes a secretion of viscid fluid rich in enzymes.

(*b*) **Humoral.**—This mechanism involves two hormones—*secretin* and *pancreozymin*. Both are liberated from the mucous membrane of the duodenum and proximal jejunum chiefly by acid but also by food. The hormones are carried via the bloodstream to the pancreas. *Secretin* alone produces a thin watery alkaline secretion poor in enzymes but rich in sodium bicarbonate. *Pancreozymin* evokes a juice rich in enzymes.

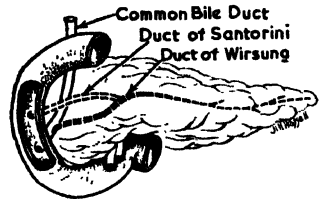


FIG. 1088.—The most usual arrangement of the pancreatic ducts.

Giovanni Domenico Santorini, 1681–1737. Professor of Anatomy, Venice.
Ruggiero Oddi, 1846–1906. Surgeon and Anatomist, Rome.
Abraham Vater, 1684–1751. Professor of Pathology and Therapeutics, Wittenburg, Germany.
Johann Wirsung, Prosector at Padua, was murdered when entering his house at night in 1643.

Pancreatic juice, which contains the enzymes amylase, lipase, and trypsin (the last secreted in an inactive form trypsinogen, and later activated by the succus entericus), is strongly alkaline. The alkalinity, due to the sodium bicarbonate it contains, is about pH 8.3, and no less than 3 to 4 G. sodium is secreted daily in this way. It is therefore not surprising that sodium deficiency develops if the juice is lost to the exterior. The normal adult secretes about 1 litre of pancreatic juice daily.

CONGENITAL ABNORMALITIES

Annular pancreas is due to failure of complete rotation of the ventral segment during development so that a collar of pancreatic tissue surrounds the second part of the duodenum. Usually it does not cause symptoms. In a minority it causes duodenal



FIG. 1089.—Annular lobe of the pancreas causing duodenal obstruction.

obstruction, which is characterised by a typical gas shadow seen on a plain X-ray film, and known as the 'double bubble', viz.:

One-third of those with symptoms are infants—the remainder are adults.



Treatment.—Since excision may be followed by pancreatic fistula the usual treatment is duodeno-jejunostomy, anastomosing the jejunum to the dilated first part of the duodenum. Sometimes, in adults, duodeno-duodenostomy is possible and this is ideal.

An ectopic pancreas is found in the submucosa of some part of the stomach, duodenum, or small intestine (including Meckel's diverticulum), the gall-bladder, adjoining the pancreas (e.g. in the hilum of the spleen) or, (in 2 per cent. of carefully conducted necropsies) within the liver. Were it not that an ectopic pancreas in the wall of the intestine is apt to be the starting-point of an intussusception, all save those in one situation would remain symptomless. The exception is an ectopic pancreas in the wall of the stomach, which is liable to undergo cystic degeneration and give rise to epigastric discomfort. Usually the rounded filling defect produced by this swelling is diagnosed on radiographic findings as a benign neoplasm.

Congenital cystic disease of the pancreas sometimes accompanies congenital disease of the kidneys and liver.

Mucoviscidosis (*syn.* fibrocystic disease) of the pancreas is but one manifestation of a hereditary congenital abnormality of mucus secretion that renders mucus exceedingly viscid. Mucoviscidosis affects all mucus-secreting glands, in particular those of the pancreas and bronchioles. Viscid mucus obstructs the pancreatic ducts, and retention of pancreatic secretions results; some of the alveoli rupture, pancreatic enzymes escape and become activated by tissue juice (Bodian). Thus, from birth or before it, the infant suffers from pancreatitis with subsequent fibrosis. The secretion of abnormally viscid mucus into the bronchial tree predisposes to respiratory infection, which takes the form of bronchitis, and frequently progresses to bronchiectasis. In 25 per cent. of cases the liver shows focal biliary cirrhosis. The sweat glands produce sweat containing two or four times more sodium chloride than normal. Thus, this remarkable disease affects not only mucus-secreting glands but all exocrine glands.

Clinical Features:

The life of one afflicted with fibrocystic disease of the pancreas is fraught with dangers.

At Birth.—In 10 per cent. of cases the infant is born with meconium intestinal obstruction (p. 938), or more rarely with meconium peritonitis (p. 875).

During Infancy.—In most cases a few weeks or months after birth bronchiolitis develops. There is dyspnoea, with inspiratory indrawing of the lower chest and the suprasternal notch, and a distressing spasmodic cough. In a number of instances the chest becomes barrel-shaped, and a radiograph shows bronchiectasis. In 90 per cent. of cases steatorrhoea is present, and usually the stools are unduly frequent, greasy, and offensive. In the absence of severe respiratory distress the appetite is voracious, in spite of which the infant is marasmic, except for a distended abdomen.

In Older Children.—Steatorrhoea with wasting (fig. 1090) continues, and is distinguished from coeliac disease by the early onset, the excellent appetite, the almost constant accompaniment of attacks of respiratory infection, and, possibly, by a history

of death in infancy from intestinal obstruction or pneumonia of siblings. In hot weather excessive loss of electrolytes in sweat sometimes results in severe dehydration and even death. Cirrhosis of the liver and hypertension frequently develop; indeed, one-third of all cases of portal hypertension at this time of life occur in subjects with this disease. If the patient reaches late childhood without gross pulmonary or hepatic damage, improvement is remarkable and often sustained.

In Adults: (a) Middle-aged patients with long-standing pulmonary suppuration and steatorrhœa should always be suspected of suffering from mucoviscidosis. Opacities in the vitreous humour, choroiditis, and bilateral enlargement of the submaxillary salivary glands are frequent accompaniments of mucoviscidosis in adult life.

(b) Staphylococcal infections are frequent and difficult to eradicate. This organism thrives in a medium containing a high concentration of sodium chloride.

(c) The maintenance of fluid balance following any operation on a patient with mucoviscidosis is a severe test of applied physiology. The amount of sodium, potassium, and chloride generally allowed in making up fluid balance charts is so much exceeded (because of loss of these ions in the sweat) as to call for "courage lest we falter."

Confirmatory Tests:

1. The amount of trypsin in the duodenal content is very low or absent.

2. *Sweat Tests:* (a) *The Full Sweat Test.*—The patient is encased in a polythene bag. The sweat is collected and subjected to chemical analysis. Neither in health nor in any other disease does sweat contain more than 70 mEq/L. sodium, 60 mEq/L. chloride, or 20 mEq/L. potassium. In 99 per cent. of patients

FIG. 1090.—Fibrocystic disease of the pancreas in a girl two years of age. (Dr. A. P. Norman, London.)

suffering from mucoviscidosis these figures are (often greatly) exceeded. During the test the patient requires careful supervision; fatal outcomes (heat stroke) have resulted from its application.

(b) *The Finger-print Sweat Test.*—A finger-print is taken on an agar plate. The agar is impregnated with silver nitrate and potassium chromate. In mucoviscidosis the excessive sodium chloride in the sweat bleaches the suspended chromate in under twenty minutes. The test lacks reliability.

3. The electric conductivity of saliva secreted by healthy persons declines after stimulation. In mucoviscidosis alone the value remains constant, or even increases.

Treatment should aim at controlling respiratory infection, which is nearly always staphylococcal. The antibiotic selected will depend on the laboratory tests of sensitivity of the infecting organism. A major problem is development of resistance by the organisms to one antibiotic after another.

Much can be done for fibrocystic disease of the pancreas by symptomatic treatment. Extra proteins should be given. The sugar intake should be high, but that of starch must be moderate. Fat is curtailed drastically, whereby the frequency, bulk, and foulness of the stools are diminished. Provided this diet is adhered to and enteric-coated capsules of pancreatin, 5 to 10 G. are taken before feeds, and 200 units of vitamin D and 10,000 units of vitamin A are given daily, there is nearly always a substantial gain in weight.

Lastly, there should be liberal salting of food. In hot weather extra salt, 2 G. per day, should be supplied. The dietetic regimen must be followed throughout life.

INJURIES

On account of its deeply placed, protected position, injuries to the pancreas are uncommon. Blows on the abdomen and traffic accidents are the usual causes; these

injuries can also occur from under-water blast. In many instances when the pancreas is damaged, other organs, particularly the spleen and the duodenum, are involved simultaneously. That the pancreas has been severely damaged can be suspected pre-operatively only by a high serum amylase estimation.

Type 1.—The patient, who has had a severe injury to the upper abdomen, presents signs of a serious lesion of some intra-abdominal organ, and it is thought wise to explore. When the pancreas is the injured organ, there is often blood-stained fluid in the lesser sac, and fat necroses are sometimes present.

Type 2 (Milder Injuries).—The first intimation that the pancreas has been lacerated is often the development of a pseudo-pancreatic cyst (p. 858).

Treatment (Type 1 Injury).—If the tail of the pancreas is completely or nearly severed, it should be removed, the duct closed by two very fine absorbable sutures, and the raw surface covered by a free omental graft. When the head or body is deeply lacerated it is best to approximate the two fragments with a single stitch, which is not tied tightly (multiple stitches cause necrosis of the pancreas). The lesser sac is drained, and the abdominal wall closed with non-absorbable sutures. Propantheline (p. 853) may expedite healing (see also Post-operative Acute Pancreatitis, p. 854).

INFLAMMATION

PANCREATITIS

It is now agreed¹ that this disease should be considered under the following headings:

- | | |
|-----------------------------------|---|
| 1. Acute Pancreatitis | } <i>These return to normal when the primary cause is removed.</i> |
| 2. Relapsing Acute Pancreatitis | |
| 3. Chronic Pancreatitis | } <i>In these, even if the cause is removed, structural damage still remains.</i> |
| 4. Relapsing Chronic Pancreatitis | |

ACUTE AND RELAPSING ACUTE PANCREATITIS

Pancreatitis is always a serious disorder, the acute attack carrying a mortality of 20 per cent. The great problem in this disease is the multiplicity of aetiological factors.

Ætiology.—In this country about 75 per cent. of cases have concurrent disease of the biliary tract. The remainder may be associated with one of the following—hyperparathyroidism (especially where calculi are present), hypothermia, mumps, or vascular disease. A few cases are familial and are associated with hyperlipæmia, fibrocystic disease of the pancreas, or a defective amino-acid metabolism (especially for cystine or leucine). In the U.S.A., about 50 per cent. are associated with biliary disease, 25 per cent. with chronic alcoholism, and the remainder are miscellaneous as above. Post-operative pancreatitis requires special mention (p. 854) because of its high mortality.

The basic process in acute pancreatitis is one of auto-digestion following activation of trypsinogen. There are many hypotheses as to the mechanism of this. Some postulate reflux of bile or of duodenal juice into the pancreatic duct system due to anatomical or mechanical causes, or pressure differences between the pancreatic and biliary system. Others claim to find pancreatic duct obstruction or infection, in addition to the vascular and metabolic conditions mentioned already. It is important to add, however, that evidence is

A SHORT PRACTICE OF SURGERY

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¹ The Marseilles classification was adopted in 1963 in order to standardise analysis of this condition.

accumulating that the process may start as an intracellular derangement. There is no evidence that it is an auto-immune disease.

None of these theories gives adequate explanation of the disease and the actual cause remains unknown.

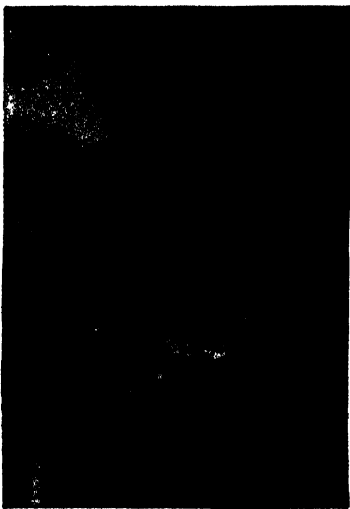


FIG. 1091.—Widespread fat necroses of omentum. A test-tube has been filled with the blood-stained peritoneal fluid. This specimen was rich in amylase. (G. D. Adhia, F.R.C.S., Bombay.)

Pathology.—Hitherto, the terms œdematous pancreatitis and hæmorrhagic pancreatitis have been used. These are not really separate conditions, but different stages of one process. They have, of course, a vastly different prognosis; œdematous pancreatitis is rarely fatal.

The following are the outstanding features:

(a) **The peritoneal cavity** contains a blood-stained exudate; the omentum and subperitoneal fat are studded with areas of fat necrosis (fig. 1091).

Fat necroses are dull, opaque, yellow-white areas suggestive of drops of tallow. They are most abundant in the vicinity of the pancreas, but are widespread in the greater omentum and the mesentery. At necropsy they can sometimes be demonstrated beneath the pleura and pericardium, and even in the subsynovial fat of the knee joint. Fat necroses consist of small islands of saponification caused by the liberation of lipase, which splits fat into glycerol and fatty acids. Free fatty acids combine with calcium to form soaps=fat necrosis.

(b) **The Pancreas.**—In the mildest form the whole or part of the organ is œdematous, but if severe, it may be grossly hæmorrhagic and some-

times necrotic. The retroperitoneal tissues around the pancreas may then be engorged with blood-stained fluid.

(c) **Associated Pathology.**—As already mentioned, gall-stones and infected bile are frequently found.

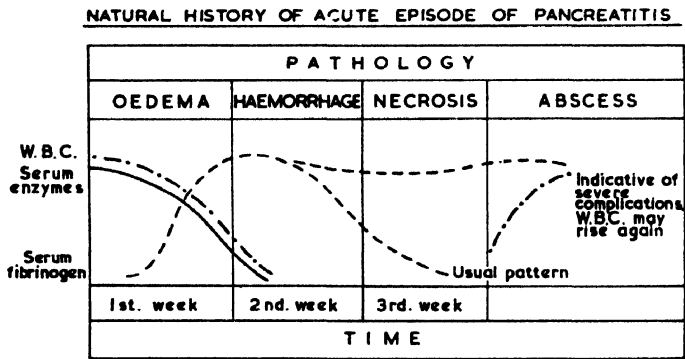


FIG. 1092.—Compiled from clinical evidence and autopsy data. (J. E. Trapnell, F.R.C.S., Bristol.)

When the lesser sac has been drained and the patient survives, portions of necrotic, pancreatic, and peri-pancreatic tissue may be discharged through the wound as putty-like material. On analysis this material has been shown to be composed largely of calcium stearate.

Clinical Features.—Unlike acute cholecystitis, acute pancreatitis shows no predilection for obese persons, no sex discrimination, and while the age of

the patient is extraordinarily variable, the maximum incidence is between fifty and sixty years. The salient features of acute pancreatitis are as follows:

Onset.—The attacks come on at any time but frequently follow the ingestion of a heavy meal and/or alcohol.

Pain is agonising, mainly epigastric, radiates to the back (fig. 1093) and slowly rises to a crescendo over several hours (in contradistinction to the pain of perforation). Moynihan described it as 'illimitable agony'.

Vomiting and retching, repeated and noisy, are a marked feature.

Rigidity, like pain, develops slowly and may become generalised.

Tenderness can be elicited in the early hours of the disease by firm and deep palpation over the pancreas and occasionally in the left renal angle where the tail of the pancreas is comparatively near the surface.

Profound shock is not uncommon—the patient is sweating and collapsed, the nose is cold, and the blood-pressure depressed. This feature calls for urgent infusion of plasma. The shock may be due partly to circulating 'kinins' which are formed by the action of trypsin on the plasma proteins.

Cyanosis.—Cyanosis is a common accompaniment of the acute forms of acute pancreatitis.

Faint jaundice may sometimes be seen especially during the second day. Probably it is obstructive, and due to œdema of the head of the pancreas.

Ileus in the early stages is limited to the duodenum and proximal jejunum. The gas-filled solitary loop may show on plain X-ray (a useful diagnostic feature if present). Unless prevented by gastro-intestinal aspiration, peristalsis diminishes after twelve hours and abdominal distension supervenes.

A tender, palpable mass may appear in the epigastrium towards the end of the second week. This is usually a pseudo-cyst (p. 858). If the mass appears later—after the third week—it is due to abscess formation.

Discoloration of the skin is a rare manifestation of acute pancreatitis seen in cases of two or three days' standing. Grey Turner first described it in the loins, and likened it in appearance to that of late extravasation of urine. Other observers have described a similar discoloration around the umbilicus. The cause of the phenomenon is the action on the subcutaneous fat of pancreatic ferments that have escaped from the retroperitoneal tissues, either directly or via the round ligament of the liver.

Laboratory and Other Investigations:

Leucocyte Count.—A leucocytosis above 10,000 per c.ml. occurs frequently, and it may rise as high as 30,000.

The Serum-amylase Estimation.—The normal value is 80 to 150 Somogyi units; 400 units is suggestive of abnormality. A level of 1,000 units is the minimum necessary to support a diagnosis of acute pancreatitis. The highest level is attained within an hour after the onset of symptoms. Subsequently there is a rapid fall to normal level within forty-eight hours.

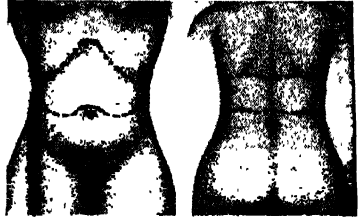


FIG. 1093.—The usual distribution of the pain of acute pancreatitis.

The diastatic index of the urine is normal (below 50 units) during the early hours of the attack. After twelve to twenty-four hours it often rises to 100 units or more; sometimes it is as high as 500 and, rarely, 1,000 units. The importance of the diastatic index of the urine lies in the fact that it remains elevated after the serum amylase has returned to normal.

The serum calcium (normal—10 mg./100 ml.).—Concentrations below 7 mg./100 ml. are often followed by the death of the patient, in spite of the administration of calcium. The test is of some prognostic value as is the appearance of tetany. The serum calcium level may be an indication of the amount of calcium fixed in the formation of calcium soaps.

The serum bilirubin (normal—not exceeding 0.2 mg./100 ml.) is nearly always raised, even when no icteric tint of the sclera is apparent.

The serum fibrinogen is raised at the end of the first week and should return to normal in the third. This is of considerable prognostic value (fig. 1092).

Methæmalbumen—the presence of this in the blood indicates a hæmorrhagic necrosis and is associated with a bad prognosis.

Electrocardiographic Changes.—It is by no means uncommon to find abnormal electrocardiographic recordings in patients with acute pancreatitis; in a few the record is so abnormal as to support a provisional diagnosis of myocardial infarction. These electrocardiographic changes disappear with recovery of the patient, but may return during subsequent attacks (Pollock).

Radiography.—The chief value of this is to demonstrate the ileus, which shows as a gas shadow in the duodenum and first loop of jejunum.

Differential Diagnosis.—The most important point in diagnosis is to remember it as a possible cause in every case of upper abdominal pain and to request a serum amylase. Coronary occlusion, high intestinal obstruction, perforated peptic ulcer, leaking dissecting aortic aneurysm, and gall-stone colic are the conditions that may have similar features. The absence of gas beneath the diaphragm helps to exclude a perforated peptic ulcer.

ABDOMINAL PAIN WITH RAISED SERUM AMYLASE

The significance of a serum amylase above 1,000 units associated with abdominal pain is of great importance to the surgeon. Acute pancreatitis is best treated conservatively whereas other conditions may require laparotomy. The following conditions should therefore be considered:

1. *Afferent loop obstruction* following gastrectomy—this is the commonest cause. It occurs usually in the early post-operative period but has been reported at 5 years. Back pressure in the distended loop up the pancreatic duct is presumed to raise the serum amylase (sometimes up to 3,000 units or more). It can be confidently diagnosed in these circumstances because there is no aspirate of bile-stained fluid from an indwelling gastric tube. This distension factor may also be present in high intestinal obstruction apart from gastrectomy.

2. *Absorption through the peritoneum* as in biliary peritonitis—due to a perforated duodenal ulcer, leaking duodenal stump, a leak following biliary surgery or perforation of the gall bladder.

3. *Damage to the Pancreas or its blood supply*—as in direct trauma, thrombosis of the aorta or the inferior vena cava or a ruptured aortic aneurysm.

4. *Spasm of the ampulla of Vater*—may follow administration of morphine (p. 853). Raised serum amylase has also been reported in ectopic gestation and bronchial carcinoma.

Treatment.—Operation in this condition increases the mortality. Conservative treatment is the basis of modern management. The essentials of treatment, whatever the cause, are:

Relief of Pain.—The most effective drug is atropine in adequate amounts, but this may be contraindicated in the elderly. Pethidine¹ (100 mg. I.M.) is also

¹ Pethidine is known as demerol in Canada, and as demerol hydrochloride in the U.S.A.

valuable. Morphine should *not* be used as it increases spasm of the sphincter of Oddi.

Rest for the Pancreas.—This means suppression of secretory activity by abolition of stimuli. Vagal influence can be abolished by atropine gr. $\frac{1}{100}$ (0.6 mg.) four-hourly, or propantheline 15 to 30 mg. intravenously in a slow drip, repeated, with caution, in six hours, if necessary. Hormonal stimulation (secretin and pancreozymin) is best subdued by continuous and efficient gastric suction and nothing must be given by mouth. These measures, by resting the pancreas, contribute greatly to the relief of pain.

Replacement of Fluid and Electrolytes.—Rapid correction is urgent and life-saving. It is wise to start with plasma pending estimation of electrolytes. It is best to avoid excessive use of glucose, as this may stimulate the pancreas. Calcium gluconate 10 ml. of a 10 per cent. solution. I.V. should be given daily. If hypotension persists in spite of the use of plasma, intravenous noradrenaline (given via a long catheter introduced into one of the great veins) may be valuable.

Other Therapy.—An antibiotic should be used to prevent secondary infection—one of the tetracyclines is probably best. Despite the fact that some cases of acute pancreatitis have arisen during therapy with adrenal hormones, hydrocortisone or ACTH may be valuable in the desperate case. Trasylol, which destroys trypsin *in vitro* has not yet been conclusively proved to be clinically effective.

If the diagnosis is made at laparotomy, it is safer in the majority of cases to do nothing but close the abdomen with non-absorbable sutures. Any manipulation around the head of the pancreas will increase the mortality. Occasionally, associated pathology such as fulminating cholecystitis requires extirpation. Subsequently, patients are treated as already described.

Complications of Acute Pancreatitis.—While the treatment of acute pancreatitis is preferably non-operative, the treatment of most of its complications requires surgical intervention. In epitome the complications are:

1. *The development of a pseudo-cyst* occurs in about 12 per cent. of cases (p. 858).
2. *Abscess formation* (rare), which gives rise to a swelling most commonly in the left flank, a swinging temperature (sometimes masked by antibiotics), and a raised plasma fibrinogen level (fig. 1092). The abscess should be drained posteriorly.
3. *A collection of blood in the lesser sac* (rare), characterised by the very early and sudden appearance of an epigastric swelling, is best treated by evacuation of the clot under local anaesthesia—a measure which slightly improves the very poor prognosis.

Convalescence.—The diet should be bland, consisting essentially of small frequent meals avoiding fried food, spices, and thick soups. Alcohol is forbidden. Food is best eaten dry, fluids being taken between meals. An antispasmodic, i.e. tinct. belladonna, just before meals may be beneficial. Some patients derive considerable benefit from the regular use of the slow-release glyceryl-trinitrate preparation 'Sustac'. By far the most important feature of convalescence is to determine those cases likely to have recurrences, in particular those with associated biliary disease. Cholecystography is mandatory but should be delayed for a month and a fat meal should *not* be given during the X-ray to encourage the gall-bladder to contract. In those cases with stones, cholecystectomy and choledocholithotomy should be done as a safeguard against recurrence. In the group in which gall-stones are not

found, the prognosis with regard to recurrence is uncertain. In general, however, acute pancreatitis or relapsing acute pancreatitis does not proceed to chronic pancreatitis.

POST-OPERATIVE ACUTE PANCREATITIS

In this condition, the mortality is greater than 50 per cent. It may follow any surgical procedure but occurs most commonly after operations on the biliary tree, the stomach, and the spleen. Manipulation in and around the sphincter of Oddi is especially liable to cause this condition. The real cause is as uncertain as it is in pancreatitis generally. The essential treatment lies in early diagnosis and conservative management as described.

CHRONIC PANCREATITIS

It is best to regard this condition as an entirely separate entity from acute pancreatitis. The latter rarely if ever proceeds to the chronic disease. The essential change in the pancreas is slowly progressive with destruction of acini, increasing fibrosis, and often with corresponding functional failure. In some, there may be calcification in the gland—this is seen especially if there is a background of alcoholism or where hereditary or endocrine factors are prominent.

Clinical Features.—There are five main types of presentation:

- (1) As persistent upper abdominal *pain* passing through to the back (p. 851).
- (2) As a cause of obstructive *jaundice*.
- (3) As a *malabsorption* disorder—i.e. failure of external secretion, with steatorrhœa, weight loss, and vitamin deficiencies.
- (4) As *diabetes*—i.e. failure of internal secretion.
- (5) Sometimes the diagnosis is made solely by palpation of the pancreas during *laparotomy*. The features may include nodularity, irregularity, fixity, induration, or the presence of a mass. It is unwise to make the diagnosis on nodularity alone (the normal pancreas may be nodular when it is turgid with secretion).

Age, Sex, and Habitude.—Unlike cholecystitis, chronic pancreatitis occurs much more frequently among men than among women. There is no predilection for fat persons, and the age incidence, while having a more restricted ambit, is not dissimilar from that of carcinoma of the pancreas.

RELAPSING CHRONIC PANCREATITIS

Clinical Features.—These cases characteristically fall into type 1 (above).

Pain.—The hall-mark of relapsing pancreatitis is recurrent attacks of pain which may exceptionally last for only an hour but usually continue for two or three days (cf. Biliary Colic, p. 828, the attacks of which last from minutes up to two or three hours).

Periodicity.—The attacks vary in frequency; they may occur as often as once a week or as seldom as three times a year. As a rule, with the passage of time the attacks become more frequent. Happily, in a few patients the remissions lengthen.

Vomiting is usually oft-repeated, and commences some time after the pain; the contents of the duodenum, as well as those of the stomach, are ejected.

Weakness or fatigue is common, and it is not unusual for this to herald an oncoming attack by two or three days.

Jaundice is somewhat exceptional ; in several large series of cases it has been present in under 15 per cent. of cases. Observing that gall-stones, if present, are nearly always confined to the gall-bladder, it would seem that mild jaundice is hepatogenous in origin (Cattell), although undoubtedly in the more advanced cases it is the result of pressure upon the terminal part of the common bile duct.

Atherosclerosis and chronic relapsing pancreatitis often go hand in hand (Saint).

Alcoholism or drug addiction is so often present (40 to 50 per cent.), that either is to be regarded as an important diagnostic datum. The habit is sometimes acquired in an endeavour to seek relief from the intolerable pain.

Radiography.—It is important to have a plain film before any barium series to ascertain whether there is any pancreatic calcification (fig. 1094). When this is present it is usually intraductal. The barium series may show widening and indentation of the duodenal circuit. Routine cholecystography should always be done. If the gall-bladder has been removed, an intravenous cholangiogram is necessary.

Special Investigations in Pancreatic Disease

Serum Amylase.—This may be of use in the relapsing cases. It is increased during the attack. The elevation may not be great but it must be related to the base-line level obtained between attacks (e.g. 250 to 300 mg. per 100 ml. in an attack with a base-line level of 50 mg. per 100 ml.).

Stools.—*Steatorrhœa* is present when chemical examination (to measure the fatty acids) shows the fat content to be greater than 18 gr. in three days (Van de Kamer). The stools become bulky, pale, frothy, offensive, and float on water. Malabsorption occurs late in the disease and only appears when some 80 to 90 per cent. of the gland has been destroyed. Having recognised the presence of *steatorrhœa*, pancreatic disease can only be identified for certainty by duodenal intubation tests to determine the presence or absence of pancreatic enzymes.

Duodenal Intubation (Howat)—*The Secretin-Pancreozymin Test.*

Pancreatic secretory capacity can be measured by collecting duodenal contents quantitatively after stimulating the pancreas with secretin or secretin and pancreozymin given intravenously. This is the most helpful of all tests in the diagnosis of early chronic pancreatitis. A double-lumen radio-opaque tube (Dreiling) is passed and so sited that one lumen drains the stomach, the other the duodenum; in this way duodenal contents can be collected free of gastric contamination. Continuous suction with negative pressure not greater than 50 mm. of Hg. is applied and the duodenal contents collected in fractions. After requisite control periods, secretin is given and the duodenal contents collected in ten-minute fractions for thirty minutes. Then pancreozymin is given and the collection is continued for a further thirty minutes.

The characteristic features of chronic pancreatic disease, either chronic pancreatitis or carcinoma of the body of the pancreas, are reduced bicarbonate concentration and output, after secretin, and reduced output of the enzymes—amylase, trypsin, and lipase after pancreozymin. Obstruction of the duct due to stones, stricture, a pseudo-cyst, or carcinoma leads to a reduced volume of the duodenal contents.

The feature which distinguishes chronic pancreatitis from an attack of relapsing pancreatitis in which restitution to normal takes place, is a permanently diminished concentration and output of bicarbonate after secretin. The output of enzymes after pancreozymin is a slightly more sensitive index of pancreatic damage than a reduction of bicarbonate, particularly in carcinoma of the pancreas. Since pancreozymin contains cholecystokinin which contracts the gall-bladder, the secretin-pancreozymin test in

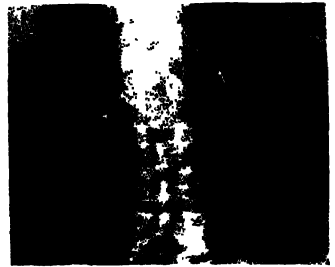


FIG. 1094.—Diffuse pancreatic calcinosis.

early obstructive jaundice is a useful guide to the site of the primary obstruction either in the pancreatic or bile ducts. Impaired tests are always found in steatorrhœa of pancreatic origin. The duodenal contents obtained in these tests are particularly suited to examination for exfoliated carcinoma cells in both biliary duct and pancreatic growths (Papanicolaou test, p. 864).

Morphine Provocation Test.—Pain may be provoked by morphine or codeine, together with an associated rise of serum amylase.

Glucose Tolerance Test.—With progressive pancreatitis, glycosuria appears and a diabetic type of blood sugar curve is obtained. This is in contradistinction to intestinal causes of malabsorption, e.g. idiopathic steatorrhœa, which give flat curves.

Occult blood in stools is usually absent in chronic pancreatitis, but may be present in carcinoma of the pancreas (p. 864).

Differential Diagnosis from Carcinoma of Pancreas

It is very difficult to be certain about this: (1) Because the length of the history is of little value and the duodenal intubation tests may be equivocal. (2) If the swelling found at laparotomy is confined to the pancreas alone, then it is still impossible to be certain. Biopsy is of little or no value and it may be dangerous. Birnstingl records ten cases with obstructive jaundice declared on pancreatic biopsy to be chronic pancreatitis, yet all were dead with carcinoma of the pancreas within three years. However, if the disease has spread to the lymph nodes along the superior border, a valuable biopsy can be easily carried out.

Prognosis.—A patient is unlikely to die of chronic pancreatitis, or even from one of the complications of this disease such as steatorrhœa, diabetes mellitus, or gastro-intestinal hæmorrhage, but is likely to succumb eventually to some unrelated disorder such as cerebrovascular disease.

Treatment.—All cases need a high-protein, high-calorie diet. Sustac (p. 853) should be tried. Alcohol is completely forbidden. A concentrated triple-strength enteric-coated tablet of pancreatic enzyme is given with each meal, together with a multivitamin capsule. Anæmia should be corrected.

Further treatment will vary according to the presenting features, but in general those with diabetes and malabsorption phenomena are best treated medically and rarely need surgery. When pain or jaundice is present surgery is usually necessary. In relapsing chronic pancreatitis sphincterotomy and biliary tract procedures usually fail and partial or total excision is favoured. In relapsing acute pancreatitis, however, sphincterotomy and biliary tract surgery are often successful (Howat).

OPERATIONS FOR CHRONIC PANCREATITIS

(a) **For Pain.**—This may be so severe that it leads to drug addiction. The variety of procedures recommended is an index of the unsatisfactory treatment of this condition.

(1) *Pathology in the biliary tree* must be thoroughly eliminated. This will involve *cholecystectomy* for stones and possibly exploration of the common bile duct. Some surgeons recommend *transduodenal sphincterotomy* or *external choledochoduodenostomy* (p. 842). In general the results of the latter, where possible, are better than those of the former. A guide as to the necessity for sphincterotomy is the production of pain on morphia provocation.

Transduodenal Sphincterotomy.—The second part of the duodenum and the head of the pancreas are mobilised and the region of the duodenal papilla is palpated

as described on p. 846. The common bile duct is located, opened, and a Bakes' dilator is (fig. 1095) introduced through the opening and passed in a downward direction. If a Bakes' dilator, size No. 4, cannot be made to enter the duodenum with ease, the papilla should be located by palpation against the head of the pancreas through the duodenal wall with the dilator in place. A vertical incision is then made through the duodenal wall exactly opposite the papilla. With a small Bakes' dilator in the common bile duct the papilla can be elevated into the mouth of the duodenotomy incision (fig. 1096). A grooved director is insinuated into the orifice of the papilla as the Bakes' dilator is withdrawn. At 11 o'clock (the clock is viewed from the right) a wedge of tissue, including Oddi's sphincter, is excised from the whole thickness of the papilla.

Sphincterotomy performed in this way gives free exit to the terminal portions of both the common bile duct and the duct of Wirsung. It is wise to place an ordinary T-tube in the common bile duct. The duodenum is closed meticulously either transversely or longitudinally. If the latter method is employed, a minimal amount of duodenal wall is turned in.

In either event the outer layer is closed with fine interrupted silk sutures. The abdominal wall is closed, and the sub-hepatic space is drained. This operation is effective in some cases, but it is not of great value if there are structural changes in the gland, calcification, or duct strictures.

(2) *Interception of Nerve Pathways—Splanchnicectomy.*—Resection of 3 cms. of the right great splanchnic nerve may be used in the treatment of intractable pain, but the long-term results are uncertain.

(3) *Partial Excision and Drainage—Retrograde Pancreatojejunostomy.*—Protagonists of this method state that in chronic pancreatitis the major pancreatic duct is often strictured for a greater length than could be remedied by performing sphincterotomy. With careful selection, therefore, this operation can be very effective. The tail of the pancreas is mobilised and amputated obliquely. The duct of Wirsung is located and slit for a short distance, the cut edges being held apart by sutures as shown in fig. 1097 inset. Any calculi are removed. The divided distal end of the jejunum is then brought over the stump of pancreas like the finger of a glove, and sutured

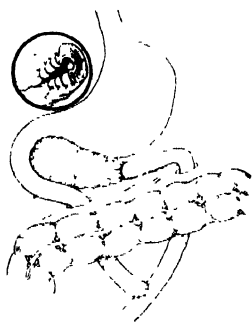


FIG. 1097.—Retrograde pancreatojejunostomy. (After M. K. Du Val, Jnr.)

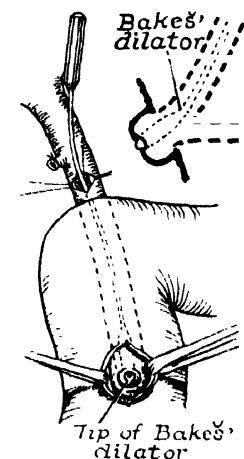


FIG. 1096.—Transduodenal sphincterotomy, displaying the duodenal papilla preparatory to division of Oddi's sphincter.

into place. The operation is completed as shown in fig. 1097. During the course of the operation *pancreatography* may be done to demonstrate the topography of the duct. This procedure is not without risk; hence the need for a small volume of contrast media (about 2 ml.) introduced at low pressure. A short piece of ureteric catheter is passed down the duct and some diluted Hypaque (70 per cent.) injected slowly. It should be possible to assess obstruction and the presence of further calculi in this way.

(4) *Excision of Pancreas—Pancreatoduodenectomy* (p. 865).—When the lesion is situated mainly in the head of the pancreas, the patient is in adequate condition, and the pain is intolerable, it may be necessary to perform this formidable operation which offers the prospect of relief of symptoms.

By and large, if any of these operations are undertaken, a good result may be expected in about half the cases.

(b) **For Jaundice.**—If this is the presenting feature, biliary drainage is essential.

Cholecyst-jejunostomy (fig. 1098) is the oldest operation for this condition, and, for a patient in poor condition, is inferior to none, provided the gall-bladder is thin-walled and distended with bile (i.e. that there is no obstruction to the cystic duct).

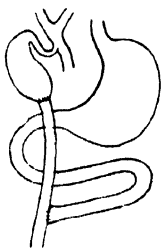


FIG. 1098.—Cholecyst-jejunostomy—en-Y (Roux).

PANCREATIC CALCULI

These are intraductal and consist of calcium carbonate. The usual picture is one of multiple radio-opaque stones. Parathyroid disease should be excluded. They are the *result* of the disease and not the cause—but if surgery is performed on the pancreatic duct (see above), the stones should be removed. Surgery is *not* mandatory for stones alone.

PANCREATIC CYSTS: Pseudo-cyst 80%.
True 20%.

Pseudo-cyst of the Pancreas.—This is essentially a collection of fluid in the lesser sac (fig. 1099) and occurs most frequently as a result of acute pancreatitis, or sometimes after injury. In order of frequency the pseudo-cyst projects (a) between the stomach and the transverse colon; (b) between the stomach and the liver; (c) behind or below the transverse colon.

Clinical Features.—An epigastric swelling appears during the conservative treatment of pancreatitis, or in traumatic cases during or after convalescence for an injury that did not merit laparotomy. Exceptionally the patient is admitted with a pseudo-cyst of the pancreas and gives a history of an attack of severe abdominal pain that occurred a week or more previously.

On examination there is a swelling, sometimes as large as a melon, placed centrally above the umbilicus: the swelling is fixed, and in many instances it is so tense that fluctuation cannot be elicited. As a rule, transmitted pulsation from the abdominal aorta is very noticeable and the diagnosis of an aneurysm may be considered, although in the latter case the pulsation is expansile and, in the knee-elbow position, the pulsation is much diminished. Moreover, in the case of a pancreatic cyst, a bruit is absent and the swelling,

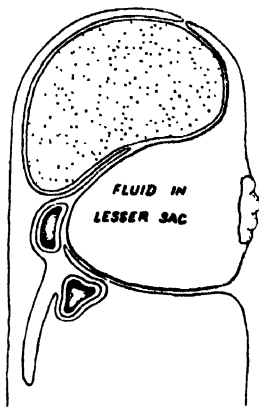


FIG. 1099.—Pseudo-pancreatic cyst.

which is the presenting feature, is much broader. Mesenteric cysts are rare and are very mobile. A barium meal shows projection of the cyst into hollow viscera, especially the stomach (fig. 1100). Every case of acute pancreatitis must be observed for the development of a pseudo-cyst. With this in mind, the width of the vertebro-gastric interval as seen on lateral X-ray should be assessed on the tenth day of the disease. The earlier a pseudo-cyst appears the worse the prognosis.

Treatment.—As soon as a cyst is confidently diagnosed, operation should be done. If it is delayed, infection may occur, frequently with tragic results. The treatment is cysto-gastrostomy. The stomach is opened through its anterior wall and an incision of about 2 inches (5 cm.) made through the posterior wall into the cyst. Hæmostasis is secured and the anterior wall of the stomach is closed (fig. 1101). The contents of the cyst then drain into the stomach.

Convalescence is shortened thereby, and the complications referred to are obviated. Curiously, food does not enter the sac, as might be expected. The opening must be large enough to allow for subsequent contraction.



FIG. 1100.—Barium meal—lateral view. Pseudo-pancreatic cyst pushing the stomach forward. (A. V. Pollock, F.R.C.S., Scarborough.)

True Cysts of the Pancreas.—In 20 per cent. of cases a cyst of the pancreas is a true cyst, and falls into one of the following categories :

(a) *Cyst-adenoma.*—Most true cysts belong to this group. Paradoxically these usually present as a solid swelling. The cysts are multiple and lie in an adenomatous mass. They may move on respiration and often have a narrow attachment to the pancreas. Removal is therefore accomplished without undue difficulty.

(b) *Mucoviscidosis of the Pancreas* (p. 847).

(c) *Congenital Cystic Disease.*—Possibly associated with polycystic disease of the kidneys and liver.

(d) *Retention Cyst.*—This is part of the pathological change in chronic pancreatitis and is due to ductal obstruction.

(e) *Hydatid Cyst.*—The ubiquitous hydatid completes the list.

Clinical Features.—Epigastric discomfort and the presence of a swelling are the usual reasons that cause the patient to seek advice. When the swelling arises in the tail of the pancreas it may be impossible to differentiate it from a hydronephrosis until pyelography has been performed. When the cyst arises in the head of the pancreas, the symptoms are identical with those of a choledochal cyst (p. 824).

Treatment.—A comparatively small cyst can be removed by dissection. A large cyst of the head of the pancreas is suitable for cystoduodenostomy. A similar cyst of the body of the organ can be treated by cysto-gastrostomy. When the cyst (or cysts) are situated towards the tail of the pancreas (this is the site of election for cystadenoma) the affected portion of the pancreas can be amputated.

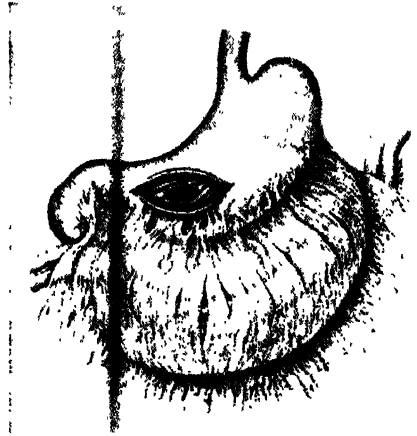


FIG. 1101.—Cysto-gastrostomy for pseudo-pancreatic cyst.

PANCREATIC FISTULA

This usually follows trauma to the gland either by closed external injury or operations for splenectomy or rarely gastrectomy. In most cases following operation, the injury was not suspected at the time.

Treatment.—The problems in the management of these fistulae are:

(1) *Fluid and Electrolyte Loss.*—In the presence of a fistula the pancreas tends to hypersecretion and anything from a few ml. to 2 litres may be lost in twenty-four hours. Such a loss of fluid and electrolytes may constitute a menace to life; they must therefore be replaced either by mouth or intravenously. In large losses, daily fluid and electrolyte balance must be maintained. If there is much loss, pancreatic extract should be given by mouth. Pancreatic secretion can be partially suppressed by propantheline (p. 853). The diet should consist of high protein and low carbohydrate.

(2) *Autodigestion of the Parietes.*—The skin should be protected by an ointment composed of 1 per cent. HCl in zinc oxide cream.

(3) *'Pooling' of the discharge* deeply in the wound. If possible, completely dependent drainage must be instituted; if necessary, the patient must lie prone. If this is not possible, continuous motor suction sump drainage must be carried out (fig. 1102). In any case, the daily loss must be measured.

Spontaneous closure occurs in 90 per cent. of cases, though this may take several months. Failure is usually due to inadequate dependent drainage.

If in spite of all these measures the fistula fails to close, it

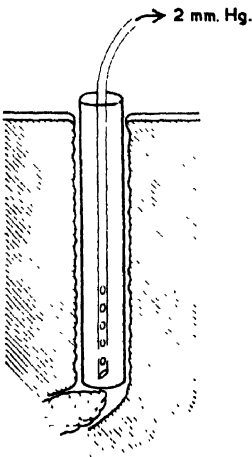


FIG. 1102.—Continuous suction sump drainage for pancreatic fistula.

should be dissected to its source in the pancreas and drained into the stomach or into a Roux-en-Y jejunal loop.

ISLET-CELL TUMOURS OF THE PANCREAS

The islets of Langerhans contain three types of cells: alpha, beta, and gamma, and of these the beta-cell is most common.

Pathology.—The actual islet-cell lesion may be one of the following:

(1) Hyperfunction without morphological change; (2) generalised hyperplasia; (3) discrete adenomata; (4) generalised adenomatosis; (5) carcinomata. Two-thirds of islet lesions occur in the body or tail of the pancreas. Approximately in one-third of cases they are multiple and in one-third they are malignant.

Such lesions may be associated with similar states in other endocrine glands, especially the anterior pituitary, the parathyroids, and the adrenal cortex (multiple adenoma syndrome).

Clinically, in overt cases, there may be: (1) Hyperinsulinism. (2) Features of other endocrine lesions, e.g. enlarged sella turcica, altered phosphorus and calcium blood levels, and increased keto-steroids in the urine. (3) Peptic ulceration. If a patient has peptic ulceration and hyperinsulinism (p. 744), it is certain that he has a multiple adenoma syndrome. (4) Zollinger-Ellison syndrome.

Of these it is only necessary to discuss (1) and (4).

HYPERINSULINISM-BETA-CELL TUMOUR (INSULINOMA) (fig. 1103)

Clinical features are not stereotyped, and unless the attending clinician is alive to the bizarre symptoms to which it gives rise, insulinoma of the pancreas, still somewhat of an enigma, will often continue to be displayed only at that final Court of Appeal—the post-mortem room, for so often the patient is diagnosed as psychoneurotic, epileptic, a malingering, or a sufferer from organic nervous disease.

The attacks, due to hypoglycæmia, come on at irregular intervals and often with progressively increasing frequency and severity. Four stages are recognised, and in the beginning the attacks do not necessarily progress beyond the first or second stages.

Stage 1.—Often the symptoms simulate duodenal ulcer, awakening the patient in the early hours of the morning with vague abdominal discomfort, relieved by carbohydrates. At other times nervous symptoms predominate. There is a vague feeling of being unwell, a disinclination for exertion, and possibly some disorientation.

Stage 2.—Trembling, sweating, dizziness, blurring of vision, and great hunger.

Stage 3.—Sluggish mind, inarticulate speech, inco-ordinated movements, diplopia, sometimes hallucinations.

Stage 4.—Fits indistinguishable from epilepsy¹ passing into semi-consciousness or coma, with dilated pupils

and extreme spasticity. In course of time intellectual deterioration is liable to follow.

Usually the patient is an adult under forty years of age, but a few cases have occurred in children. Because eating relieves the symptoms, over-weight not infrequently results. Pain is not a feature of benign insulinoma, but is present in most cases where a malignant change has occurred.

In a few instances the tumour contains sufficient calcium to show in a radiograph. **Diagnosis.**—The three criteria (Whipple's triad), which, if present, establish the diagnosis, are:

1. An attack, as described above, occurs in the fasting state (i.e. in the morning) or with exercise.

2. During the height of the attack there is a hypoglycæmia below 45 mg. per 100 ml. of blood.

¹ The brain cannot store dextrose, which it requires in large amounts. Deprivation of this essential brings about a burst of nervous energy; the higher centres are affected first.

Paul Langerhans, 1847–1888. Professor of Pathology, Freiburg, Germany.

Robert M. Zollinger, Contemporary. Professor and Chairman, Department of Surgery, Ohio State University, Columbus, Ohio, U.S.A.

Edward Homer Ellison, Contemporary. Associate Professor of Surgery, Columbus, Ohio

Allen Oldfather Whipple, 1881–1963. Professor of Surgery, Columbia University, New York City.



FIG. 1103. — Beta-cell islet tumour of the pancreas.

3. The symptoms are relieved by glucose.

Confirmation depends on the identification of hypoglycæmia. If an islet-cell tumour is present, this will become manifest with fasting food, not water) if need up to be seventy-two hours (fig. 1104).

Treatment.—The only curative treatment is extirpation of the tumour or tumours. An intravenous drip infusion of isotonic dextrose solution is started, laparotomy is performed and the pancreas is displayed. When the tumour (fig. 1105) is not located at once, the pancreas should be mobilised (p. 846) to enhance thorough palpation. In 12 per cent. of cases more than one tumour is present. Once found by palpation, an incision is made in the pancreas over the tumour.

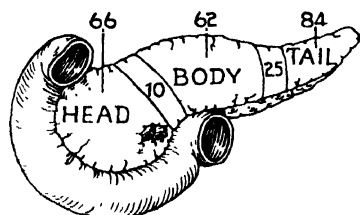
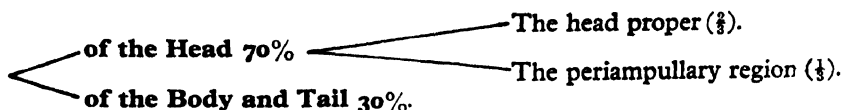


FIG. 1105.—Site of the insulin-secreting adenoma in 254 cases. Seven were situated in an ectopic pancreas. Twenty were overlooked at operation. (J. M. Howard et al., Philadelphia.)

florid, rapidly progressive, and often situated in the upper jejunum. (2) Gastric hypersecretion of the order of 500 ml. per hour. (3) Diarrhoea with accompanying hypokalaemia in 30 per cent. of cases. (4) Occasionally steatorrhoea. The condition can be easily identified by gastric analysis (p. 743) and it should be thought of in every case of recurrent peptic ulceration.

Treatment.—Theoretically, if the tumour can be identified and isolated, all that is required is its removal. There are three great difficulties however: (1) the tumour may be small and impossible to feel or see, (2) it may be multiple, (3) it may be malignant with extra-pancreatic deposits. Zollinger himself recommends total gastrectomy as well as removal of the tumour (which may involve partial pancreatectomy). In any event, during the operation for this condition, constant gastric suction should be maintained through an indwelling stomach tube. As soon as the tumour is removed, gastric secretion falls in amount and acid content (Dragstedt). If no tumour can be recognised after very thorough search, excision of the body and tail of the pancreas is advisable. Multiple small tumours may be present which are unrecognisable to the naked eye and close serial sections will be necessary in order to discover them.

CARCINOMA OF THE PANCREAS



Primary carcinoma of the pancreas accounted for 3,942 deaths in England and Wales in 1959. Most of the victims of the disease are past the meridian

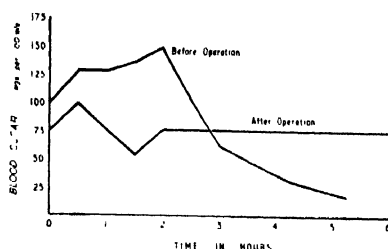


FIG. 1104.—The hypoglycæmia caused by the islet-cell tumour is cured by operation.

As a rule complete enucleation is possible. Drainage of the area is essential. Undue hardness of the tumour calls for partial pancreatectomy, but this measure is required most often because of failure to locate the tumour, in which event more than half the specimens removed reveal no abnormality. Consequently, the result in these instances is not always what is hoped for. In cases where the tumour is removed, 87 per cent. of patients are cured; in 4 per cent. there is slight or no relief. The mortality is about 8 per cent.

Ulcerogenic Tumour—Non-beta-cell Adenoma¹ (Zollinger-Ellison syndrome).—This is a tumour, situated usually in the tail of the pancreas, which secretes a gastrin-like substance. In half of the cases, the tumour is malignant and there may be metastases. The clinical manifestations are: (1) Intractable peptic ulceration which is frequently

¹ So-called because it is likely, but not absolutely certain, that alpha-cells are involved.

of life, the average being fifty-seven years of age ; it is, however, not very unusual to encounter this condition in patients who are in their thirties. Contrary to general belief, men are attacked only a little more often than women.

Pathology :

Usually the lesion is scirrhus, almost gritty to the knife edge, and fibrous in appearance. Occasionally it is medullary ; exceptionally it is a cystadenocarcinoma.

(a) **Carcinoma of the head proper** never attains the size of a fist ; rarely is it more than one-quarter of this size. The reason for this is that while still comparatively small it compresses the common bile duct (fig. 1106), and unless the obstruction is relieved early, death from cholæmia results. As a small growth always lies deeply in the pancreas, obtaining a reliable specimen for biopsy is problematical (p. 864).

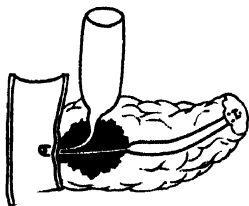


FIG. 1106.—Carcinoma of the head of the pancreas causing obstruction to the common bile duct and the duct of Wirsung.

(b) **Periampullary Carcinoma.**—In less than one-third of cases of carcinoma of the head of the pancreas the neoplasm arises on the duodenal papilla, in the ampulla of Vater, or in the duodenal mucosa adjacent to the papilla. When such an operation or necropsy specimen is examined, it is usually impossible to decide which of the three was the seat of origin—hence they are grouped together under the comprehensive term ‘periampullary.’ It usually presents as jaundice associated with anæmia due to slow bleeding from the neoplasm.

(c) **Carcinoma of the body and tail** tends to attain a greater size than the foregoing varieties ; it also metastasises more widely.

Spread : Local Spread.—Carcinomata belonging to the first and third categories tend to adhere to, and invade, adjacent structures, particularly the duodenum, occasionally the pyloric antrum, and rarely the colon or the kidney.

Lymphatic spread from the head is first to the lymph nodes along the hepatic artery ; that from the body and tail is to the gastric, coeliac, mesenteric, and para-aortic lymph nodes. The supraclavicular lymph nodes are involved less commonly than in cases of carcinoma of the stomach.

Spread by the blood-stream occurs somewhat later. Metastases are carried especially to the liver ; usually the hepatic metastases are small and numerous.

Peritoneal implantation resulting in ascites occurs in 10 per cent. of cases.

Clinical Presentation

Primary carcinoma of the pancreas presents in two distinct ways: (1) As a progressively obstructive jaundice where the growth is in the head of the pancreas. Pain is present but may not be a marked feature and must be carefully enquired for. (2) An unremitting pain in the back and epigastrium where the growth is in the body and tail. Both of these may have associated

symptoms of anorexia, weight loss, or occasional vomiting or diarrhoea—symptoms that may be associated with any pancreatic disease.

(1) **As Obstructive Jaundice.**—This group includes the vast majority of cases, and pruritus due to bile salts in the blood coincides with the onset of jaundice and may be severe. The jaundice usually progresses steadily until the patient is almost green in colour.

An exception to this rule sometimes occurs in cases of periampullary carcinoma. After about a month of progressive (usually painless) jaundice, necrosis of a portion of the growth sometimes occurs, and pent-up bile escapes into the duodenum. Variations in the depth of the jaundice and pyrexia heighten the similarity of the symptoms caused by this neoplasm to those of obstruction of the common bile duct by a gall-stone.

Jaundice may occur when the growth is in the body and tail, but then it is due either to malignant lymph nodes in the portal fissure or metastases in the liver.

Pain.—This must be carefully enquired for. It is usually a steady dull ache in the epigastrium radiating through to the back; it may be colicky and simulate gall-stone pain; sometimes it is paroxysmal radiating from the umbilicus and through to the back. In only 16 per cent. of cases is pain absent and in these the growth is confined to the ampulla. A distended gall-bladder is palpable in 60 per cent. of cases (Courvoisier's law, p. 836). *Anorexia* is present in about 65 per cent. of cases. It is usually not severe.

Loss of weight occurs in 90 per cent. of cases. It averages about 5 lb. (2.3 kg.) per month. Often the patient may not have noticed it. A feeling of profound weakness is not uncommon.

Enlargement of the Liver.—The liver is palpably enlarged in 65 per cent. of cases of carcinoma of the head of the pancreas. As a rule this enlargement is due to biliary obstruction, but it may be due to hepatic metastases.

Acute cholecystitis and/or acute pancreatitis is occasionally the first indication of carcinoma of the pancreas, and in patients of the older age group this possibility should come to mind.

(2) **As Intractable Pain without Jaundice.**—It must be remembered that any patient who complains of intractable pain in the back at the level of the lumbar vertebræ 1 and 2, and especially on the left side, may be suffering from carcinoma of the tail of the pancreas. The usual picture is that of a persistent epigastric or back pain with a normal barium series. The patient usually says that the pain is worse when he lies down and it tends to keep him awake at night. As with most cases of retroperitoneal inflammatory or neoplastic disease of the posterior abdominal wall, the pain is eased by sitting up and leaning forward in bed, and this is the position the patient usually adopts. The pain is not related to food, but a marked feature is that it persists, week in, week out. In such cases a laparotomy should be done as it will probably supply the only means of diagnosis. Unfortunately many of these cases are regarded as having a backache of functional origin. Weakness, loss of weight,

anorexia, and a systolic bruit over the tumour are not pronounced but are commonly present.

Thrombophlebitis migrans may be an indication of the presence of pancreatic carcinoma of either type. The thrombophlebitis appears spontaneously in almost any superficial vein; it resolves, only to re-appear elsewhere. The explanation of this phenomenon is still obscure. It was first described by Trousseau and also occurs in other types of abdominal carcinoma.

Diabetes and Carcinoma of the Pancreas.—(a) Diabetes mellitus is occasionally the first sign of carcinoma of the pancreas. Typical pain may be a pointer or completely absent. The condition should be suspected when a middle-aged person develops diabetes and, in spite of adequate diabetic treatment, continues to lose weight. The sedimentation rate is helpful (Lawrence). In these patients the carcinoma may run a galloping course.

(b) Carcinoma of the pancreas is about ten times more frequent in diabetics than in the population at large.

Calcinosis and Carcinoma of the Pancreas.—While pancreatic calcinosis is relatively rare among patients with pancreatic carcinoma, carcinoma of the pancreas develops in patients with pancreatic calcinosis with such frequency as to warrant the assumption that pancreatic calcinosis is a pre-carcinomatous condition.

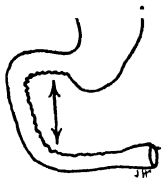


FIG. 1107.—
The pad sign.

Investigations:

Radiography.—It will be appreciated that radiography has little to offer in cases of carcinoma of the body or tail of the pancreas. In the case of carcinoma of the head of the organ sometimes the C of the barium-filled duodenum is widened—the 'pad' sign (so-called because it appears as if the C is crammed with padding) (fig. 1123). On infrequent occasions a periampullary carcinoma

provides a filling defect.

Laboratory Tests:

The *stercobilinogen content of the faeces* is often lower than that found in chronic pancreatitis—less than 10 mg. per day.

Occult blood in the stools is a valuable finding. It is especially frequent in cases of periampullary carcinoma.

Hypoproteinæmia occurs earlier, and more frequently, than it does in carcinoma of the stomach.

The use of *pancreatic enzyme tests* (p. 855) are of doubtful value but the serum lipase level may be elevated whilst other enzymes are normal.

Duodenal Cytological Analysis.—A duodenal aspiration tube is passed, and a radiograph is taken to confirm that the tip of the tube is in the duodenum. Secretin, 100 units in 20 ml. water, is then injected intravenously, and methacholine chloride (a very strong parasympathetic stimulant) 10 mg. is given subcutaneously. Cytological examination of the duodenal aspirate is then made. On the result of this cytological examination a diagnosis of carcinoma has been established on many occasions (Papanicolaou).

When jaundice is present the problem of distinguishing obstructive jaundice from infective hepatitis may be difficult. Liver function tests (p. 793) must be done, several times if necessary. In jaundice due to carcinoma it is nearly always possible to show progressive obstruction. Liver biopsy and trans-hepatic cholangiography p. 845 in these cases are not without risk.

Management:

Exploratory laparotomy is called for under the following circumstances:
(1) Patients presenting with upper abdominal pain and loss of weight which

Armand Trousseau, 1801–1867. Physician, Hôtel-Dieu, Paris, France.

Robert Daniel Lawrence, Contemporary. Consulting Physician, King's College Hospital, London.

George Nicholas Papanicolaou, 1884–1962. Professor of Anatomy, Cornell University, New York.

might be pancreatic in origin, (2) patients presenting with jaundice persisting for a month who have equivocal laboratory evidence. In such cases preliminary vitamin K injections are necessary.

Confirmation of the Diagnosis.—There are two schools of thought as to the need for obtaining positive evidence before resecting the affected part of the pancreas :

School I regards positive biopsy evidence of carcinoma as mandatory before proceeding with radical resection. It admits that biopsy of the pancreas is difficult, but feel that the risks entailed are worth it.

School II maintains that pancreatic biopsy is so often hazardous and unreliable that they prefer to do without it. They regard biopsy of regional lymph nodes, particularly those along the hepatic artery, as more trustworthy, but rely mainly on palpation of the mobilised pancreas in the decision as to whether to proceed with resection.

PANCREATODUODENECTOMY

Assessment of Operability.—A long right paramedian incision is made. Explore the area thoroughly. The presence of lymph nodes, fixity, and liver secondaries completely rule out radical surgery. Expose the duodenum by separating the right half of the transverse colon and the hepatic flexure from their peritoneal attachments and dissect out the common bile duct above the duodenum. Mobilise the duodenum by dividing the peritoneum along its right border so that it can be lifted off the inferior vena cava. Clear the superior mesenteric vessels and make sure that these and the portal vein are not involved in growth. If the growth is operable the operation may proceed. Divide the common bile duct above the duodenum, mobilise the duodenum and pyloric antrum and ligate and divide the right gastric, right gastro-epiploic, and gastro-duodenal vessels. The duodeno-jejunal flexure is exposed and divided and the inferior pancreatico-duodenal vessels are ligated. The superior mesenteric vessels in the region of the uncinate process must be carefully exposed. The veins are very short and it is safer to divide the uncinate process leaving a thin slice of pancreas on the superior mesenteric vein, attending to bleeding points with sutures. Reconstruction is now carried out as in fig. 1108.

The abdomen is now closed, but the areas of the biliary and pancreatic anastomoses must be drained.

Post-operative Treatment.—Transfusion of blood should be continued after the operation, as necessary, and vitamin K injections given for several days.

Mortality and Results.—The general mortality of this extensive procedure is about 20 per cent. The complications are principally those of leakage from the pancreas or common bile-duct anastomoses. Acute dilatation of the stomach is prone to occur, and must be guarded against by gastric aspiration. The five-year survival rate of this operation is disappointingly low for carcinoma of the pancreas proper (6.4 per cent.); when performed for carcinoma of the ampulla the rate is much better (21 per cent.).

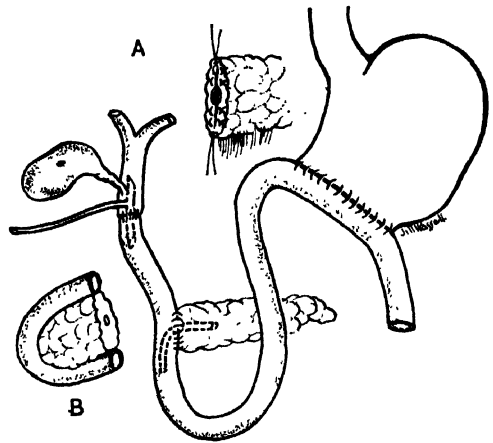


FIG. 1108.—Reconstruction after pancreatoduodenectomy. Inset A, the stump of the body of the pancreas prepared for anastomosis with the jejunum. Inset B, the portion removed. (After H. A. Zintel.)



FIG. 1109.—Cholecyst-jejunostomy.

Palliative Operation.

—In the presence of jaundice when the neoplasm is considered to be inoperable, cholecyst-jejunostomy (fig. 1109) is performed. This measure, by short-circuiting the obstruction of the common bile duct, relieves the jaundice and may give six to nine months of life.

Perhaps the major blessing it confers is relief from the intolerable itching.

From time to time, to the astonishment of all concerned, the patient continues to improve and recovers completely, which shows how closely chronic pancreatitis of the head of the organ can mimic a neoplasm in this situation (fig. 1110).

Treatment of Carcinoma of the Body and Tail of the Pancreas.—When laparotomy is performed before a growth situated towards the tail of the organ has metastasised, it can be resected with comparative ease, and some lasting cures have been reported. The outlook in cases of papillary cystadenocarcinoma is particularly favourable.

Total pancreatectomy is a formidable procedure but has been done for both carcinoma of the pancreas and chronic pancreatitis.

Patients who have undergone total pancreatectomy require surprisingly little insulin—30 to 70 units daily. Pancreatin, 10 to 12 G. daily, is given to improve fat absorption.



FIG. 1110.—Obstructive jaundice with markedly distended gall-bladder (Courvoisier's sign, p. 836). ? carcinoma of pancreas. ? chronic pancreatitis.

CHAPTER 37

THE PERITONEUM, OMENTUM, MESENTERY,
AND RETROPERITONEAL SPACE

Surgical Physiology.—The peritoneal cavity is the largest cavity in the body, the surface area of its lining membrane being nearly equal to that of the skin. This veil-like serous membrane is composed of flattened polyhedral cells, one layer thick, resting upon a thin layer of fibro-elastic tissue, the two layers constituting the peritoneum. Beneath the peritoneum, supported by a small amount of areolar tissue, lies a network of lymphatic vessels and rich plexuses of capillary blood-vessels from which all absorption and exudation must occur. In health, only sufficient peritoneal fluid, which is a pale yellow, somewhat viscid fluid containing lymphocytes and leucocytes, is secreted to ensure that the more mobile viscera glide easily. Under the influence of decreased intra-abdominal pressure, occasioned by the upward movement of the diaphragm during expiration, aided by capillary attraction, this secretion travels continuously in an upward direction to the subdiaphragmatic spaces, where experimental evidence shows that fine coloured particulate matter and bacteria are absorbed rapidly into the subperitoneal lymphatic network. In a matter of minutes these coloured particles reach the lymph nodes *above* the diaphragm.

When there is an outpouring of many ounces of peritoneal fluid, and, perhaps in addition, the contents of the stomach or duodenum escape into the peritoneal cavity through a perforation, it is extremely doubtful if the forces just described are powerful enough to counteract the law of gravity. In all probability, excess of free fluid within a peritoneal cavity unobstructed by adhesions, runs downwards.

The peritoneum is conveniently divided into two parts—the *visceral* surrounding the viscera, and *parietal* lining the rest of the cavity. The parietal portion is richly supplied with nerves and, when irritated, causes severe pain accurately localised to the affected area. The visceral peritoneum, on the other hand, is poorly supplied with nerves and pain arising therefrom is vague and poorly localised (p. 892).

PERITONEOSCOPY

For many years peritoneoscopy has been advocated for inspecting intraperitoneal organs without the necessity of laparotomy, but comparatively few surgeons employ it. The chief value of the method is to visualise the available portion of the liver in cases of hepatomegaly of undetermined origin. Its use has been extended to inspect the gall-bladder and the stomach, and also the uterus and adnexæ, in subjects in whom laparotomy is inadvisable.

The peritoneoscope resembles a cystoscope. The abdomen is prepared as for laparotomy. Preferably under general anaesthesia, an incision $\frac{1}{4}$ inch (6 mm.) long is made in the middle line close to the umbilicus. A small trocar and cannula are pushed into the peritoneal cavity and the requisite amount of air (1 to $1\frac{1}{2}$ litres) is introduced with a pneumoperitoneum apparatus. The patient is tilted so that the air will rise into the upper abdomen or the pelvis, as required. A special trocar and cannula is then introduced, the trocar withdrawn, and the telescope inserted. Biopsy of a particular area of the liver is possible, but hæmorrhage from it is not infrequent. Air embolism is another possible danger.

ACUTE PERITONITIS

Nearly all varieties of peritonitis are due to an invasion of the peritoneal cavity by bacteria. To such an extent is this true that when the term 'peritonitis' is used without qualification, bacterial peritonitis is implied.

Paths of Bacterial Invasion.—

- Direct infection {
 - Via perforation of some part of the gastro-intestinal canal.
 - Through a penetrating wound of the abdominal wall.
 - Post-operative.
- Local extension {
 - From an inflamed intraperitoneal organ, e.g. appendicitis, cholecystitis.
 - Transmigration through damaged gut wall, e.g. unrelieved intestinal obstruction.
 - From or via the Fallopian tubes.
- Blood-stream {
 - Part of general septicæmia.

Even in non-bacterial peritonitis (e.g. intraperitoneal rupture of the bladder or a hæmoperitoneum) the peritoneum soon becomes infected by transmigration of organisms from the bowel, and it is not long (often a matter of hours) before chemical peritonitis becomes a peritonitis in the usual meaning of the term.

Bacteriology is very variable and accounts to a large extent for the mildness or virulence of the peritonitis.

Bacteria from the Alimentary Canal.—Bacteria causing peritonitis are nearly always derived from the alimentary canal. Usually the infection is caused by two or more strains. The commonest invaders are *Escherichia coli*, ærobie and anærobie streptococci, and the bacteroides. Less frequently the *Clostridium welchii* is found; still less frequently staphylococci or *Klebsiella pneumoniae* (Friedländer's bacillus), and so on. Many of the strains of *Esch. coli*, bacteroides, and *Cl. welchii* produce toxins which cause severe illness or death when they invade a large absorptive area.

The Bacteroides¹ are the predominant organisms in the lower intestine, where they greatly outnumber *Escherichia coli*. Only recently has the frequency and importance of the presence of bacteroides in the causation of peritonitis been realised. These Gram-negative, non-sporing organisms often escape detection because they are strictly anærobie, and slow to grow on culture media unless there is an adequate carbon-dioxide tension in the anærobie apparatus (Gillespie). In many laboratories, the culture is discarded if there is no growth in forty-eight hours.

These organisms, which are resistant to penicillin and streptomycin but sensitive to tetracycline and chloramphenicol, are often the cause of peritonitis (in conjunction with other organisms), and suppuration of the abdominal wall following laparotomy.

Bacteria NOT from the Alimentary Canal.—Examples are peritonitis due to the gonococcus, beta-hæmolytic streptococcus, pneumococcus, and the *M. tuberculosis*. In time past hæmolytic streptococcal peritonitis was associated with a high mortality, but since the advent of antibiotics it has lost much of its dreaded lethal properties.

Factors which favour localisation of peritonitis are anatomical and pathological.

Anatomical.—Excluding the subphrenic spaces, which will be considered later, the greater sac of the peritoneum is divided into (a) the pelvis, and (b) the peritoneal cavity proper. The latter is re-divided into a supracolic and an infracolic compartment by the transverse colon and transverse mesocolon,

¹ Bacteroides = bacteria with rounded ends.

Gabriele Falloppio, 1523–1562. Anatomist and Professor of Surgery, University of Padua.
 Theodor Escherich, 1857–1911. Professor of Paediatrics, Vienna.
 William Henry Welch, 1850–1934. Professor of Pathology at the Johns Hopkins University Baltimore.
 Theodor Albrecht Edwin Klebs, 1834–1913. German Bacteriologist.
 Carl Friedländer, 1847–1887. Professor, Berlin-Friedrichshain Hospital.
 Hans Christian Joachim Gram, 1853–1938. Professor of Medicine, Copenhagen.
 William Alexander Gillespie, Contemporary. Professor of Clinical Bacteriology, University of Bristol.

which deter the spread of infection from one to the other. The lower abdomen is divided into a right and a left compartment by the mesentery and the bodies of the vertebræ, both of which hinder the passage of infection across the middle line, but each compartment communicates freely with the pelvis. When the supracolic compartment overflows, as is often the case when a gastric ulcer perforates, it does so over the colon into the right infra-colic compartment, and so, by way of the right paracolic gutter to the right iliac fossa, and thence to the pelvis.

Pathological.—Inflamed peritoneum loses its glistening appearance and becomes reddened and velvety. Flakes of fibrin appear and cause coils of intestine to become adherent to one another and to the parietes. There is an outpouring of serous fluid rich in leucocytes and antibodies, that soon becomes turbid; should the infection not be overcome quickly, and especially if localisation occurs, the turbid fluid becomes frank pus. Peristalsis is retarded in affected coils, and this helps in preventing distribution of the infection to other coils. The greater omentum, by enveloping and becoming adherent to inflamed structures, often forms a substantial barrier to the spread of infection. In short, the clinical course is largely governed by the manner in which adhesions form around the affected organ.

Factors which tend to cause diffusion of peritonitis.

(a) The prime factor in the spread of peritonitis is whether it develops rapidly or slowly. If an inflamed appendix (fig. 1111) or other hollow viscus perforates before the natural defences have had time to come into action, there is a gush of intestinal contents into the peritoneal cavity that spreads over a large area almost instantaneously.

(b) The ingestion of food, or even water, by stimulating peristaltic action, hinders localisation. Violent peristalsis occasioned by the administration of a purgative or an enema, causes a widespread distribution of an infection that would otherwise have remained localised.

(c) When the virulence of the infecting organism is so great as to render the localisation of the infection difficult or impossible.



FIG. 1111.—Sudden perforation, especially if engendered by purgation, often results in an immediate widespread bacterial peritonitis.

CLINICAL FEATURES

Localised peritonitis is bound up intimately with the causative lesion, and the initial symptoms and signs are those of that lesion. When the peritoneum becomes inflamed the temperature, and especially the pulse-rate, rise. The pain increases and usually there is repeated vomiting. The most important sign is rigidity of the abdominal wall over the area of the abdomen

which is involved. In cases of pelvic peritonitis arising from an inflamed appendix in the pelvic position or from salpingitis, the abdominal signs are often slight, deep tenderness of one or both quadrants alone being present, but a rectal or vaginal examination reveals tenderness, often exquisite, of the pelvic peritoneum. With appropriate treatment localised peritonitis usually resolves. In about 20 per cent. of cases an abscess follows. Infrequently, localised peritonitis becomes diffuse. Conversely, in favourable circumstances diffuse peritonitis can become localised, most frequently in the pelvis.

Diffuse (syn. Generalised) Peritonitis.—The most typical example is that caused by perforation of a peptic ulcer. In this instance, the onset of symptoms is followed in a matter of minutes by widespread board-like abdominal rigidity. The commonest cause of diffuse peritonitis is perforation of an inflamed appendix occurring before there has been time for the infection to become walled-off from the general peritoneal cavity. Diffuse peritonitis from any cause may be divided into three stages:

1. *Early.*—This is the stage of diffusing or spreading peritonitis. Pain, which commenced in one part of the abdomen, becomes more widespread; the exception to this rule is post-operative peritonitis, in which pain is almost absent. Vomiting becomes very frequent, bile-stained, and often effortless. The patient lies supine, with his knees flexed. The temperature is usually raised, but in fulminating cases it may be subnormal. A rising pulse-rate, as shown by a two-hourly pulse-chart, is an indication that peritonitis is advancing. On examination the tongue is moist and the face somewhat flushed. Inspection of the abdomen shows that there is little or no respiratory movement of the abdominal wall. Palpation reveals widespread rigidity. On auscultation the abdomen is silent, i.e. no sound of peristalsis can be heard.



FIG. 1112.—The facies in terminal diffuse peritonitis.

2. *Intermediate.*—The third day is the critical one in diffuse peritonitis. In cases where a favourable outcome can be confidently expected, the pulse-rate commences to fall. If not, the rigidity to some extent passes off, and gives place to distension. The whole abdomen is acutely tender. The clinical picture is that of ileus.

3. *Later Stage.*—If by the fourth or fifth day no semblance of localisation has occurred, the patient's condition becomes extremely grave. The whole abdomen is grossly distended (meteorism). The pulse becomes thready and rapid. The eyes are sunken but bright, the nose is pinched, there are sordes on the lips, the tongue is dry and shrivelled, the forehead and the hands are cold and clammy, and the facies drawn and anxious (Hippocratic facies). Finally the patient lapses into semi-consciousness (fig. 1112) and the end is not far distant.

TREATMENT

The essential principles are:

Rest to the Alimentary Canal.—This is achieved by gastric aspiration via a Ryle's or plastic tube (preferably passed transnasally). It is continued until the gastric aspirate is clear, the patient is in 'positive gastric balance' and is passing flatus. Reliance must not be placed on normal bowel sounds alone. (The small intestine is usually the first part of the alimentary canal to regain peristalsis and is followed sometimes thirty-six hours later by the colon and the stomach.) The patient is allowed to drink an ounce of water hourly—this will probably be aspirated later. Nothing else is given by mouth and the bowels are left confined. A flatus tube may be passed but no enemas are given.

In order to assess progress, three daily observations must be made.

(1) The abdomen is auscultated and the girth measured at the umbilicus. When ileus is present a high 'tinkling' note is heard (p. 950). As soon as peristalsis returns, loud borborygmi and explosive bowel sounds are heard. (2) Has the patient passed flatus? (3) Careful assessment must be made of gastric intake and aspiration and the electrolyte balance.

Sedation.—The best drug is Morphine (10–15 mg.) given four-hourly as necessary. This rests the gut, permits localisation of the peritonitis and calms the patient. Contrary to what might be expected, it does not delay, but seems to hasten, return of peristalsis. As soon as bowel sounds are heard the drug should be stopped.

Intravenous Alimentation.—Parenteral fluids and electrolytes must be given to ensure adequate hydration and urinary output. The blood electrolytes should be estimated daily and maintained at a physiological level, especially potassium which tends to fall after the third day (p. 86). If the potassium level is low the return of peristalsis is often delayed. Vitamins in the form of Parenterovite should also be given in the intravenous fluid. Blood transfusion if the hæmoglobin falls below 70 per cent. is valuable, and plasma may be helpful.

Antibiotic Therapy.—A combination of penicillin, 500,000 units, and streptomycin, 0.5 G. twice a day given intramuscularly and continued until the temperature and pulse-rate have been normal for three days, is justly popular. Nevertheless, the surgeon should be prepared to change to tetracycline 0.5 G. in a litre of dextrose-saline given slowly into a vein by the drip method during each period of twelve hours, should the former drugs not prove effective.

Posture.—Fowler's position (fig. 1113), by which the aid of gravity is invoked to direct purulent fluid within the peritoneal cavity towards the pelvis, is not considered so essential as formerly. Nevertheless, during the first forty-eight hours of treatment of peritonitis, most surgeons consider it advisable to prop the patient up with a back-rest and pillows.

The high position, whereby the head of the bed is raised 18 inches (45 cm.) on blocks, has to a large extent been abandoned in favour of the low position, where 6-inch (15 cm.) blocks are employed. In no circumstance should a pillow be placed under the thighs or knees, or a mechanical device (e.g. a 'donkey' or cranking frame) be employed under the mattress to maintain the position—because these devices may

predispose to phlebothrombosis. Frequent changes of position are highly desirable, i.e. the patient is urged to lie on alternate sides. These expedients discourage the development of phlebothrombosis decubiti, and they also facilitate deep respiration.



FIG. 1113.—The bulwarks of the conservative and post-operative treatment of peritonitis: (1) Modified Fowler's position. (2) Transnasal gastric aspiration. (3) Continuous intravenous dextrose-saline. (4) Charts. A two-hourly pulse and temperature chart and a fluid balance chart.

Treatment of the Cause.—If the condition is amenable to surgery, such as in perforated appendicitis, cholecystitis, diverticulitis, peptic ulcer or rarely in perforation of the small bowel, operation must be carried out as soon as the patient is fit. This is usually within a few hours. In peritonitis due to salpingitis, pancreatitis, a perforated uterus in septic abortion, or in cases of primary peritonitis of streptococcal or pneumococcal origin, conservative treatment is the procedure of choice (if the diagnosis can be made with certainty).

The reader is referred for details to the diseases which commonly cause peritonitis, e.g. appendicitis (p. 954), perforated peptic ulcer (p. 751), cholecystitis (p. 829), diverticulitis (p. 900), also pneumococcal and streptococcal peritonitis (pp. 876 to 877).

Peritoneal Toilet.—In surgery for general peritonitis it is essential that after the cause has been dealt with, the whole peritoneal cavity should be explored with the sucker and mopped dry, if necessary until all sero-purulent exudate is removed. It is inadvisable to put in any antibiotic or antiseptic; the peritoneal fluid which is secreted is one of nature's best defence mechanisms and should not be interfered with.

Prognosis.—With modern treatment diffuse peritonitis carries a mortality of about 10 per cent. The lethal factors are (a) bacterial toxæmia, (b) paralytic ileus, and (c) bronchopneumonia.

COMPLICATIONS OF PERITONITIS

All the complications of a severe bacterial infection are possible, but the special complications of peritonitis are as follows:

(1) *Acute intestinal obstruction due to peritoneal adhesions* (p. 944). This usually gives central colicky abdominal pain with evidence of gas confined to the upper portion of the small intestine on X-ray. It is essential to distinguish this from:

(2) *Paralytic ileus* where there is usually little pain and gas is seen distributed throughout the intestines on the X-ray film.

(3) *Residual abscesses*.—Abscess formation following local or diffuse peritonitis usually occupies one of the situations shown in fig. 1114. When palpable, an intraperitoneal abscess should be treated by marking out its limitations on the abdominal wall, and careful daily examination. In the majority of cases, with the aid of antibiotic treatment, the mass becomes smaller and smaller, and finally is impalpable. In others, the abscess fails to resolve, or becomes larger, in which event it must be opened. In many situations, by waiting a few days the abscess becomes adherent to the abdominal wall, so that it can be drained without opening the general peritoneal cavity.

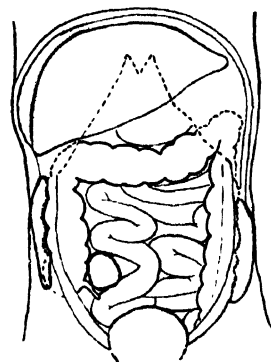


FIG. 1114. — Common situations for residual abscesses.

In the case of a laterally-placed abscess, the incision is made on the lateral side of the swelling. The layers of the abdominal wall are divided until the peritoneum is reached. With the finger, the extraperitoneal tissues are separated from the peritoneum until the abscess is opened. A drainage tube is inserted; if the path is tortuous, a Penrose drain (a thin tube of latex rubber containing a wick of gauze) is valuable.

PELVIC ABSCESS

The pelvis is the commonest site of an intraperitoneal abscess, because the vermiform appendix is often pelvic in position and also the Fallopian tubes are frequent foci of infection. A pelvic abscess can also occur as a sequel to any case of diffuse peritonitis. Pus can accumulate in this area without serious constitutional disturbance, and unless the patient is examined carefully from day to day, such abscesses may attain considerable proportions before being recognised. The most characteristic symptoms of a pelvic abscess are diarrhoea and the passage of mucus in the stools. It is no exaggeration to say that the *passage of mucus, occurring for the first time in a patient who has, or is recovering from, peritonitis, is pathognomonic of pelvic abscess*. Rectal examination reveals a bulging of the anterior rectal wall which, when the abscess is ripe, becomes softly cystic. Left to Nature, a proportion of these abscesses



FIG. 1115. — Opening a pelvic abscess into the rectum.

burst into the rectum, after which the patient nearly always recovers rapidly. It is too hazardous to wait for this possible happy termination. A pelvic abscess should be drained deliberately. In certain cases, notably those where the primary focus is in the Fallopian tubes, vaginal drainage through the posterior fornix is chosen. In other cases, where the abscess is definitely pointing into the rectum, rectal drainage (fig. 1115) is employed. If any uncertainty exists as to the presence of pus, an aspirating needle introduced through the rectal wall into the bulging swelling will settle the question. Drainage of a pelvic abscess into the rectum is exceedingly efficacious in selected cases, but occasionally, in the case of a large abscess which can be palpated above the pubes, after the bladder has been emptied by catheterisation, lower laparotomy should be undertaken in order to be quite certain of the diagnosis. Provided the abscess is shut off from the general peritoneal cavity, a point which can be ascertained unambiguously when the abdomen has been opened, rectal drainage of a pelvic abscess is preferable to suprapubic drainage, which in many cases unavoidably breaks down Nature's barriers and exposes the general peritoneal cavity to the dangers of spreading infection.

SUBPHRENIC ABSCESS

Anatomy.—The complicated arrangement of the peritoneum results in the formation of four intraperitoneal and three extraperitoneal spaces in which pus may

Charles Bingham Penrose, 1862–1926. Professor of Gynecology, University of Pennsylvania, Philadelphia.

collect. Three of these spaces are on either side of the body, and one approximately in the midline (fig. 1116).

Left Anterior Intraperitoneal, bounded above by the diaphragm, behind by the left triangular ligament and left lobe of the liver, the gastro-hepatic omentum and

anterior surface of the stomach. To the right is the falciform ligament, and to the left the spleen, gastrosplenic omentum, and diaphragm. The common cause of an abscess here is following operations on the stomach, the tail of the pancreas, the spleen or the splenic flexure of the colon or in diverticulitis.

Left Posterior intraperitoneal is another name for the 'lesser' sac. The commonest type of suppuration here is the pancreatic pseudo-cyst (p. 858). In practice a perforated gastric ulcer rarely causes a collection here, because the potential space is obliterated by adhesions.

Right Anterior intraperitoneal, which lies between the right lobe of the liver and the diaphragm. It is limited posteriorly by the anterior layer of the

coronary and the right triangular ligaments, and to the left by the falciform ligament. Common causes here are perforating cholecystitis, a perforated duodenal ulcer, a duodenal cap 'blow out' (p. 759) and appendicitis.

Right Posterior intraperitoneal (*syn.* Rutherford Morison's kidney pouch) lies transversely beneath the right lobe of the liver. It is bounded on the right by the right lobe of the liver and the diaphragm. To the left is situated the foramen of Winslow, and below this lies the duodenum. In front are the liver and gall-bladder, and behind, the upper part of the right kidney and diaphragm. The space is bounded above by the liver, and below by the transverse colon and hepatic flexure. It is the deepest space of the four and the commonest site of a subphrenic abscess which usually arises from appendicitis, cholecystitis, a perforated duodenal ulcer or following upper abdominal surgery.

Extraperitoneal.—There are three of these:

Right and Left extraperitoneal which is the term sometimes given to perinephric abscess (p. 1107).

Midline extraperitoneal which is another name for the 'bare' area of the liver which may develop an abscess in amœbic hepatitis (p. 798) (the commonest cause) or it may be a pyogenic liver abscess (p. 797).

The extraperitoneal abscesses are usually not called subphrenic at all and are best considered under their appropriate sections in the book.

Clinical Features.—The symptoms and signs of subphrenic infection are frequently obscure, and it is well to remember the aphorism, "Pus somewhere, pus nowhere else, pus under diaphragm".

Symptoms.—A common history is that when some infective focus in the abdominal cavity has been dealt with, the condition of the patient improves temporarily, but after an interval of a few days or weeks, symptoms of toxæmia reappear. Owing to rapid absorption of toxins, the condition of the patient steadily, and often rapidly, deteriorates. Sweating, wasting, and anorexia are present. There is sometimes epigastric fullness, abdominal

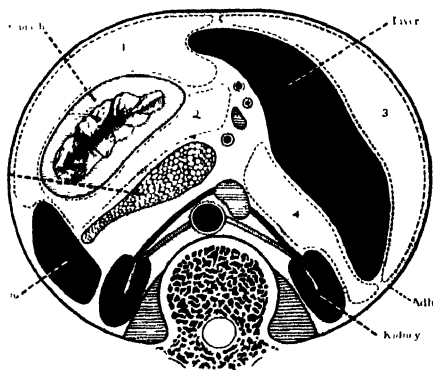


FIG. 1116.—Transverse section through the lesser sac of the peritoneum showing situations which extraperitoneal subphrenic abscesses may occupy. 1. left anterior space. 2. left posterior space (lesser sac). 3. right anterior space which becomes shut off by adhesions from 4, right posterior space.

discomfort, or pain in the shoulder on the affected side, owing to irritation of sensory fibres in the phrenic nerve, referred along the descending branches of the cervical plexus. Persistent hiccup may be a presenting symptom.

Signs.—If the abscess is anterior, abdominal examination will reveal some tenderness, rigidity, or even a palpable swelling. Sometimes the liver is displaced downwards, but more often it is fixed by adhesions. Examination of the chest is important, and in the majority of cases collapse of the lung or evidence of effusion or empyema is to be found.

Accessory Investigations.—(i) *Blood Count.*—A relative and absolute leucocytosis is the rule.

(ii) *X-ray.*—A plain radiograph sometimes demonstrates the presence of gas (fig. 1117) or a pleural effusion. On screening, the diaphragm is often seen to be elevated (so-called 'tentcd' diaphragm) and its movements impaired.

(iii) *Needling* is the final court of appeal.

If the needle has penetrated a subphrenic abscess, the movements of the diaphragm are transmitted to the needle, which consequently oscillates during respiration. In the case of an empyema these movements are absent. Needling should never be performed except in the operating theatre, so that if pus is discovered the needle is left *in situ* as a guide to the abscess, and the operation performed forthwith.

Differential Diagnosis.—Pylephlebitis, tropical abscess, pulmonary collapse, and empyema give rise to most of the diagnostic difficulties.

Treatment.—Many cases of subphrenic infection do not proceed to suppuration, and resolution is encouraged by antibiotic therapy. The clinical course of suspected cases is watched, and blood and radiological examinations are made at suitable intervals. If suppuration seems probable, surgical intervention is indicated.

If a swelling can be detected in the subcostal region or in the loin, an incision is made over the site of maximum tenderness, or over any area where œdema or redness is discovered. The parietes usually form part of the abscess wall, so that contamination of the general peritoneal cavity is unlikely.

If no swelling is apparent, the subphrenic spaces should be explored from behind after removal of the *outer half* of the twelfth rib. This step will ensure that the diaphragm is incised below the pleural reflection. After the fibres of the diaphragm have been separated a finger is inserted beneath the diaphragm so as to explore the adjacent area. This method of approach obviates opening either the pleura or peritoneal cavity.



FIG. 1117.—Radiograph in erect position showing subphrenic abscess containing gas.

SPECIAL FORMS OF ACUTE PERITONITIS

Meconium Peritonitis.—Meconium is a sterile mixture of epithelial cells, mucin, salts, fats, and bile, and is formed when the fœtus commences to swallow amniotic fluid. By the third month of intra-uterine life the upper third of the small intestine has become filled with meconium; by the fourth month the accumulation has reached the ileo-caecal valve; during the remainder of intra-uterine life the colon becomes increasingly filled.

Meconium peritonitis is an aseptic peritonitis which develops late in intra-uterine life or during, or just after, delivery. Meconium enters the peritoneal cavity through an intestinal perforation, and in over 50 per cent. of cases the perforation is the result of some form of neonatal intestinal obstruction; in the remainder no cause for the perforation is discernible. When meconium, which is sterile, enters the peritoneal

cavity an exudate is secreted that organises rapidly; matting of intestinal loops occurs, and in many cases in a matter of weeks the extruded meconium becomes calcified.

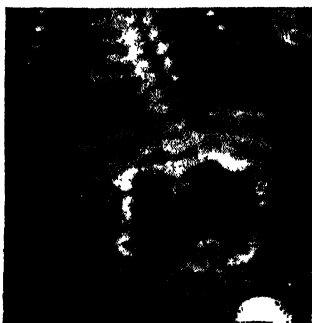


FIG. 1118.—Meconium peritonitis. Note: Free air and fluid in the peritoneal cavity; intra-abdominal calcification [x] and on the spleen; air in the small intestine; microcolon shown by a barium enema. (Dr. Jack Lester, Copenhagen.)

Meconium remains sterile until about three hours after birth; thereafter, unless the perforation has become sealed, sterile meconium peritonitis gives place to acute bacterial peritonitis which, unless treated promptly, is rapidly fatal.

Clinical Features.—Meconium peritonitis should always be considered when a baby is born with a tense abdomen. There is vomiting, and failure to discharge meconium. The differential diagnosis between neonatal intestinal obstruction and peritonitis is, in many cases, virtually impossible; indeed, in half the cases both are present. Free fluid in the peritoneal cavity is often sufficient to give a fluid thrill.

Radiography (fig. 1118).—Free air in the peritoneal cavity, an abundant quantity of abdominal fluid, fluid levels, calcification (often most distinct on the surface of the liver or the spleen, and most readily seen in a lateral view) are characteristic findings, all of which are unlikely to be present in every case. Meconium peritonitis has been diagnosed by radiography of the foetus in utero two days before birth.

Treatment.—The prognosis is bad, but recovery has followed prompt operation in a few cases. The greatest chance of survival is in those patients who have an intestinal perforation but no intestinal obstruction, in which case closing the perforation and draining the peritoneal cavity is all-sufficient, and can be performed expeditiously.

Pneumococcal Peritonitis.—There are two forms of this disease :

1. Primary.
2. Secondary to pneumonia.

Primary pneumococcal peritonitis is much the more common. The patient is often an under-nourished girl between three and six years of age, and it is probable that the infection sometimes occurs via the vagina and Fallopian tubes, for pneumococci have been cultured from patients' vaginae. At other times, and always in males, doubtless the infection is blood-borne from the upper respiratory tract or the middle ear. After the age of ten years pneumococcal peritonitis is most unusual. Children with nephrosis are more liable to this condition than others. During the past thirty years the incidence of pneumococcal peritonitis has declined greatly and the condition is now rare. Perhaps this is on account of the greater cleanliness and higher standard of living of the poorer classes.

Clinical Features.—The onset is sudden, and the earliest symptom is pain localised to the lower half of the abdomen. The temperature is raised to 103° F. (39.8° C.) or more, and there is usually frequent vomiting. Should an inguinal hernia be present it is likely to be distended, but the contents are easily reduced. After twenty-four to forty-eight hours profuse diarrhoea, occasionally blood-stained, is characteristic. There is usually increased frequency of micturition. The last two symptoms are due to severe pelvic peritonitis. Herpes on a lip or nostril is often present. In acute forms of the disease, even in cases where there is no involvement of a lung, there is a tinge of cyanosis of the lips and cheeks, and movement of the alæ nasi is often discernible. On examination rigidity is usually bilateral, and is less than in most cases of acute appendicitis.

Differential Diagnosis.—A leucocytosis of 30,000 or more with approximately 90 per cent. polymorphs speaks more for pneumococcal peritonitis than appendicitis.

Even so, it is often impossible, especially in males, to exclude perforated appendicitis. The other condition which is extremely difficult to differentiate from primary peritonitis in its early stage is pneumonia. An unduly high respiratory rate and the absence of abdominal rigidity are the most important signs supporting the diagnosis of pneumonia, which is usually clarified by a radiograph of the thorax.

Treatment.—*Early operation* is always required. Under local infiltration anaesthesia if the child's condition is poor, a short paramedian incision is made over the right rectus abdominis. The peritoneum is incised. Should the exudate be odourless and sticky, the diagnosis of pneumococcal peritonitis is practically certain, but it is desirable to perform a routine laparotomy to exclude other lesions. Assuming that no other cause for the peritonitis is discovered, some of the exudate is removed with a syringe, and sent to the laboratory for culture and sensitivity tests. Thorough peritoneal toilet is carried out and the incision closed. The patient is returned to bed and antibiotic and fluid replacement therapy continued.

Primary streptococcal peritonitis of infants and children is rather more frequent than the foregoing but still uncommon. When a streptococcus is the infecting organism, the peritoneal exudate is thin, slightly cloudy, and contains flecks of fibrin. From the standpoints of clinical aspects and treatment, streptococcal peritonitis in infants and children does not differ from those detailed in the account of pneumococcal peritonitis (*vide supra*), but the mortality is higher.

Idiopathic streptococcal peritonitis in adults is fortunately rare, for prior to the antibiotic era it was nearly always fatal, and the mortality is still very high. Rightly, in early cases the abdomen is opened, usually on a diagnosis of acute appendicitis. In streptococcal peritonitis the peritoneal exudate is odourless, thin, contains small flecks of fibrin, and may be blood-stained. In these circumstances pus is removed by suction, the abdomen closed with suprapubic drainage, and the measures detailed in the conservative treatment of peritonitis carried out.

Peritonitis following Abortion Parturition.—The abortionist has usually pushed an instrument through the uterine vault and streptococcal peritonitis follows. When peritonitis follows puerperal infection, it is a notifiable disease. It is more common after first deliveries. Rigidity is seldom much in evidence; this, at any rate in part, is due to the stretched condition of the abdominal musculature. The lochia may be offensive but not necessarily so. Diarrhoea is common.

Treatment.—Provided the infection is limited strictly to the pelvis, the correct treatment is rest for the alimentary canal, intravenous fluids, the required antibiotic, and attention to electrolyte balance. Posterior colpotomy (p. 873) may be necessary if a pelvic abscess forms. If the peritonitis is generalised, the patient is usually extremely ill and drainage is advisable. This is best carried out by making a small suprapubic incision under local anaesthesia and inserting a drain. This can be done with the patient in bed, if necessary.

In the pre-antibiotic era the mortality of general peritonitis following parturition or abortion was at least 50 per cent. ; with antibiotic therapy and timely operation, the mortality has fallen to less than 10 per cent. (Brews).

Post-operative Peritonitis.—It is hard to decide whether the signs are due to operative trauma or to infection. Rigidity, one of the mainstays of the recognition of other forms of peritonitis, is frequently in abeyance. Tenderness, though present, is likely to be attributed to the recent incision. More likely than not, a narcotic has been administered and this, of course, masks the signs, indefinite as they are. Absence of bowel sounds, abdominal

distension (which is slight in fulminating cases) and the bile-stained gastric aspirate will almost certainly be thought to be due to paralytic ileus. There is often evidence of post-operative pulmonary collapse, to which can be attributed the rise in pulse and temperature.

Peritonitis after abdominal operations occurs more often than is realised. The principles of treatment do not differ from those of peritonitis of other origin. Antibiotic therapy *per se* is insufficient; no antibiotic can stay the onslaught of bacterial peritonitis due to leakage from a suture line.

Periodic peritonitis is characterised by abdominal pain and tenderness, mild pyrexia, polymorphonuclear leucocytosis, and occasionally pain in the thorax and joints. The duration of an attack is twenty-four to seventy-two hours, when it is followed by complete remission, but exacerbations recur at regular intervals. Most of the patients have undergone appendicectomy in childhood. The disease, often familial, is limited principally to Arabs, Armenians, and Jews; other peoples occasionally are affected. At laparotomy, which may be necessary to exclude other causes, the peritoneum—particularly in the vicinity of the spleen and the gall-bladder—is inflamed. There is no evidence that the interior of these organs is abnormal.

Differential Diagnosis.—Patients with abdominal epilepsy do not have positive physical signs or pyrexia, and their attacks are usually controlled by anti-convulsive medication.

The ætiology of periodic peritonitis is unknown, and no form of treatment has been found to be of the slightest avail.

Bile Peritonitis.—Unless there is reason to suspect that a bile duct was damaged at an operation in which drainage was not provided, it is improbable that bile as a cause of peritonitis will be thought of until the abdomen has been opened and bile is seen therein. The common causes of bile peritonitis are: (1) following biliary surgery—damage to the common bile duct, slipping of a ligature on the cystic duct, leakage from a divided accessory bile duct in the gall bladder bed or dislodgement of a drain in the post-operative phase; (2) following perforation or gangrene of the gall bladder or leakage from a choledochus cyst; and (3) following gastro-duodenal surgery, e.g. duodenal cap 'blow out' (p. 759) or leakage from a suture line.

Unless the bile has extravasated slowly, and the collection becomes shut off from the general peritoneal cavity there are signs of diffuse peritonitis with a degree of shock. After a few hours a tinge of jaundice is not unusual. Local drainage, and when necessary suprapubic peritoneal drainage, is imperative, and if performed early enough, these measures will often save the patient's life. When bile is seen issuing from a perforation of some part of the biliary tree, a drainage tube should be passed through the opening and secured there. The gall-bladder, if present, should be drained. A ruptured duodenum must be drained; it is too œdematous to repair.

TUBERCULOUS PERITONITIS

Acute Tuberculous Peritonitis.—It is doubtful if tuberculous peritonitis is ever acute. However, tuberculous peritonitis sometimes has an onset that resembles so closely acute peritonitis that the abdomen is opened. Straw-coloured fluid escapes, and tubercles¹ are seen scattered over the peritoneum and greater omentum. Tubercles occasionally simulate fat necroses (p. 850) or the nodules of peritoneal carcinoma.

¹ Early tubercles are greyish and translucent. They soon undergo caseation, and appear white or yellow, and are then less difficult to distinguish from carcinoma.

matosis (p. 882). On opening the abdomen and finding tuberculous peritonitis, the fluid is evacuated, a portion of the diseased omentum is removed for histological confirmation of the diagnosis, and the wound closed without drainage.

At other times, although acute abdominal symptoms arise, the presence of ascites makes the diagnosis of acute tuberculous peritonitis reasonably evident.

Chronic Tuberculous Peritonitis.—Although the incidence of tuberculous peritonitis has declined in Britain, in many parts of the world where measures for preventing tuberculosis (especially the disease in cows) are enforced less strictly, the condition is still common.

Usually children are affected, but it is not rare for the disease to make its first appearance in early adult life, when females outnumber males by two to one. Exceptionally the disease becomes manifest in patients over forty years of age.

Origin of the infection:

1. From tuberculous mesenteric lymph nodes.
2. From tuberculosis of the ileo-cæcal region.
3. From a tuberculous pyosalpinx.
4. Very occasionally it is due to a blood-borne infection from pulmonary tuberculosis.

There are four varieties of tuberculous peritonitis :

1. **Ascitic Form.**—The peritoneum is studded with tubercles, and the peritoneal cavity becomes filled with pale, straw-coloured fluid. The onset is insidious. There is loss of energy, facial pallor, and some loss of weight. The patient is usually brought for advice because of enlargement of the abdomen (fig. 1119). Pain is often completely absent ; in other cases there is considerable abdominal discomfort which may be associated with constipation or diarrhoea. On inspection dilated veins can be seen coursing beneath the skin of the abdominal wall. Shifting dullness can be elicited readily. In the male child congenital hydroceles sometimes appear, due to the patent processu vaginales becoming filled with ascitic fluid from the peritoneal cavity. Because of the increased intra-abdominal pressure, an umbilical hernia commonly occurs. On abdominal palpation a transverse solid mass can often be detected (fig. 1120). This is rolled-up greater omentum infiltrated with tubercles.

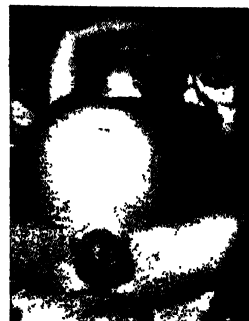


FIG. 1119.—Tuberculous peritonitis, ascitic form. The patient has also left-sided tuberculous epididymitis. (F. H. Roberts, F.R.C.S., Edinburgh.)



FIG. 1120.—Physical signs recorded in a case of the ascitic form of tuberculous peritonitis.

The diagnosis is seldom difficult, except when it occurs in an acute form or when it first appears in an adult, in which case it has to be differentiated from other forms of ascites. A positive Mantoux test in a child with ascites strongly suggests, and a negative test is good evidence against, tuberculosis. In adults this test is of negligible value. The diagnosis of tuberculous peritonitis having been made, it is always important to look for tuberculous disease elsewhere, and in this respect the possibility of tuberculous salpingitis in females should be remembered.

The fluid is pale yellow, usually clear, and rich in lymphocytes. The

specific gravity is comparatively high, often 1020 or over. Even after centrifugation, rarely can the *mycobact. tuberculosis* be found, but its presence can be demonstrated by culture or by guinea-pig inoculation.

Treatment.—Sanatorium treatment is helpful.

Chemotherapy.—A combination of streptomycin, P.A.S. and isoniazid is given as detailed on p. 23. If the general condition is good, the patient can return home and, if an adult, to light work, before the course of chemotherapy has been completed.

2. **Encysted (syn. Loculated) form** is similar to the above, but one part of the abdominal cavity alone is involved. So is produced a localised intra-abdominal swelling which gives rise to difficulty in diagnosis. In a female above the age of puberty, when the swelling is in the pelvis, an ovarian cyst will probably be diagnosed. In the case of a child it is sometimes difficult to distinguish the swelling from a mesenteric cyst. For these reasons laparotomy is often performed, and if an encapsulated collection of fluid is found, it is evacuated and the abdomen is closed. The general treatment already detailed is required, but the response to this treatment is more rapid. Late intestinal obstruction is a possible complication.

3. **Fibrous (syn. plastic) form** is characterised by the production of widespread adhesions, which cause coils of intestine, especially the ileum, to become matted together and distended. These distended coils act as a 'blind loop' (p. 897) and give rise to steatorrhœa, wasting and attacks of abdominal pain. On examination the adherent intestine with omentum attached, together with the thickened mesentery, may give rise to a palpable swelling or swellings. The first intimation of the disease may be subacute or acute intestinal obstruction. Sometimes the cause of the obstruction can be remedied easily by the division of bands. Lateral anastomosis between an obviously dilated loop and a collapsed loop of small intestine should not be done or the 'blind loop' syndrome (p. 897) is a certain outcome. If the adhesions are accompanied by fibrous strictures of the ileum as well, it is best to excise the affected bowel, provided not too much of the small intestine needs to be sacrificed. If adhesions only are present a Noble plication operation (p. 951) or intestinal intubation (p. 951) should be done. This is a dangerous condition and the surgeon must adhere to strict physiological principles in its treatment. Fortunately, chemotherapy after adequate surgery will rapidly bring it under control.

4. **Purulent form** is rare. When it occurs, usually it is secondary to tuberculous salpingitis. Amidst a mass of adherent intestine and omentum, tuberculous pus is present. Sizeable cold abscesses are wont to form, and point on the surface, commonly near the umbilicus, or burst into the bowel. In addition to prolonged general treatment, operative treatment may be necessary for the evacuation of cold abscesses and possibly for intestinal obstruction. If the patient survives long enough to overcome the infection, it may be possible to close a faecal fistula, which otherwise usually persists because of obstruction distal to it. Closure must therefore be combined with some form of anastomosis between the segment of intestine above the fistula and an unobstructed area below. The prognosis of this variety of tuberculous peritonitis is relatively poor.

PERITONEAL BANDS AND ADHESIONS

Congenital bands and membranes occur in the peritoneum as described in textbooks of anatomy. None of them cause intestinal obstruction except an obliterated vitello-intestinal duct (p. 1026).

Inflammatory Peritoneal Adhesions p. 944.

Talc Granuloma.—Talc (silicate of magnesium) should never be used as a lubricant for rubber gloves for it is a cause of peritoneal adhesions and granulomas in the Fallopian tubes. Potassium bitartrate, which is completely soluble, is free from these serious objections.

ASCITES

Ascites, an excess of serous fluid within the peritoneal cavity, can be recognised clinically only when the amount of fluid present exceeds 1,500 ml. ;

in the obese a greater quantity than this is necessary before there is clear evidence of the presence of intra-peritoneal fluid.

Clinical Features.—The abdomen is distended evenly, with fullness of the flanks, which are dull to percussion. Usually shifting dullness is present, but when there is a very large accumulation of fluid, this sign is absent. In such cases, on flicking the abdominal wall, a characteristic fluid thrill is transmitted from one side to the other. In the female, ascites must be differentiated from an enormous ovarian cyst (fig. 1121).

Type 1. Due to Congestive Heart Failure.—This, the commonest cause of ascites, is due to chronic venous stasis in the thoracic segment of the inferior vena cava, and consequent obstruction to the venous outflow from the liver. There is stasis also in the superior vena cava, as evinced by engorgement of the veins of the neck—a striking sign in this condition. The ascitic fluid is light yellow serum of low specific gravity, about 1.010.

Type 2. Due to Hepatic Cirrhosis.—In cirrhosis of the liver there is obstruction to the venous outflow of the liver due to obliterative fibrosis of the intrahepatic venous bed (p. 805).

Type 3. Due to Tuberculous Peritonitis (p. 878).

Type 4. Secondary Carcinoma of the Peritoneum.—Again the ascites is due to excessive outpouring, this time due to irritation of the peritoneum by the parasitic neoplastic cells. The fluid is dark yellow and frequently blood-stained. The specific gravity is high—1.020 or over. Microscopical examination often reveals cancer cells.

Type 5. Chronic Constrictive Pericarditis (syn. *Pick's Disease*).—In addition to the peritoneal effusion, effusions occur into the pleural cavity. These effusions are due to engorgement of the venæ cavæ consequent upon diminished capacity of the right side of the heart.

Type 6. Due to depletion of blood protein consequent upon albuminuria or starvation. The ascites in this instance is due to alterations in the osmotic pressure of the capillary blood.

Type 7. Meigs' Syndrome.—This is ascites and pleural effusion associated with solid fibroma of the ovary. The ascites disappears when the tumour is excised.

Treatment.—Repeated tapplings of the ascitic fluid lead to loss of valuable protein, and tend to induce low serum sodium levels as the fluid re-accumulates. Particularly when ascites is due to a non-malignant condition or when specific treatment is not possible, medical treatment is frequently successful. Dietary sodium restriction to 200 mg. per day will usually control ascitic fluid formation, and will sometimes lead to prompt diuresis



FIG. 1121.—The absence of any cause for ascites made the diagnosis of ovarian cyst probable in this case. Diagnosis confirmed by operation.

and abatement of the ascites. Many patients, however, do not have diuresis when such a regimen is instituted, but continue to harbour ascitic fluid which, however, does not increase in amount. In selected cases an effort is made to increase its excretion with diuretics used with great care. Some patients with hepatic cirrhosis benefit from porta-caval shunt (p. 814) if the above measures fail. If abdominal distension becomes severe, paracentesis (tapping) sometimes becomes necessary.

Paracentesis Abdominis.—The bladder having been emptied by a catheter, under local anæsthesia puncture of the peritoneum is carried out with a moderate-sized trocar and cannula at one of the points shown in fig. 1122. In cases where the effusion is due to cardiac failure the fluid must be evacuated slowly. In other circumstances this precaution is unnecessary. If the cannula becomes blocked with fibrin, it is cleared with a stylet. After the fluid has been evacuated the puncture is sealed, and a tight binder is applied to the abdomen.



FIG. 1122.—Usual points of puncture for tapping ascites. The bladder must be emptied by a catheter before the puncture is made. Note the relationship of the sites of puncture to the inferior epigastric artery.

prevent its adhesion to the ileal

Permanent Drainage of Ascitic Fluid.—In rare cases where ascites accumulates rapidly after paracentesis, and the patient is otherwise fit, permanent drainage of the ascitic fluid renders the patient more comfortable. A number of procedures have been described, the best of which is probably the operation of ileo-cntectomy (segmental eversion of the ileum). A 15-inch (40-cm.) loop of isolated terminal ileum is opened along its anti-mesenteric border, turned inside out and sutured with catgut in the triangular area of posterior abdominal wall between the root of the mesentery and the right colon. The ileal mucosa then absorbs the ascitic fluid (Neumann). To prevent its adhesion to the ileal mucosa, the greater omentum should be removed.

PERITONEAL LOOSE BODIES

Peritoneal loose bodies almost never cause symptoms. Occasionally one or more are found in a hernial sac. The probable origin of a peritoneal loose body is an appendix epiploica that has undergone axial rotation, followed by necrosis of its pedicle, and detachment. These hyaline bodies rarely attain the size of a pea.

NEOPLASMS OF THE PERITONEUM

Carcinoma peritonei is a common terminal event in many cases of carcinoma of the stomach, colon, ovary, or other intraperitoneal organ and also of the breast. The peritoneum, both parietal and visceral, is studded with secondary growths, and the peritoneal cavity becomes filled with clear, straw-coloured, or blood-stained ascitic fluid.

The main forms of peritoneal metastases are :

1. Discrete nodules (fig. 1123). This is by far the most common variety.
2. Plaques varying in size and colour.
3. Diffuse adhesions. This form occurs at a late stage of the disease, and gives rise, sometimes, to a 'frozen pelvis'.



FIG. 1123.—Peritoneal metastatic nodules secondary to carcinoma of the pelvic colon. (Owen Daniel, F.R.C.S., Sheffield.)

Gravity probably determines the distribution of free malignant cells within the peritoneal cavity. Cells not caught in peritoneal folds along the attachments of mesenteries gravitate into the pelvic pouches or into a hernial sac, enlargement of which is occasionally the first intimation of the condition. Implantation occurs also on the greater omentum, the appendices epiploicæ, and the inferior surface of the diaphragm.

Differential Diagnosis.—Early discrete tubercles common in tuberculous peritonitis are greyish and translucent, and closely resemble the discrete nodules of peritoneal carcinomatosis, but the latter feel hard when rolled between the finger and the thumb, making the differential diagnosis tolerably simple. Fat necroses usually can be distinguished from carcinomatous nodules by their opacity. Peritoneal hydatids can also simulate malignant disease after rupture of a hydatid cyst.

It is remarkable how often patients riddled with intraperitoneal carcinoma preserve their nutrition, and look and feel comparatively well until the terminal stage.

Treatment.—Ascites due to carcinomatosis of the peritoneum can often be considerably ameliorated by instillations of radio-active gold.

Radio-active gold (^{198}Au) has a half-life of two and a half days, and is supplied as a purple colloidal solution. One hundred millicuries or more of the solution are introduced into the peritoneal cavity after paracentesis. To improve distribution the foot of the bed is raised; then the patient lies on one side and then on the other, and finally prone, each for fifteen minutes. There follows a period of nausea, but approximately half the patients so treated are benefited for a time. The treatment is of no avail if, after paracentesis, the secondary deposits can be palpated, as the isotope can penetrate only to a depth of 1 millimetre.

Radio-active chromic phosphate (^{32}P) is also supplied as a colloidal solution, and is as effective as gold. Not only is it less expensive, but this solution requires none of the troublesome precautions connected with the protection of personnel. It emanates pure beta radiation, and has a longer half-life, viz. 14.3 days. In its administration rubber gloves are the only protection needed.

Pseudomyxoma Peritonei.—This rare condition occurs more frequently in females. The abdomen is filled with a yellow jelly, large quantities of which are often more or less encysted. The condition arises in one of two ways: more often from rupture of a pseudomucinous cyst of the ovary, less often from rupture of a mucocele of the appendix. The condition is painless, and there is no impairment of general health for a long time. When the condition arises from the appendix the mass is often more localised, but in cases of ovarian origin the whole peritoneal cavity is involved. Although an abdomen distended with what seems to be fluid that cannot be made to shift should suggest the possibility, it is highly improbable that a correct pre-operative diagnosis will be made. At laparotomy masses of jelly are scooped out, and the primary focus, if it can be found, is removed. Unfortunately, recurrence is usual. When pseudomyxoma peritonei arises from a mucocele of the appendix, repeated recurrence is less common. Pseudomyxoma peritonei is locally malignant but does not give rise to metastases. Occasionally the condition responds to radioactive isotopes, which certainly should be employed in recurrent cases.

THE GREATER OMENTUM

Rutherford Morison called the greater omentum 'the abdominal policeman.' Relatively larger and structurally more substantial in the adult than in the child, the discharge of its life-saving constabulary duties becomes more effective after puberty, and remains unabated throughout life. The greater omentum attempts, often successfully, to limit intraperitoneal in-

fective and other noxious processes (fig. 1124). For instance, an acutely inflamed appendix is often found wrapped in omentum, and this saves many a patient from developing diffuse peritonitis. Some sufferers from

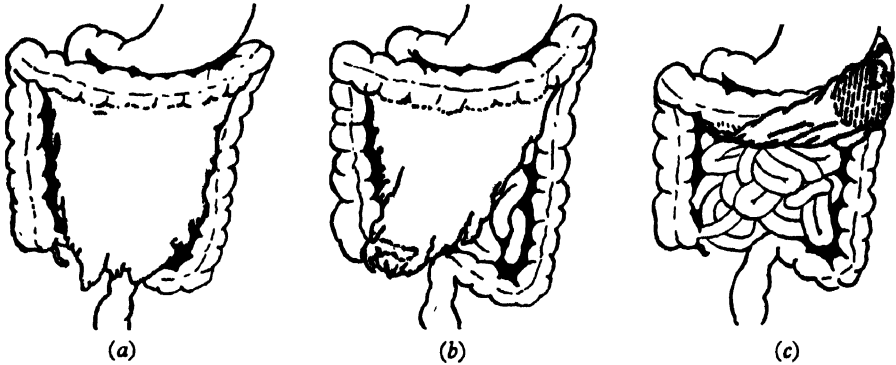


FIG. 1124.—The greater omentum. (a) Normal. (b) In appendicitis. (c) In a (comparatively small) laceration of the spleen.

herniæ are also greatly indebted to this structure, for it often plugs the neck of a hernial sac and prevents a coil of intestine from becoming strangulated.



FIG. 1125.—Torsion of the greater omentum. Specimen removed by operation. Actual size. (Archibald Ronald, F.R.C.S., Barrow-in-Furness.)

Apart from a small portion of it becoming gangrenous while performing the last-mentioned duty (strangulated omentocoele), this good Samaritan of the peritoneal cavity seldom itself becomes diseased; when it does become overwhelmed, as in tuberculous peritonitis and carcinomatosis peritonei, it becomes rolled like a scroll.

Torsion of the Omentum.—Torsion of the omentum (fig. 1125) is a rare emergency, and consequently is seldom diagnosed correctly. It is usually mistaken for appendicitis with somewhat abnormal signs. It may be primary or secondary to an adhesion of the omentum, to an old focus of infection, or to a hernia. Successive herniations of a portion of the omentum into a hernial sac of irregular bore are credited with giving the necessary stimulus to omental torsion.

The patient is most frequently a middle-aged, obese male. A tender lump may be present in the abdomen. The blood supply having been jeopardised, the twisted mass sometimes becomes gangrenous, in which case bacterial peritonitis soon follows.

Treatment.—The abdomen having been opened, the pedicle above the twist is ligated securely and the mass removed.

Omental Cyst (p. 890).

THE MESENTERY

A wound of the mesentery can follow a severe abdominal contusion¹, and is a cause of hæmo-peritoneum. In about 60 per cent. of cases the

¹ If a car accident occurs when a seat belt is worn, sudden deceleration can result in a torn mesentery. This possibility should be borne in mind particularly as multiple injuries may distract attention from this injury.

mesenteric laceration is associated with a rupture of the intestine. If the tear is a large one, and especially if it is transverse (fig. 1126), the blood supply to the neighbouring intestine is cut off, and a limited resection of

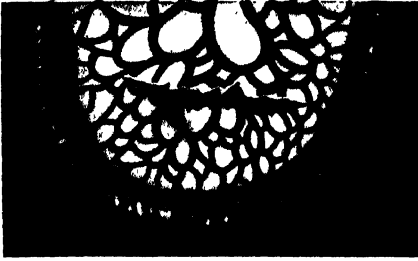


FIG. 1126

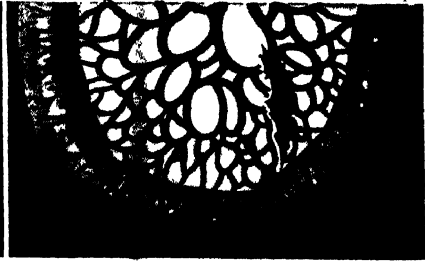


FIG. 1127

Laceration of the mesentery. A transverse tear (fig. 1126) often imperils the blood supply of a segment of intestine, making resection necessary. A longitudinal tear (fig. 1127) can be closed by suture.

gut is imperative. Small wounds and wounds in the long axis (fig. 1127) should be sutured.

Torsion of the Mesentery (see Volvulus Neonatorum, p. 937, and Volvulus of the Small Intestine, p. 943).

Embolism and Thrombosis of the Mesenteric Vessels (p. 948).

ACUTE NON-SPECIFIC MESENTERIC ADENITIS

Ætiology.—Non-specific mesenteric adenitis was so named to distinguish it from specific (tuberculous) mesenteric adenitis. It is now very much commoner than the tuberculous variety. Despite much investigation, the ætiology remains unknown. As so often happens in other inflammatory diseases when no causative bacterium can be found, an unidentified virus is blamed. In about 25 per cent. of cases a respiratory infection precedes an attack of acute non-specific mesenteric adenitis. In spite of the fact that the vermiform appendix is not diseased in this condition, which is definitely recurrent, appendicectomy does reduce the incidence of further attacks, perhaps by removing what is sometimes known as 'the abdominal tonsil'. This self-limiting disease is never fatal.

Living Pathology.—There is a small increase in the amount of peritoneal fluid. As seen and felt between the leaves of the mesentery, the mesenteric lymph nodes are enlarged, being firmly elastic and usually about the size of a haricot bean. In very acute cases they are distinctly red, and many of them are the size of a walnut. The nodes nearest the attachment of the mesentery are the largest. The nodes are not adherent to their peritoneal coats, and if a small incision is made through the overlying peritoneum, a node is extruded easily. The adenitis is most in evidence in the lower third of the mesentery.

Clinical Features.—During childhood, acute non-specific mesenteric adenitis is a common condition, the ratio of acute appendicitis to acute

non-specific adenitis being about 3:1. It is rare after puberty and it is often possible to make a correct pre-operative diagnosis in children, particularly if several cases occur over a short period of time. Admittedly it is often difficult to rule out the possibility of anomalous acute or subacute appendicitis in the isolated case. The typical history is one of attacks of abdominal colic, worse on rolling over, presumably due to drag on the mesentery. Vomiting is usual, but there is no alteration of bowel habit.

On Examination.—There are spasms of general abdominal colic, usually referred to the umbilicus, with intervals of complete freedom, which never appertains in obstructive appendicitis. The patient seldom looks ill. In more than half the cases the temperature is elevated; in severe examples it exceeds 101°F. (38.3°C.). Abdominal tenderness is greatest along the line of the mesentery. When present, shifting tenderness is a valuable sign for differentiating the condition from appendicitis. After lying the patient on the left side for a few minutes, the maximum tenderness moves to the left of the original site.

The pelvic peritoneum is tender to rectal palpation in 30 per cent. of cases. The neck, axillæ, and groins should be palpated for enlarged lymph nodes—if these nodes are enlarged, brucellosis¹ should come to mind (p. 887).

Leucocyte Count.—There is often a leucocytosis of 15,000 or more on the first day of the attack, but this falls on the second day.

Treatment.—When the diagnosis can be made with assurance, bed rest for a few days is the only treatment necessary. If at a second examination, an hour or two after confinement to bed, acute appendicitis cannot be excluded, it is safer to perform appendicectomy.

TUBERCULOSIS OF THE MESENTERIC LYMPH NODES



FIG. 1128. — Massive tuberculosis of the lymph nodes of the mesentery.

Tuberculous mesenteric lymphadenitis is considerably less common than acute non-specific lymphadenitis, and it has become increasingly less frequent in Britain during the past thirty years. Tubercle bacilli, usually but not necessarily bovine, are ingested, and enter the mesenteric lymph nodes by way of Peyer's patches. It is possible for one draught of raw milk to start the infection; it is equally possible that a toddler can become infected with human tubercle bacilli by placing one dust-covered small object in its mouth. Sometimes only one lymph node is infected; usually there are several; occasionally massive involvement occurs (fig. 1128).

Presentation:

1. Demonstrated Radiologically.—The shadows cast by one or more calcified tuberculous lymph nodes are seen frequently in a plain radiograph

¹ Sir David Bruce described Malta Fever in 1887.

*Johann Conrad Peyer, 1653–1712. Professor of Medicine, Schaffhausen, Switzerland.
Sir David Bruce, 1855–1931. Major-General, Royal Army Medical Service.*

of the abdomen (fig. 1129). Often the shadow cast by such a lymph node or nodes is situated in the ileo-cæcal region, but nearly as many are displayed along the line of attachment of the mesentery. Usually the radiological characteristics are unmistakable. Each node is round or oval, not homogenous, but mottled, and its outline is not regular, but bosselated like a blackberry. Calcification of these lymph nodes occurs at the earliest in eighteen months. It is often assumed that because a tuberculous lymph node is calcified, the infection is necessarily defunct. Especially in children, this sweeping assumption is not factual. Firstly, radio-opaque lymph nodes (fig. 1129) do not become white in a single night,¹ and secondly, calcification does not eliminate the presence of uncalcified tuberculous sister lymph nodes.

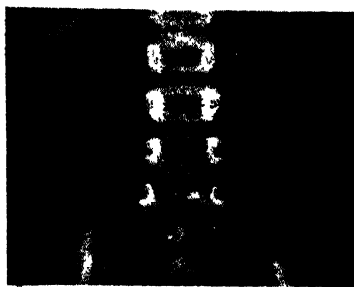


FIG. 1129.—Radiograph showing calcified tuberculous lymph nodes of the mesentery. (Dr. H. R. E. Wallis, Bath.)

2. **As a Cause of General Symptoms.**—Less frequently the tuberculous process is more active, and gives rise to general symptoms. The patient, usually a child under ten years of age, loses appetite, looks pale, and there is some loss of weight; sometimes evening pyrexia occurs. In children with these symptoms, especially those who live in the country, if the Mantoux test is negative, brucellosis, the ‘disease of mistakes,’ should be thought of, and an intradermal test with brucellin performed.

3. **As a Cause of Abdominal Pain.**—Sometimes abdominal pain is the cause of the patient being brought for advice; usually this pain is central, not severe, but rather a discomfort, and is often constant. On examination the abdomen is somewhat protuberant and there is tenderness on deep pressure to the right of the umbilicus. In these circumstances the condition resembles acute non-specific mesenteric lymphadenitis. On deep palpation inflamed mesenteric lymph nodes are sometimes palpable as firm, discrete, tender bean-like objects most frequently to the right of and near the umbilicus. In both conditions a normal leucocyte count favours tuberculosis, and in a child a positive Mantoux test is confirmatory evidence of tuberculosis.

4. **Symptoms Indistinguishable from those of Appendicitis.**—On occasions the abdominal pain is acute and may be accompanied by vomiting. This, combined with tenderness and some rigidity in the right iliac fossa, makes the diagnosis from subacute appendicitis almost impossible. When, as is sometimes the case, the tuberculous infection of the mesenteric lymph nodes becomes reactivated in adolescent or adult life, the diagnostic difficulties are even greater. A radiograph may show calcified lymph nodes, but as such a condition can co-exist with appendicitis, in some cases laparotomy for

¹ My hair is grey, but not with years,
Nor grew it white
In a single night,
As men's have grown from sudden fears :
Byron, *The Prisoner of Chillon*.

appendicectomy and visualisation of the lymph nodes is necessary. If the mesentery is found to be in an inflamed state with caseation of some of the lymph nodes, the diagnosis of active tuberculosis of the nodes is confirmed.

Treatment is similar to that of other surgical tuberculosis (p. 530).

Most cases subside, but from time to time a local abscess forms, usually in the right iliac fossa, when the tuberculous pus should be evacuated and the abdomen closed without drainage (see Pseudo-mesenteric Cyst, below).

Tuberculous Mesenteric Lymph Nodes as a Cause of Intestinal Obstruction.—Remote, rather than recent, tuberculous mesenteric adenitis can be the cause of intestinal obstruction. For instance, a coil of small intestine becomes adherent to a caseating node, and is thereby angulated (fig. 1130), or a free coil may become imprisoned in the tunnel beneath the site of adherence and the mesentery.



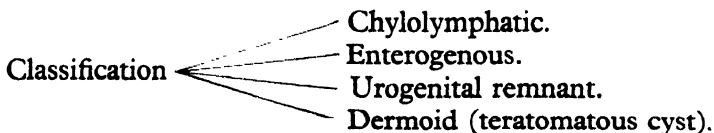
FIG. 1130.—Obstruction by angulation. The gut had become adherent to a tuberculous lymph node.

As a Cause of Pseudo-mesenteric Cyst.—When tuberculous mesenteric lymph nodes break down, the tuberculous pus may remain confined between the leaves of the mesentery, and a cystic swelling having the characteristics of a mesenteric cyst is found. When such a condition is confirmed at operation the tuberculous pus should be

aspirated without soiling the peritoneal cavity, the wound closed, and general treatment continued until the infection has been overcome.

Calcifying Mesenteric Lymph Nodes as a Confusing X-ray Shadow.—Sometimes the shadow cast by a calcified mesenteric lymph node or nodes simulates that of a ureteric or renal calculus. A change of posture often causes a lymph-node shadow to alter in position. Pyelography clarifies the diagnosis in doubtful cases.

MESENTERIC CYSTS



Cysts arising from a urogenital (Wolffian or Mullerian) remnant are essentially retroperitoneal, but they are included in the classification because it is not impossible for such a cyst to project forward into the mesentery.

The following, while not being mesenteric cysts in the academic meaning of the term, give rise to the same physical signs. From the practical point of view they are mesenteric cysts:

Serosanguineous cyst is probably traumatic in origin, but a history of an accident is seldom obtained.

Tuberculous Abscess of the Mesentery (see above).

Hydatid Cyst of the Mesentery.

Chylolymphatic cyst, the commonest variety of mesenteric cyst, probably arises in congenitally misplaced lymphatic tissue that has no efferent communication with

the lymphatic system: it arises most frequently in the mesentery of the ileum. The thin wall of the cyst, which is composed of connective tissue lined by flat endothelium, is filled with clear lymph or, less frequently, with chyle varying in consistency from watered milk to cream. Occasionally the cyst attains a great size (fig. 1131). More often unilocular than multilocular, a chylolymphatic cyst is almost invariably solitary, although there is an extremely rare variety in which myriads of cysts are found in the various mesenteries of the abdomen. A chylolymphatic cyst has a blood supply independent of that of the adjacent intestine, thereby enucleation is possible without the necessity of resection of gut.

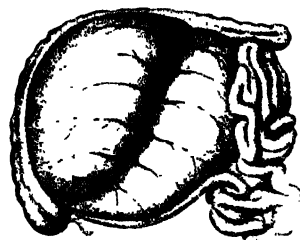


FIG. 1131.—A large chylolymphatic mesenteric cyst. (Higgins and Lloyd.)



FIG. 1132.—A mesenteric cyst moves freely in the direction of the arrows, i.e. at right angles to the attachment of the mesentery.

Enterogenous cyst is believed to be derived either from a diverticulum of the mesenteric border of the intestine which has become sequestered from the intestinal canal during embryonic life, or from a duplication of the intestine. An enterogenous cyst has a thicker wall than a chylolymphatic cyst, and it is lined by mucous membrane, sometimes ciliated. The content is mucinous, and is either colourless or yellowish-brown from bygone hæmorrhage into the cyst. As can be seen at operation, the muscle in the wall of an enterogenous cyst and the bowel with which it is in contact have a common blood supply; consequently removal of the cyst always entails resection of the related portion of intestine.

Clinical Features of a Mesenteric Cyst.—A mesenteric cyst is encountered most frequently in the second decade of life, less often between the ages of one and ten years, and infrequently in infants under one year.

The patient presents on account of:

(a) *A painless abdominal swelling.*—A cyst of the mesentery presents characteristic physical signs.

1. There is a fluctuant swelling near the umbilicus.
2. The swelling moves freely in a plane at right angles to the attachment of the mesentery (fig. 1132).
3. There is a zone of resonance around and, classically, a belt of resonance across the cyst.

(b) *Recurrent attacks of abdominal pain* with or without vomiting. The pain results from recurring temporary impaction of a food bolus in a segment of bowel narrowed by the cyst, or possibly from torsion of the mesentery.

(c) *An acute abdominal catastrophe* arises as a result of (1) torsion of that portion of the mesentery containing the cyst; (2) rupture of the cyst, often due to a comparatively trivial accident; (3) hæmorrhage into the cyst; (4) infection.

Radiography.—In most instances the patient should be submitted to X-ray after a barium meal. The hollow viscera will be found to be displaced around the cyst, and not infrequently some portion of the lumen of the small intestine will be narrowed. In order to exclude or confirm the diagnosis of a hydronephrosis an excretory pyelogram should not be omitted. In cases of painless enlargement of the abdomen this examination should be undertaken first.

Treatment.—As has been indicated already, many chylolymphatic cysts can be enucleated *in toto*.

When, after aspiration of about half the contents of the cyst, the major portion of the cyst has been dissected free but one portion abutting on the intestine or a major

blood-vessel seems too dangerous to remove, this portion can be left attached and its lining destroyed by diathermy.

In the case of an enterogenous cyst, enucleation must not be attempted. If a comparatively short segment of the intestine is involved, resection of the cyst with the adherent portion of the intestine, followed by intestinal anastomosis, is the correct course. Should a very large segment of small intestine be implicated, an anastomosis should be made between the apex of the coil of small intestine and the cyst wall which, in this instance, holds sutures well.

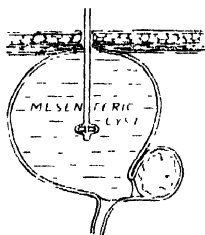


FIG. 1133.—Method of treating a large mesenteric cyst by marsupialisation.

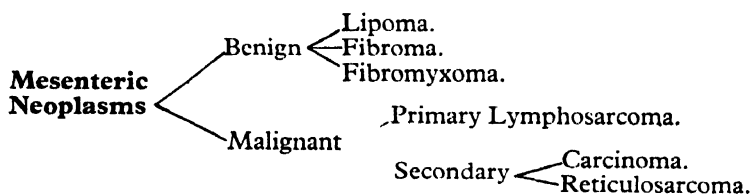
The older treatment of marsupialisation of a mesenteric cyst has little to recommend it, for a fistula or recurrence sometimes results. Occasionally, however, on account of its simplicity, it is advisable in a poor-risk subject (fig. 1133).

Omental cyst occurs nearly as frequently as a mesenteric cyst. Preoperative differentiation is possible because a lateral radiograph shows the cyst in front of the intestines. Treatment is omentectomy.

Cyst of the mesocolon is uncommon, and it is differentiated from a mesenteric cyst only at operation. The treatment is similar.

NEOPLASMS OF THE MESENTERY

Tumours situated in the mesentery give rise to physical signs similar to those of a mesenteric cyst, the sole exception being that they sometimes feel solid.



A benign tumour of the mesentery is excised in the same way as an enterogenous mesenteric cyst, i.e. with resection of the adjacent intestine. When possible, a malignant tumour of the mesentery is subjected to the same treatment. In inoperable cases radiotherapy can be employed if the biopsy specimen reveals that the growth is probably radio-sensitive.

THE RETROPERITONEAL SPACE

Pus or blood in the retroperitoneal space tends to track to the corresponding iliac fossa. If a retroperitoneal hæmatoma or an abscess develops, it should be evacuated by an incision through the abdominal wall, meticulously avoiding opening the peritoneum. Should the retroperitoneal collection be found at laparotomy, it must be drained by a counter-incision in the flank.

Retroperitoneal Cyst.—A cyst developing in the retroperitoneal space often attains very large dimensions, and has at first to be distinguished from a hydro-nephrosis. Even after the latter condition has been eliminated by pyelography, a retroperitoneal cyst can seldom be diagnosed with certainty from a retroperitoneal tumour, until displayed at operation. The cyst may be unilocular or multilocular. Many of these cysts are believed to be derived from a remnant of the Wolffian duct, in which case they are filled with clear fluid. Others are teratomatous, and are filled with sebaceous material.

Excision of these and other retroperitoneal swellings is best performed through a transperitoneal incision (see below).

Idiopathic Retroperitoneal Fibrosis.—See p. 1099.

PRIMARY RETROPERITONEAL NEOPLASMS

Although neuroblastomas and ganglioneuromas of the adrenal gland are retroperitoneal tumours, they have now been segregated as clinical entities, and have been dealt with on p. 583.

Retroperitoneal lipoma, in the first instance, is usually mistaken for a hydro-nephrosis, a diagnosis which is ruled out by pyelography. Women are more often affected. These swellings sometimes reach an immense size. We have removed such a tumour weighing 5½ lb. (2.5 kgm.), and much larger specimens have been recorded. A retroperitoneal lipoma sometimes undergoes myxomatous degeneration, a complication which does not occur in a lipoma in any other part of the body. Moreover, a retroperitoneal lipoma sometimes becomes malignant (liposarcoma) (fig. 1134).

Retroperitoneal sarcoma presents signs similar to a retroperitoneal lipoma. The patient may seek advice on account of a swelling or because of indefinite abdominal pain. On other occasions the tumour, by pressure on the colon, causes symptoms of subacute intestinal obstruction. On examination a smooth fixed mass, which is not tender, is palpated. The most probable original diagnosis is that of a neoplasm of the kidney. This is ruled out by pyelography. The ureter, however, is liable to become displaced by the tumour. Exploratory laparotomy should be performed, and when possible the tumour is removed. Often it is found widely disseminated in the retroperitoneal space, rendering complete removal impossible, in which case a portion is excised for microscopy. Even when excised at a comparatively early stage, recurrence always takes place, and these tumours must be looked upon as being necessarily fatal. Deep X-ray therapy sometimes keeps recurrences in abeyance for a time.

Removal of a Retroperitoneal Cyst or Neoplasm.—After the anterior abdominal wall has been opened and the diagnosis of a retroperitoneal tumour has been confirmed, the incision is extended as necessary. The small intestine is packed away in the upper abdomen, and the cæcum and the sigmoid are relegated to their respective fossæ. The posterior peritoneum is then incised throughout its length over the area to be exposed, the incision paralleling the left border of the aorta. The peritoneum is dissected from the tumour which is removed as completely as possible, the intestines being kept out of the way with packs or exteriorised temporarily, depending on which manœuvre is the more useful.

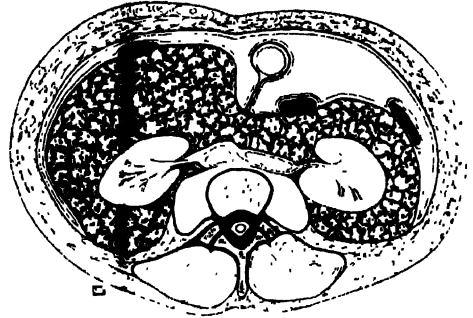


FIG. 1134.—Rapidly growing retroperitoneal liposarcoma.

CHAPTER 38

THE INTESTINES

Abdominal Pain arising from the Alimentary Canal is of two types—

1. *Visceral Pain*.—The alimentary tract is primarily a midline structure with a bilateral nerve supply. Although rotation about the midline occurs during development, nevertheless true visceral pain is referred to the midline as illustrated in fig. 1135. It is dull and poorly localised. The presence of a bilateral nerve supply may

account for contralateral pain from some diseased structures as with left sided pain in cholecystitis.

2. *Peritoneal Pain* however is of the somatic type—and is much more precise, more severe and localised to site of origin. These components account for the changes in character and site of pain which occurs in appendicitis.

Surgical Anatomy.

—It is of great practical importance to be able:

1. To distinguish various portions of the intestinal canal at sight.
2. To know in which part of the abdomen the upper coils, as opposed to the lower coils, of the small intestine lie in relationship to the anterior abdominal wall.
3. To be able to decide which is the proximal and which the distal end of any coil under consideration.

4. To distinguish irrefutably large from small intestine.

For practical purposes these problems are settled as follows :

(a) The mesentery of the jejunum has only two series of arches of blood-vessels, whereas the lower ileum has several series of arches.

(b) Monks's method of intestinal localisation roughly indicates the disposition of the upper, middle, and lower thirds of the small intestine (fig. 1136).

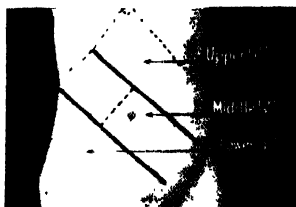


FIG. 1136.—Monks's method of localising the small intestine upon the surface.

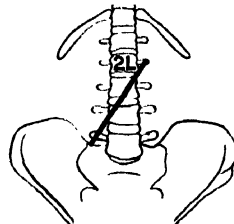


FIG. 1137.—The attachment of the mesentery.

(c) The mesentery, after being made taut, is examined. As the mesenteric attachment runs from left to right (fig. 1137), if palpation reveals that the mesentery is not twisted, then the upper end of the bowel in the wound is the proximal end. Such

1 test is useful, but not as easy to perform in the living as in the dissecting-room subject.

(d) As the 'small' intestine is sometimes found enormously distended and the 'large' intestine entirely collapsed, size is no criterion. The large intestine is characterised by its *tæniæ coli* and appendices epiploicæ.

CONGENITAL MALFORMATIONS OF THE INTESTINES

Congenital Atresia of the Duodenum (p. 936).

Congenital Atresia of the Small Intestine (p. 937).

Volvulus Neonatorum (p. 937).

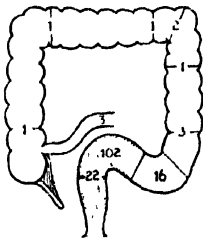
Failure of Descent of the Cæcum.—The cæcum remains under the right lobe of the liver—a normal situation of the structure in the mangabey monkey. This anomaly is associated with displacement of the vermiform appendix.

MEGACOLON

There are two varieties of this condition (1) *primary* or true megacolon (*syn.* Hirschsprung's disease, congenital aganglionic megacolon) and (2) *secondary* or acquired megacolon (fig. 1138).

Primary Megacolon.—*Hirschsprung's disease*:

Pathology.—This disease is characterised by enormous dilatation and hypertrophy of the pelvic colon, sometimes extending into the descending colon but rarely involving the more proximal parts of the large intestine. The pelvic mesocolon is elongated and thickened and its blood-vessels are large and prominent. All coats of the dilated intestine show gross pathological changes. The mucous lining is chronically inflamed and frequently ulcerated. There is a terminal constricted, non-hypertrophied segment of bowel involving the anal canal, the rectum, and a variable part of the large intestine. In nine out of ten cases the upper limit of the contracted segment is the pelvi-rectal junction; occasionally the deficiency extends to a






GANGLION CELLS		
normal	few	absent
		
dilated hypertrophied segment	cone 2-3 cms long	spastic segment 4-26 cms long

FIG. 1139.—The extent of the aganglionic segment in 152 cases of Hirschsprung's disease treated by operation at the Hospital for Sick Children, Great Ormond Street. (After G. G. Wyllie.)

FIG. 1140.—Showing the essential pathological histology in congenital Hirschsprung's disease. (After M. Bodian.)

higher level (fig. 1139). It is in this contracted segment that physiological obstruction¹ lies, and the dilatation hypertrophy of the colon above is due to absence of peristalsis in the spastic segment.

On histological examination the cause of the immotility of the spastic segment is evident; there is a complete absence of parasympathetic ganglion cells, and this ganglion deficiency extends for a distance of 0.4

to 2 inches (1 to 5 cm.) into a transitional zone or cone (fig. 1140) between the terminal spastic segment and the hypertrophied portion (Bodian)

Above the transitional zone, parasympathetic ganglion cells are present as in normal intestine.

Clinical Features.—Hirschsprung's disease shows a familial tendency. It is much more common in males than females. In 90 per cent. of cases symptoms appear within three days following birth.



FIG. 1141.—Hirschsprung's disease showing enormous colonic dilatation and a wave of visible peristalsis. (The late Sir Denis Browne, F.R.C.S., London.)

Constipation.—The infant fails to pass meconium during the first two or three days of life, and then only after the insertion of a little finger or a tube into the rectum. Subsequently motions are tooth-paste-like, and inadequate in amount; straining is in evidence during their passage.

Abdominal distension is usually unmistakable by the third day. In a proportion of cases the abdominal distension progresses, and sometimes it is evident that the colon is obstructed (fig. 1141); in others it is impossible clinically to differentiate large from small intestinal obstruction. Loud borborygmi and visible peristalsis are much in evidence.

Rectal Examination.—The anus is free from fissures and excoriation and there is no perianal soiling. The rectum is empty and *grips the examining finger*.

Complete intestinal obstruction occurs quite frequently within a few days of birth and may be fatal. As a rule, the attacks are recurring and relief is given by a small enema, by passing a greased examining finger or by the spontaneous passage of a large stool sometimes followed by diarrhœa. Owing to the enormous abdominal distension, chest infection may supervene. In any case, if the child survives, malnutrition and stunted growth are obvious features together with enormous abdominal distension.

Radiography (p. 921). **Barium Enema.**—When the clinical findings are atypical, a barium enema is often helpful in confirming the diagnosis. demonstrate the contracted aganglionic segment, if such be present, and to this end preliminary wash-outs are withheld. For reasons explained below, the barium should be admixed with normal saline solution, not tap-water. By using only a little dilute barium emulsion—just sufficient to run over the fœcal masses—a good outline of the bowel can be obtained (fig. 1142).

Biopsy of the ano-rectal wall is employed in order to prove or disprove the presence of ganglia in the myenteric plexus. A small incision is made through the mucosa between the columns of Morgagni to secure a specimen of the circular muscle fibres, together with the longitudinal fibres externally. The presence or absence of ganglion cells between these layers can then be determined. The indications for biopsy are:

1. When the barium enema findings are inconclusive.
2. When the whole colon, or the greater part thereof, is spastic.
3. When the symptoms include diarrhœa instead of the conventional constipation.

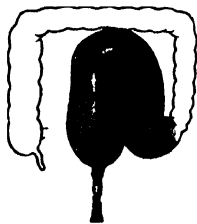


FIG. 1142.—Radiological appearances in Hirschsprung's disease; coning, as well as dilatation, is diagnostic. (After B. C. H. Ward.)

Laparotomy to establish the diagnosis is required when the patient has acute-on-chronic intestinal obstruction, and other causes cannot be eliminated. If the case proves to be one of Hirschsprung's disease it is advisable to establish a temporary transverse colostomy or, in the rare event of the whole colon being spastic, an ileostomy.

Pre-operative Treatment.—Colonic lavage in Hirschsprung's disease is dangerous, because the use of tap-water in the enemas in this condition may cause water intoxication. The megacolon absorbs water much more rapidly than does a normal colon, and the disturbance of electrolytic balance, especially of sodium, may prove fatal. Provided the general condition is good, normal saline solution can be used with safety. In patients with impaired cardiac or renal reserve, 7 per cent. gelatine solution should be substituted. Unless measurements show that distension is increasing, four weeks' pre-operative preparation is desirable (Swenson). During the last five pre-operative days, Sulfasuxidine is given for bowel sterilisation.

Treatment.—The only curative treatment of Hirschsprung's disease is excision of the entire aganglionic segment. No reparative operation should be done till the child is 18 lb. in weight (Brown).

Operation.—The patient is placed in a semi-lithotomy position, to give good access to the abdomen and the peri-anal region. Through a suitable incision the rectum, the contracted part of the adjoining colon, and the commencement of the hypertrophied portion are freed from their attachments as far downwards as possible towards the anal canal. In contrast to excision for carcinoma of the rectum, the dissection must be kept immediately outside the fascia propria. In this way the seminal vesicles and the autonomic nerve trunks to the bladder are protected. Should doubt exist as to whether the aganglionic segment has been encompassed, frozen section biopsy will provide the answer. The intestine is then transected at an indubitably motile level, and the distal end is closed with a purse-string suture. The mobilised aganglionic segment is then everted through the anus (fig. 1143(a) and (b)). The mucosa thus exposed is painted with antiseptic solution, and dried. The anterior half of the junction of the inverted rectum with the anal canal is opened transversely, and the proximal colon is pulled through the opening (fig. 1143(c)). End-to-end anastomosis between the colon and the anal canal is carried out (fig. 1143(d)) as the everted agang-

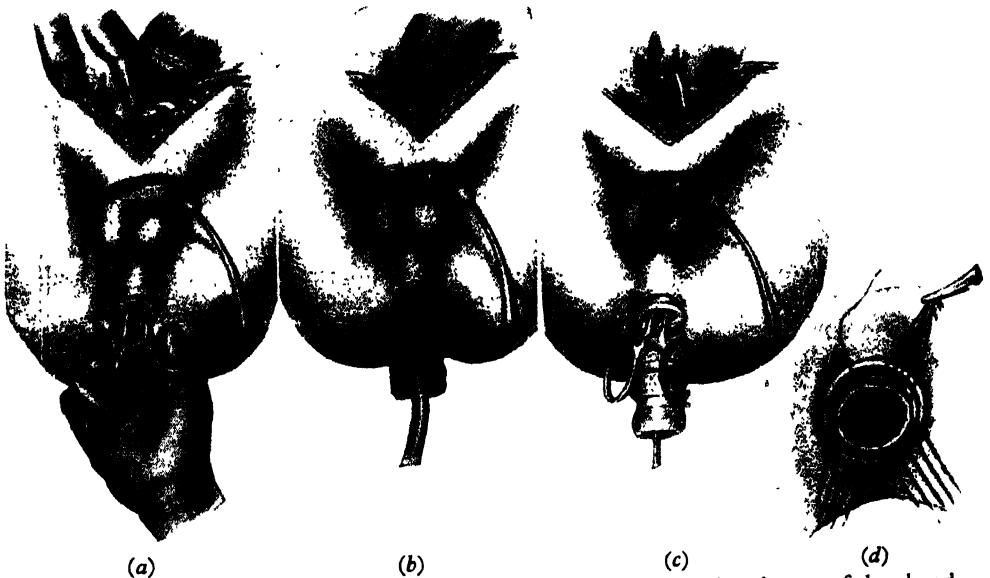


FIG. 1143.—(a) Long hæmostat introduced through the anus so that the top of the closed bowel can be grasped. (b) Mobilised segment is everted through the anus. (c) Long hæmostat introduced through the incision in the rectal wall to grasp the proximal end of the divided colon. (d) Anastomosis of the colon to the anal canal. (After O. Swenson.)

Oscar Swenson, *Contemporary*. Professor of Surgery, Northwestern University, Chicago, Illinois, U.S.A.
James Johnston Mason Brown, 1908–1964. Surgeon-in-Charge, Royal Hospital for Sick Children, Edinburgh.

lionic segment is excised. The union having been completed, it is reduced into the anal canal. Gloves having been changed, the abdominal incision is repaired. By two teams working simultaneously, one abdominally and one perineally, the operation can be performed expeditiously. Cases of Hirschsprung's disease involving the entire colon have been treated successfully by total colectomy and ileoproctostomy.

Modified Duhamel Operation.—This operation is devised to preserve recta sensation. Preliminary colostomy and bowel preparation is carried out as above. The aganglionic segment is removed down to the level of the peritoneal reflection over the rectum. At this point the rectum is divided, turned in, and closed over. The sacral hollow is then opened up and the normal colon brought down to the posterior aspect of the rectal stump. The anus is now widely stretched and a transverse incision is made in the posterior wall just above the sphincter. Through this opening the colon lying behind the rectum is seized and brought down to present at the anus. The colon is then fixed to the rectum by a few catgut stitches to make a somewhat loose colo-rectal anastomosis. A special crushing clamp is now introduced to crush the spur between the rectum and the colon. It is left in position till the spur separates, when the clamp becomes loose and can be removed. The colostomy is closed a few weeks later.

ACQUIRED OR SECONDARY MEGACOLON

Dilatation and hypertrophy of an otherwise normal large bowel extends to the anal canal. The obstruction is due to faecal impaction. Characteristically, there is a fissured anus, a spastic sphincter and much perianal soiling. As a rule, faulty bowel care and training are the sources of the trouble, and usually they can be traced to infancy; the onset, however, is never from birth.

Rectal Examination.—This is usually painful and the finger encounters a scybalous mass just inside the anus, which is contrary to the findings in Hirschsprung's disease.

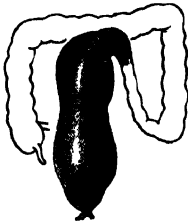


FIG. 1144.—Radiological appearances of acquired megacolon. (After B. C. H. Ward.)

Radiography.—In all cases of acquired megacolon the dilatation as shown by a barium enema ends at the anal canal (fig. 1144).

Biopsy of the Ano-rectal Wall.—When acquired megacolon cannot be differentiated from Hirschsprung's disease by clinical and radiological means, biopsy is required. The presence of ganglion cells in the myenteric plexus is the signal for conservative treatment.

Conservative Treatment.—Should an anal stricture or a fissure-in-ano be present, these lesions must be treated appropriately. The essential feature is bowel training so that a regular habit is established. It is wise to start with regular enemata and Senokot until the bowel is clean. The family environment must be thoroughly investigated. Upset of bowel habit in a child is a not uncommon result of domestic upheaval or emotional deprivation.

Redundant Colon.—Some constipated patients (usually female) are found on investigation by barium enema X-ray to have an elongated sigmoid colon. Volvulus formation is a theoretical risk, but the wise surgeon avoids removing the redundant bowel for these patients are neurotic, and will plague him after the operation with the same or other symptoms.

TRAUMATIC RUPTURE OF THE INTESTINE

The intestine can be ruptured with or without an external wound. The most frequent cause of the latter is a blow on the abdomen which crushes the bowel against the promontory of the sacrum. Rupture is particularly

able to occur in the presence of an irreducible inguinal hernia (John). Rupture typically occurs where a fixed part of the alimentary tract joins a mobile one, such as the duodeno-jejunal flexure, in which case the damage may be retroperitoneal and easily overlooked; this type of lesion is usually due to runover accidents.

In small perforations the mucosa prolapses and tends partially to seal the rent (fig. 1145); consequently the early signs are misleading. In general the signs simulate closely those of a perforated peptic ulcer.

Laceration of the mesentery (p. 884) is a frequent operative finding in the type of injury under consideration. The intestine itself is not necessarily damaged, but, owing to devascularisation, its viability may be so imperilled as to render resection of the affected segment (fig. 1146) imperative.



FIG. 1145.—Traumatic rupture of the ileum. Note the prolapse of the mucous membrane.

Traumatic rupture of the large intestine is much less frequent. Compressed-air rupture of the colon is sometimes the result of a damnable form of practical joke, whereby a hose, carrying air under considerable pressure, is turned on near the victim's anus.



FIG. 1146.—Laceration of the mesentery resulting in gangrene of the associated portion of gut.

Blast injuries of the abdomen sustained during air-raids resulted in a number of cases of traumatic rupture of the intestine. The pelvic colon was found to be injured more frequently than other segments of the intestine. Rupture of the upper reaches of the rectum is not unknown during sigmoidoscopy. In ulcerative lesions the air insufflation has been sufficient to perforate the intestinal wall.

Treatment.—In all cases of suspected rupture of the intestine immediate laparotomy must be performed. In many instances simple closure of the perforation is all that is required. In others, e.g. where the mesentery is lacerated, resection may be necessary. In the case of the large intestine, exteriorisation is the procedure of choice; if this is not feasible, the perforation is closed and proximal colostomy is performed.

THE BLIND LOOP SYNDROME

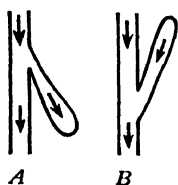
It has been shown in dogs that if a blind loop of the small intestine is made (fig. 1147*A*) defects of absorption will appear. If this occurs in the upper intestine the defect is chiefly of fat absorption; if in the lower intestine there is vitamin B₁₂ deficiency. This has been found to occur in humans and is referred to as the blind loop syndrome, or the 'stenosis-anastomosis loop' syndrome (Witts).

Essentially, the stasis produces an abnormal bacterial flora, which fouls the main intestinal pathway. Sometimes the only manifestation is anæmia, due to Vitamin B₁₂ deficiency, but if steatorrhœa appears, other serious malabsorption features follow. In general, high loops produce steatorrhœa, etc., whereas low loops tend to produce anæmia.

Temporary improvement will follow the use of antibiotics to destroy the

Howell Thomas John, *Contemporary*. Surgeon, Royal United Hospital, Bath.

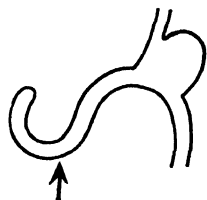
Leslie John Witts, *Contemporary*. Emeritus Nuffield, Professor of Clinical Medicine, Oxford University.



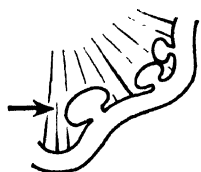
Experimental blind loops.

A. Self-filling: deficiency occurs.

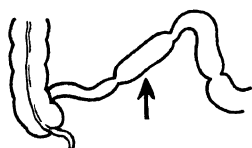
B. Self-emptying: no deficiency occurs



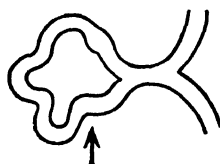
C. Long afferent loop stasis in Polya gastrectomy.



D. Jejunal diverticula.



E. Intestinal stricture causing



F. 'Stenosis-anastomosis loop' syndrome.

FIG. 1147.—Common types of blind loops.

Duodenal Diverticulum.—There are two types: (1) *Acquired*—of the duodenal cap—secondary to the stenosis of an ulcer. (2) *Congenital*—on



FIG. 1148.—Diverticulum at the mesenteric border of small intestine.



FIG. 1149.—Prestenotic diverticula of duodenal cap.



FIG. 1150.—Diverticula of second and third parts of duodenum.

bacteria causing the trouble, but the main treatment is surgical extirpation of the cause of the stasis.

ALIMENTARY DIVERTICULA

Diverticula occur in many parts of the alimentary canal from the stomach to the recto-sigmoid junction.

Diverticula are divided into two varieties:

(a) *Congenital*.—All three coats of the bowel are present in the wall of the diverticulum, e.g. Meckel's.

(b) *Acquired*.—The wall of the diverticulum lacks a muscular coat. In spite of the absence of demonstrably increased intraluminal pressure, most alimentary diverticula are thought to be acquired (Edwards). Literally, a diverticulum means a wayside house of ill-fame, and these wayside houses certainly live up to their evil reputation.

DIVERTICULA OF THE SMALL INTESTINE

The belief that a diverticulum of the small intestine originates as a mucosal herniation through a point of entrance of blood-vessels is based on the fact that most of these diverticula arise from the mesenteric side of the bowel (fig. 1148).

the inner wall of the second and third parts. Usually they are found incidentally on a barium meal and do not give rise to symptoms (p. 736).

Jejunal diverticula vary in size, are sometimes single (fig. 1151), but more often several are present (fig. 1152). Clinically they may (a) be symptomless (b) give rise to abdominal pain, flatulence and borborygmi or (c) produce a malabsorption syndrome. This latter consists of anæmia, steatorrhœa, hypoproteinæmia and avitaminosis. They are, in fact, examples of the blind loop syndrome (p. 897). Under these circumstances, resection and end-to-end anastomosis give excellent results.

Meckel's diverticulum is present in 2 per cent. of the human race; it is situated upon the anti-mesenteric border of the small intestine, 2 feet (60 cm.) from the ileo-cæcal valve, and it is usually 2 inches (5 cm.) long. Useful as is this mnemonic, many variations occur.

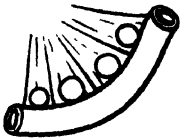


FIG. 1152.—Jejunal diverticulosis.



FIG. 1151.—Diverticulum of the jejunum three hours after ingestion of barium. The patient was a woman of fifty-six, with a fourteen years' history. She was cured by excision of the diverticulum.

In nearly 90 per cent. of cases the diverticulum arises from the anti-mesenteric border of the ileum, and, being congenital, it possesses all three coats of the intestinal wall. In 20 per cent. of cases the mucosa contains heterotopic epithelium, viz. gastric, colonic, or sometimes pancreatic tissue. When present, heterotopic tissue lines the greater part of the proximal end of the pouch, and not infrequently extends for a short distance into the nearby ileum. Although Meckel's diverticulum occurs with equal frequency in both sexes, symptomatic cases, due almost entirely to the epithelium contained in the diverticulum, are predominant in males. In order of frequency, these symptoms are as follows:



FIG. 1153.—Meckel's diverticulum.

1. **Severe hæmorrhage per rectum**, due to peptic ulceration, occurs most frequently between the ages of ten and fifteen years, but may be postponed until adult life. The blood passed is neither the bright red of a colonic lesion nor the typical, almost black, stool of melæna from a bleeding gastro-duodenal ulcer; it is intermediate (maroon) in colour. Although the patient frequently vomits, the vomitus contains no blood. Seldom is the hæmorrhage preceded by pain; sometimes bleeding precedes perforation. When operation is required for serious progressive hæmorrhage per rectum and no lesion in the stomach or duodenum is found, the next step should be the examination of the terminal 5 feet (150 cm.) of ileum.

2. **Intussusception**.—In the majority of cases the apex of the intussusception is swollen, inflamed, heterotopic epithelium at the mouth of the diverticulum—not inversion of the diverticulum, as is commonly stated. Intussusception due to Meckel's diverticulum is discussed on p. 939.

3. **Meckelian diverticulitis** with or without perforation is usually due to lodgement of coarse food residue or a sharp foreign body. The symptoms of Meckelian diverticulitis without perforation are those of acute appendicitis, and unless the appendix has been removed the diagnosis is impossible before operation. When a diverticulum perforates, so rapid is the onset of peritonitis that the symptoms simulate those of a perforated duodenal ulcer. Whether or not the diverticulum has per-

forated, urgent operation is required. In non-perforated cases an inflamed diverticulum should be sought as soon as it has been ascertained that the vermiform appendix (and, in the case of a female, the Fallopian tubes) is not culpable.

4. **Chronic Peptic Ulceration.**—The pain is similar to that of a duodenal ulcer, but unless the patient is a child, and some blood has been passed per rectum, the condition remains undiagnosed for long periods or until a serious complication arises.

5. **Intestinal Obstruction.**—The presence of a band between the apex of the diverticulum and the umbilicus may cause obstruction either by the band itself or to a volvulus around it.

Radiography.—In cases of Meckel's diverticulum giving rise to symptoms, failure to visualise the diverticulum by radiography after a barium meal, which is very common, is of no significance, because so often the entrance of the diverticulum is blocked by œdema.

'**Silent**' **Meckel's Diverticulum.**—A Meckel's diverticulum usually remains symptomless throughout life, and be found only at necropsy. When a silent Meckel's diverticulum is encountered in the course of an abdominal operation, and can be excised without appreciable additional risk, this should be done in order to exclude the possibility of subsequent complications.

Exceptionally a Meckel's diverticulum is found in an inguinal or femoral hernial sac, when it is known as a Littre's hernia.

Meckelian Diverticulectomy.—A Meckel's diverticulum should not be amputated at its base and invaginated in the same way as a vermiform appendix, because of the risk of a stricture. Moreover, it does not remove heterotopic epithelium completely, if such be present. The steps of diverticulectomy are displayed in fig. 1154. Should there be considerable induration of the base of the diverticulum,

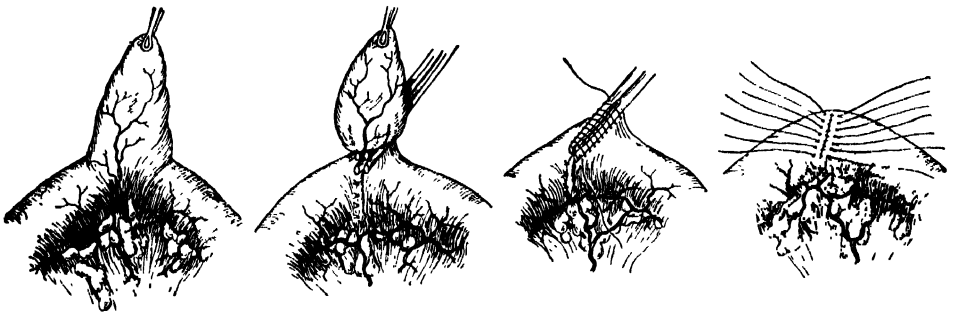


FIG. 1154.—Steps in the performance of Meckelian diverticulectomy.

and particularly when such induration extends into the neighbouring ileum, it is advisable to resect a short segment of the ileum containing the diverticulum, and to restore the continuity of the bowel by anastomosis.

DIVERTICULA OF THE LARGE INTESTINE

Diverticulum of the cæcum is congenital, for it has a complete muscular coat. It is solitary, and situated on the medial aspect of the intestine just above the ileocæcal valve, viz. →

Its neck may be narrow, and the diverticulum is then subject to attacks of acute inflammation indistinguishable from acute appendicitis. When chronically inflamed it produces gross thickening of the ileo-cæcal region and dense adhesions, the cause of which may not be evident until the cæcum has been resected.



DIVERTICULOSIS AND DIVERTICULITIS OF THE COLON

Diverticulosis is seen in 8 per cent. routine barium enema studies after the age of forty. Twenty-five per cent. of these proceed to an inflammatory state—diverticulitis, especially if they are obese, and are habitually constipated. Diverticulosis may be seen throughout the colon, but diverticulitis

is virtually confined to the sigmoid loop. The primary state of diverticulosis is associated with muscular hypertrophy and incoordination of muscle, producing segments of colon in which the pressure may increase, resulting in diverticula. The herniations of mucosa pass through the circular muscle and between the *tæniæ* (fig. 1155) at points where blood-vessels penetrate the colonic wall. This basic inco-ordination of colonic muscle and spasm of the pelvi-rectal junction is aggravated by stress and clinically appears to be decreased by phenobarbitone and Probanthine.

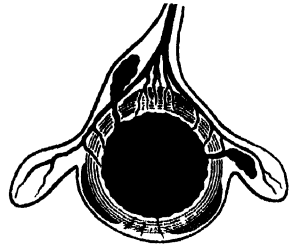


FIG. 1155.—The usual sites of diverticulation of the colon. (After Hamilton Drummond.)

Diverticulitis is a secondary state due to inflammation in the diverticulum following occlusion of its ostium. The reaction may be acute with abscess formation and perforation into the peritoneal cavity. If more chronic, there is progressive fibrosis, resulting in shortening and distortion of the colon, and stenosis. Adherence to the bladder, or rarely to the small bowel, may occur, with the possibility of fistula formation. Diverticulitis is essentially progressive—the longer the duration, the worse are the symptoms, and the greater is the risk of complications.



FIG. 1156.—Pelvic colon with multiple diverticula.

Clinical Features.—Colonic diverticulitis (fig. 1156) is a disease of exacerbations and remissions. Individual attacks may be spread over several days, but periods of freedom can last for months or even years. Virtually all patients are over forty years of age, and the sexes are equally afflicted. They complain of flatulence and dyspepsia, and have recurring pain and tenderness in the left iliac fossa with fever. They are constipated, but may have small, frequent motions. The pain is worse on movement,

but there is relief on passing flatus. In addition to local tenderness there may be palpable thickening of the colon or a mass. Some 5 per cent. of patients have associated gall stones and hiatus hernia. Urinary symptoms may occur and must be carefully watched for, as they may be the first premonition of a vesico-colic fistula. Hæmorrhage, which is often sudden and profuse (in contradistinction to the bleeding of carcinoma of the colon), perforation, obstruction, or fistula formation (vesico-colic, vagino-colic, entero-colic or colo-colic) may occur—the incidence of each being approximately 5 per cent.

Radiology.—Barium enema may show many diverticula, but a particular segment in the sigmoid especially may be distorted and shortened—and seemingly devoid of diverticula. This area bereft of diverticula and with a

'saw tooth' edge is especially suggestive (fig. 1157). Probanthine may be used to relax the colon and this may help to exclude carcinoma.



FIG. 1157.—An area of diverticulitis affecting the sigmoid colon. Alterations in the calibre of the lumen of the bowel associated with the presence of diverticula is characteristic of the condition. (Professor H. Middlemiss, Bristol)

Sigmoidoscopy.—The mucosa is usually inflamed, with an excess of mucus. The bowel is rigid and narrowed in the involved segment so that the sigmoidoscope is held up.

Management

During an Exacerbation.—Bed rest and warmth to the abdomen is essential, with luminal to ensure relaxation. Only fluids by mouth are allowed. The abdomen should be examined frequently to note if satisfactory resolution is occurring. The insoluble sulphonamides are not satisfactory. The inflammatory reaction is *intramural* rather than in the lumen of the colon, and so broad spectrum antibiotics are prescribed. Morphine and codeine are contra-indicated. As the attack settles, a full diet, including fruit, oatmeal, and salad is slowly introduced. A low residue diet, formerly in vogue, may lead to narrowing of the bowel. Regular bowel actions may be assisted

by Isogel and by liquid paraffin or sub-laxative doses of senokot. Occasional bowel washouts may be necessary. Anything which causes increase of colonic gas must be avoided, whether it is worry or dietary factors such as onions, swedes, turnips, cabbage, peas, or beans.

Some 10 per cent. of cases require surgical intervention.

Complications and Indications for Surgery

In general it may be said that the *complications* of diverticulitis are the indications for surgery:

- (1) Recurrent pericolic inflammation.
- (2) Perforation leading to general peritonitis or local abscess formation.
- (3) External or internal fistulæ especially vesico-colic.
- (4) Progressive stenosis —————> intestinal obstruction.
- (5) Hæmorrhage: diverticulitis is a common cause of *profuse* colonic hæmorrhage. Morphine and blood transfusion are usually required. These hæmorrhages are often recurrent and surgery between attacks must be seriously considered even in the elderly.
- (6) Carcinoma may coexist.

Procedures

(a) *The best operation*, done as an interval procedure after preparation of the gut (p. 921), is a *One Stage Resection*. This involves removal of the affected segment—10 to 20 cm. long, and end-to-end anastomosis.

(b) *If there is obstruction, or the bowel is loaded with fæces*, preliminary transverse colostomy must be done as a first stage; the second stage of resection is performed after three weeks. The colostomy is closed after a further two weeks.

(c) *In acute perforation*, there is general peritonitis, usually fæcal, with pneumo-peritoneum. Urgent laparotomy is indicated with peritoneal toilet. One of several procedures may be carried out, depending on the state of the patient and the mobility of the colon:

- (i) Proximal colostomy, suture of the perforation.
- (ii) Primary resection with proximal colostomy is becoming more and more popular.
- (iii) Exteriorisation of the loop.

Drainage is necessary after all these procedures.

(d) *If there is a localised pericolic abscess*, sump drainage is required at first, resection being carried out at a later date.

(e) *Urinary symptoms* need urgent treatment, as they indicate that vesico-colic fistula is imminent, with associated severe cystitis and pneumaturia. A one-stage resection with closure of the vesical defect is the ideal procedure, otherwise a staged operation (p. 1141) is performed.

DIFFERENTIATION OF DIVERTICULITIS FROM CARCINOMA OF THE COLON

These conditions co-exist in 12 per cent of cases

	<i>Diverticulitis</i>	<i>Carcinoma</i>
History	Long	Short
Pain	More common	
Mass	25 per cent. have tenderness	25 per cent. painless.
Bleeding	5-10 per cent. often profuse, periodic.	65 per cent.—usually small amounts persistently.
X-ray	Diffuse change	Localised: no relaxation with Probanthine.
Sigmoidoscopy	Inflammatory change over an area	No inflammation until ulcer reached

Exploration may be necessary, but even then, differentiation may be difficult until histology is available. Weight loss, falling hæmoglobin, and a persistently positive occult blood test are sinister features.

ULCERATIVE COLITIS

Ætiology.—The cause is unknown. In spite of intensive bacteriological studies, no organisms or group of organisms can be incriminated. Probably the disease is linked with emotional stress. It may be related to the auto-immune diseases but this is by no means certain. Some cases are allergic to milk protein. In cases of extensive ulceration secondary infection plays a large part.

Pathology.—In 95 per cent. of cases the disease starts in the rectum and spreads proximally. When the ileocæcal valve is incompetent, retrograde ileitis involving the last foot (30 cm.) of the ileum is liable to occur.

The disease is characterised by the appearance of multiple minute ulcers—sometimes the ulcers are discrete, in others there is a sea of ulceration. Microscopical evidence nearly always proves that the ulceration is more severe and extensive than the gross appearance indicates (Cuthbert Dukes). As time goes on the small ulcers are apt to coalesce to form larger ones, mainly due to the crypts of Lieberkühn becoming distended with pus and bursting into the bowel. When the ulceration extends into the submucosa it

Cuthbert Dukes, Contemporary Consulting Pathologist, St. Mark's Hospital, London. Johann Lieberkühn, 1711-1756. Anatomist, Berlin. He demonstrated his anatomical preparations in London, and was awarded the F.R.S.

causes reflex muscle spasm, and the appearance of a stricture. In long-standing cases there is always considerable intramural fibrosis, causing the affected part of the colon to become permanently contracted. In addition, attempts at healing may produce epithelial thickening between the ulcers—the so-called pseudopolyposis.

Clinical Features.—Women are attacked somewhat more often than men. The onset of the disease is in the 3rd, 4th, and 2nd decade, in that order ; exceptionally it is encountered in childhood. The first symptom is watery diarrhoea occurring day and night, in a person of previously normal bowel habit. A rectal discharge of mucus, sometimes blood-stained and sometimes purulent is very common. Pain as an early symptom is unusual. In the majority of cases, the disease is chronic, characterised by relapses and remissions. In general, a bad prognosis is indicated by (1) a severe initial attack; (2) disease involving the whole colon; (3) increasing age, especially after sixty years. If the disease remains confined to the left colon, the outlook is better.

Two types are encountered :

Fulminating type (5 per cent.) is ushered in with a temperature of 102° to 103° F. (38·9° to 39·4° C.) and incessant diarrhoea containing blood, mucus, and pus; the patient looks and feels very ill. There may be abdominal distension due to toxic paresis of the colon. In such circumstances the condition must be differentiated from dysentery and typhoid. These infections are eliminated by bacteriological examinations of the stools, blood culture, and a Widal reaction.



FIG. 1158.—Emaciation and dehydration in ulcerative colitis. (Dr. Peter Hansell, London.)

Chronic Type (95 per cent.).—As a rule the initial attack is of moderate severity, but exacerbations occur at intervals of weeks, months or, in mild cases, years. As the disease progresses the patient becomes wasted from diarrhoea, and severely anæmic from loss of blood (fig. 1158). Often during the attacks there are ten to twenty stools a day, accompanied by tenesmus. The frequency of the motions and the degree of invalidism go hand in hand, and are proportional to the extent of the involvement of the colon. Lesions of limited length are commonly found in milder cases. Sometimes the disease is confined entirely to the right hemi-colon. When the

whole colon is involved the patient may be so weak as to be partially or wholly bed-ridden.

Radiology after a barium enema shows one of the following :

- (1) The earliest sign is loss of haustration, especially in the distal colon. Established disease will show:
- (2) A narrow contracted (pipe stem) colon (fig. 1159).
- (3) Undermining of the mucosa.
- (4) Pseudopolyposis (fig. 1160) in 10 per cent. of cases.



FIG. 1159.—Advanced ulcerative colitis. Showing tubular contraction and shortening of the colon. (W. B. Gabriel, F.R.C.S., London.)



FIG. 1160.—Ulcerative colitis: showing pseudopolyposis ('stippled' area in descending colon). (Dr. Clifford Hawkins, Birmingham.)

Sigmoidoscopy is indispensable in the diagnosis of early cases and in mild cases when the pathological effects of the disease are insufficient to alter the barium shadow. The initial findings are those of proctitis (p. 1001)—the mucosa is hyperæmic, bleeds on touch and there is considerable exudate. Later, tiny ulcers may be seen and appear to coalesce (fig. 1161). This is very different from the picture seen in amœbic dysentery, where there are large deep ulcerations with the intervening mucosa comparatively healthy. As the disease progresses, the ulceration may become so severe that practically no normal mucous membrane remains.

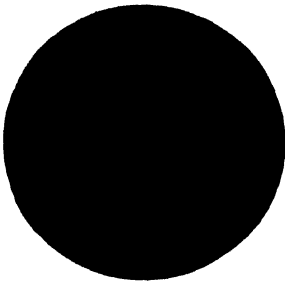


FIG. 1161.—Ulcerative colitis. The granular inflamed appearance of the mucosa is characteristic. (After the late J. P. Lockhart-Mummery.)

A barium enema and sigmoidoscopy should be avoided during the fulminating phase. Rectal biopsy is valuable not only in establishing the diagnosis, but also as an index of the effect of therapy.

Complications.—These are local and general.

LOCAL

Pseudopolyposis 15 per cent. (p. 904).

Carcinoma.—Although this is an important complication, it must be seen in its true perspective. The overall risk is about 3·5 per cent.; this risk is much less in early cases but increases with the duration of the disease. Thus, after twenty years of the disease, the risk may be as much as 12 per cent. Carcinoma is more liable to occur where the whole colon is involved and where the disease started in early life. Carcinomatous change may occur at many sites at once and these, often, are atypical. The colon is involved rather than the rectum and maximal incidence is during the 4th decade (Truelove). The golden rule is that after the disease has been present for more than ten years, regular radiological and sigmoidoscope checks must be done, even though the disease seems to be quiescent.

Fibrous Stricture occurs in 6 per cent. of cases. The common sites are the recto-sigmoid junction and the anal canal. Although they usually respond to simple dilatation where the disease is confined to the distal colon, surgery may be advisable if the whole colon is involved.

Perforation (3 per cent.) occurs in the fulminating type of the disease. It is often preceded by toxic dilatation of the colon. Excessive steroid therapy may be a factor.

Massive Hæmorrhage (3 per cent.) usually arises from the rectum rather than the colon.

Recto-vaginal Fistulæ (3 per cent.), **Fistula in Ano** (4 per cent.), **Ischio-rectal Abscess** (4 per cent.), **Ileus and Toxic Megacolon** (1.5 per cent.) and **Hæmorrhoids** (20 per cent.) also occur (Truelove).

GENERAL

Liver Changes (7 per cent.) sometimes resulting in cirrhosis, may occur as the result of heavy protein loss.

Skin Lesions (18.5 per cent.) including **Pyodermia Gangrenosum** (0.5 per cent.) and **Erythema Nodosum** (2 per cent.).

Arthritis (5 per cent.) usually of small joints, may occur at any stage of the disease.

Iritis (7 per cent.), **Ankylosing Spondylitis** (2 per cent.), **Stomatitis** (10 per cent.), **Renal Disease** (5 per cent.) and **Anæmia** (20 per cent.) are also important complications.

Treatment.—There is no specific therapy—there are several agents which may induce a remission but it is difficult to predict the outcome in individual cases. The main *general principles* are:

- (1) Maintenance of fluid and electrolyte balance.
- (2) Anæmia must be speedily corrected.
- (3) Adequate nutrition—at least 3,000 calories per day—with high protein, carbohydrate and vitamin content and low fat.
- (4) Phenobarbitone, belladonna and codeine phosphate are useful drugs.
- (5) Antibiotics for febrile toxic cases (in short courses to prevent the onset of moniliasis). Salazopyrin (4 to 6 g. daily) for seven days is specially valuable.

All are agreed on the above measures. All surgeons, however, are not agreed about the use of:

(6) Cortisone therapy. The M.R.C. trial (1950–58) showed that those receiving cortisone fared better than those receiving an inert substance and that first attacks did better than relapses. Its effect is far from permanent and is often negligible in the established disease. Topical steroids are of real value in the distal type of disease. In a moderately severe case it is probably best to try:

- (a) Salazopyrin for seven days.
- (b) Topical steroids—retention enemas of prednisolone phosphate 20 mg. in 100 ml. daily for seven days. If no response then:
- (c) Oral steroids—prednisolone 20 mg. daily for two to three weeks.

Dangers of Prolonged Cortisone Therapy.—1. Not only is there an increased risk of massive hæmorrhage and of free perforation into the peritoneal cavity, but in patients who have not responded to a course of cortisone or similar steroid over a period of four to six weeks, the colonic wall becomes excessively friable and in some areas disintegrates, its place being taken by the parietes or adjacent viscera, usually the small bowel. In such cases the surgeon may then be unable either to remove the colon or even to perform ileostomy (Brooke).

2. Cortisone therapy renders the patient more susceptible to pyogenic infection; therefore it is wise to administer penicillin in addition.

The chief indication for the exhibition of steroid therapy is in fulminating cases, as an alternative to ileostomy performed during the ultra-acute phase (Crile Jr.).

Bryan Nicholas Brooke, Contemporary. Professor of Surgery, St. George's Hospital, London.
George Crile Jr., Contemporary. Head of the Department of General Surgery, The Cleveland Clinic, Cleveland, Ohio, U.S.A.

Indications for Surgery

- (1) To save life—fulminating cases, severe hæmorrhage, perforation.
- (2) Local complications (p. 905).
- (3) General complications (p. 906).
- (4) Risk of neoplastic change—especially in pseudo-polyposis and colitis of long standing.
- (5) Onset in children or adolescents.
- (6) Chronic invalidism.

Between 15 and 20 per cent. of sufferers are recommended for surgical treatment by their physicians.

When surgical treatment is advised, about six days' intensive pre-operative preparation by the methods already enumerated is carried out.

Operations

(i) The ideal procedure is a one-stage total procto-colectomy with ileostomy. If the disease is acute, it is wise to leave the rectal stump and either close it or bring it out as a midline colostomy or mucous fistula. The rectum can be removed about eight weeks later.

(ii) Some surgeons advocate total colectomy with *ileo-rectal anastomosis*—this can only be done satisfactorily in about 5 per cent. of cases where the rectum is not involved.

(iii) *Ileostomy alone* may sometimes be indicated for a gravely ill patient with fulminating disease and localised abscess around a perforation.

Ileostomy.—The ileostomy is, of course, permanent and the success of the operation lies in scrupulous attention to the details of technique in siting and

fashioning the ideal 'spout'.

It is best to arrange the position of the stoma beforehand with the patient (fig. 1162).

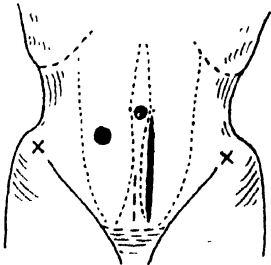


FIG. 1162.—Site of the ileostomy. The 'trephine' wound should be at least 2 inches (5 cm.) from all scars and bony points.

A disc of skin not more than the size of a florin (3 cm. in diameter) is removed at least 2 inches (5 cm.) lateral to the umbilicus (fig. 1162). The abdomen is opened by a left paramedian incision and the ileum is divided near its termination, together with its mesentery. It may be necessary to divide the ileum more proximally if the terminal ileum is diseased also.

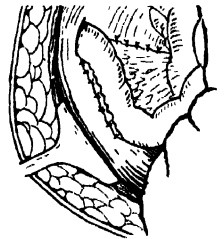


FIG. 1163.—Suture of the mesentery to the parietal peritoneum, to prevent prolapse. (After J. C. Goligher, F.R.C.S., Leeds.)

Important steps in the performance of ileostomy are (a) to close the peritoneal space on the external side of the ileostomy (otherwise a tunnel exists which invites intestinal obstruction), and (b) to anchor the anterior edge of the mesentery to the parietal peritoneum (fig. 1163). The latter prevents prolapse of the mucous membrane. The ileostomy opening must be prepared meticulously. A disc of tissue equal to the size of the skin disc should be removed from the anterior and posterior rectus sheath so that no stricture will occur in the parietics. The peritoneum is opened by a cruciform incision. About 3 inches (7.6 cm.) of the divided ileum (closed temporarily with a noncrushing clamp) is brought through the opening and its periphery is stitched to the skin edges by interrupted catgut in such a way that it is everted to form a 'spout' projecting $1\frac{1}{2}$ inches (3.8 cm.) from the skin

surface. The greatest care must be taken in the construction of this projection (fig. 1164). A proctocolectomy is carried out and the abdomen is closed. A dis-

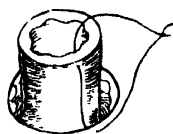


FIG. 1164.—Suturing the free extremity of the proximal ileum to the skin edges.

posable plastic ileostomy bag (fig. 1165) is cut so that it exactly fits the skin at the muco-cutaneous junction and is fixed in position by latex rubber adhesive.

Care of Ileostomy.—During the first few post-operative days the fluid electrolyte balance must be adjusted with great care. For permanent use it is wise to fit a Chiron-type bag which has a firm rubber flange to be attached around the ileostomy spout. It is supported both by a waist strap and also made adherent to the skin by special double-sided adhesive plaster. The bag is attached to and detached from the flange which remains adherent to the skin for as long as is convenient. Great care must be taken that the lower rim of the bag does not press on and penetrate the lower margin of the ileostomy 'spout'. A paste of aluminium 10 parts and zinc oxide 90 parts is most helpful if ex-cori-ation occurs. The stools thicken in a few weeks and are semi-solid in a few months. A thin discharge from the stoma may indicate bolus obstruction and stenosis; digital dilatation will help to relieve this.



FIG. 1165.—Disposable ileostomy bag. (Bryan N. Brooke, F.R.C.S., London.)

Procto-colectomy requires no special description. The cæcum and colon are detached from their mesenteries and when the rectum is reached the procedure is exactly as for a combined abdomino-perineal resection (p. 1018). If the patient is too ill to stand the perineal dissection, the rectum is freed as far as the pelvic diaphragm and is transected at this point. The large intestine is removed, the upper end of the rectum closed and the pelvic peritoneum united over it. Alternatively, the lower bowel can be brought out as a colostomy through the paramedian incision.

Provided the patient is even moderately fit, the modern trend is to undertake procto-colectomy in addition to ileostomy at the primary operation. Colectomy is not a difficult operation when it is performed on an emaciated individual whose peritoneal cavity is free from adhesions. After a successful ileostomy both these desiderata are absent; firstly, the patient has gained weight and, secondly, adhesions are bound to be present, at least in the right iliac fossa.

Whether the patient should be left with a permanent ileostomy, or whether ileo-rectal anastomosis should be carried out either at the time of the colectomy or at a later period, remains slightly controversial. The majority of surgeons do not favour the anastomosis.

On the other hand, some surgeons point out that although ileostomy, if properly performed, is no longer the severe handicap it used to be, it is, nevertheless, a nuisance and best avoided if possible. If an ileo-rectal anastomosis is performed, it is essential that no sigmoid colon be left (Aylett). After the operation a physiological bowel frequency of three to five actions a day is to be expected. It should not be performed if peri-anal complications are present. Should ileo-rectal anastomosis fail and the disease recur in the rectal stump, an ileostomy can always be performed.

THE SURGICAL ASPECTS OF INTESTINAL AMŒBIASIS

Amœbiasis denotes an infestation with *Entamœba histolytica*. Contrary to general belief, *E. histolytica* is not strictly a tropical parasite; it has a world-wide distribu-

tion, and is found in Great Britain. It is surprisingly common in overcrowded institutions. The reason why in some cases it becomes pathogenic is unknown.

Life History of the Parasite.—The active form of the parasite or trophozoite lives in the intestinal mucous membrane, where it ingests red blood corpuscles and other cells, and multiplies by mitosis. Should the parasite become pathogenic, it makes its way into the follicles of Lieberkühn, and by dissolving interglandular tissue by cytolytins, submucous loculi are produced. Some of these burst through the mucous membrane to become amœbic ulcers. While the trophozoites continue their activities in the base of the ulcer, others cease to feed, migrate towards the surface, and become transformed into cysts (fig. 1166), which pass into the outer world with the fæces. Amœbiasis is transmitted mainly in drinking water.

Pathology.—The ulcers, which have been described as ‘bottle-necked’ because of their considerably undermined edges, have a yellow necrotic floor, from which blood and pus exude. While on rare occasions the ulcers are scattered throughout the large intestine, in 75 per cent. they are confined to the lower sigmoid and the upper rectum.

Biopsy material is obtained by scraping the suspected area of mucosa with a long-handled Volkmann’s spoon; this removes a thin slice of the diseased mucosa, which must be examined immediately. It should be noted particularly that the finding of *Entamœba histolytica* is not conclusive evidence that the symptoms are due to amœbiasis. On numerous occasions a positive slide examination has caused the clinician to assume that the diagnosis has been established when there has been a proximal carcinoma of the colon and the *Entamœbæ histolyticae* have been non-pathogenic and incidental.

Clinical Features.—Dysentery is only one manifestation of the disease. In various guises amœbiasis obtrudes itself into the surgeon’s diagnostic arena:

Appendicitis or Amœbic Typhlitis¹?—In tropical countries where amœbiasis is endemic, this is a constantly recurring problem. To operate upon a patient with amœbic dysentery without the precautions subsequently described is to invite an exacerbation of amœbiasis that may prove fatal. Especially in cases where a palpable mass is present, the bowel is friable and satisfactory closure of the appendix stump becomes difficult or impossible. The death-rate from peritonitis and wound infection in the notorious Chicago epidemic of amœbiasis in 1933 was appalling, which emphasises that surgeons in temperate climates should be familiar with the condition. In the case of amœbic typhlitis there is rarely rigidity, and pain commences in the right iliac fossa. In amœbic typhlitis there are two characteristic and localised zones of tenderness on deep palpation—one over the cæcum and one over the sigmoid. The latter is sharply defined, and being comparable to McBurney’s point on the right side, it has been aptly named ‘the amœbic point’ by Sir Philip Manson-Bahr. Routine sigmoidoscopy is of great value. If doubt still exists, 1 grain (60 mg.) of emetine hydrochloride in 20 ml. (5 drachms) of normal saline solution given intravenously very slowly is likely to ameliorate the symptoms within two hours (Andreasen).

Perforation.—The most common sites are the cæcum and recto-sigmoid. Usually perforation occurs into a confined space where adhesions have previously formed, and a pericolic abscess results, which eventually needs draining. When there is sudden fæcal flooding of the general peritoneal cavity, drainage of the region of the perforation, gastro-intestinal aspiration, intravenous fluid replacement, antibiotic therapy, and a full course of emetine are sometimes successful.

Severe rectal hæmorrhage due to the separation of sloughs is liable to occur.

Granuloma.—Progressive amœbic invasion of the wall of the rectum or colon, with secondary inflammation, may produce a granulomatous mass indistinguishable from a carcinoma. The exhibition of emetine as a therapeutic test will prevent mistakes in diagnosis. Amœbiasis and carcinoma occasionally co-exist (Naunton Morgan).

Fibrous stricture may follow the healing of extensive amœbic ulcers.

Intestinal obstruction is a common complication of amœbiasis, and the obstruction is due to the adhesions associated with pericolicitis and a large granuloma.

Paracolic abscess, ischio-rectal abscess, and fistula occur from perforation by amœbæ of the intestinal wall followed by secondary infection.



FIG. 1166.—*Entamœba histolytica*, cystic stage.

¹ Typhlitis = inflammation of the cæcum.

Richard von Volkmann, 1830–1889. Professor of Surgery, Halle.
Charles McBurney, 1846–1913. Surgeon, Roosevelt Hospital, New York.
Sir Philip Manson-Bahr, 1881–1966. Physician to the Hospital for Tropical Diseases, London.
Anthony Turner Andreasen, Contemporary. Formerly Professor of Surgery, University of Calcutta.
Sir Clifford Naunton Morgan, Contemporary. Consulting Surgeon, St. Mark’s Hospital, London.

Treatment.—There is no remedy that can compare with emetine injections in the acute stage. In the chronic stages emetine bismuth iodide, given in gelatin capsules in amounts to a total of 30 grains (2 G.) over a period of ten days is usually extremely effective (enteric-coated capsules are useless—they pass through the intestine unchanged). The quinoxyl group of drugs, including iodoquin, are also effective in the chronic stage, and can be given during the interval between courses of emetine. Intestinal antibiotics improve results in the chronic stages, probably by coping with superadded infection.

THE SURGICAL COMPLICATIONS OF TYPHOID AND PARATYPHOID

Chloromycetin exerts a rapidly curative effect on typhoid and paratyphoid infections; consequently complications are uncommon. When complications of typhoid arise, chloromycetin should be given in addition to other necessary treatment, not forgetting that this antibiotic destroys the organisms responsible for the production of vitamin B complex which must be replaced.

1. *Paralytic ileus* is the commonest complication of typhoid (p. 949).

2. *Intestinal hæmorrhage* may be the leading symptom. The condition must be distinguished from purpura with intestinal symptoms, and intussusception. A Widal reaction should be employed and, if negative, repeated in suspected cases. Urgent blood transfusion will be required.

3. *Perforation.*—Perforation of a typhoid ulcer usually occurs during the third week; occasionally it is the first intimation of the disease (ambulatory typhoid). The ulcer is parallel to the long axis of the gut (fig. 1167), and is situated in the lower



FIG. 1167A.—A typhoid ulcer is longitudinal (Peyer's patch necrosis).



FIG. 1167B.—A tuberculous ulcer is transverse (because it follows the lymphatics).

ileum. In paratyphoid B, perforation of the large intestine sometimes occurs. The former treatment was to perform laparotomy under local anæsthesia, and to close the perforation. The results were so poor that the conservative treatment of peritonitis plus chloromycetin has been tried, and yields better results.

4. *Cholecystitis.*—Acute typhoid cholecystitis is not uncommon (p. 832). Gallstones occasionally contain typhoid bacilli. Chronic typhoid cholecystitis can result in the patient becoming a typhoid carrier.

5. *Phlebitis.*—Venous thrombosis, particularly of the left common iliac vein, is an occasional complication of typhoid fever.

6. *Genito-urinary Complications.*—Typhoid cystitis, pyelitis, bacilluria, and epididymo-orchitis all occur.

7. *Joints.*—All degrees of arthritis, from a mild effusion to suppuration, occur as a complication of this disease.

8. *Bone.*—Typhoid osteomyelitis and typhoid spine are discussed on p. 232.

9. *Larynx.*—Typhoid perichondritis is met with occasionally, and typhoid laryngitis has been known to obstruct the airway.

REGIONAL ILEITIS (*syn.* CROHN'S DISEASE)

Ætiology.—No causative organism has been found in the lesion or in the stools. Dogs receiving finely powdered silicates admixed with their food for long periods develop an identical condition, and it has been suggested that the ingestion of fine, insoluble particulate matter such as toothpaste is a possible cause of regional ileitis. A disease similar to regional ileitis occurs in swine.

Pathology.—Regional ileitis is essentially a cicatrising inflammation with ulceration of the mucosa. It usually commences at or near the ileo-cæcal valve, and extends upwards along the ileum for about 12 inches (30 cm.), but as little as 2 inches (5 cm.), and, more often, as much as 4 feet (1·2 metres) may be implicated. In acute cases the affected intestine is seen to be swollen, bright pink in colour, and with a fibrinous exudate on its peritoneal surface; in chronic cases hyperæmia is less in evidence. On palpation the intestinal wall feels like a hose-pipe. The mesentery of the involved intestine is exceedingly thickened, œdematous, and contains enlarged and fleshy lymph

Johann Conrad Peyer, 1653-1712. Successively Professor of Logic, Rhetoric, and Medicine at Schaffhausen, Switzerland.
Burrill B. Crohn, Contemporary. Gastroenterologist, Mount Sinai Hospital, New York. First described the disease in 1932.

nodes. Unlike tuberculosis, the affected lymph nodes neither break down nor calcify. Tracing the diseased ileum upwards, it terminates abruptly in normal intestine. Above this there is commonly another (short) area of diseased intestine; this is a so-called 'skip' lesion. Doubtless a comparatively inconspicuous additional lesion is sometimes overlooked, and is one cause of recurrence after resection. In 6 per cent. of cases there is an extension into the cæcum and at times the ascending colon is implicated in the interrupted manner referred to. Examples of primary Crohn's disease of the colon, jejunum, duodenum, and even of the stomach and anus have been reported, which justifies the newer term, regional enteritis.

Pathological Histology.—A characteristic finding is granulomatous infiltration of lymphatics of the submucosa with the presence of non-caseating giant-celled systems. In the late stages of the disease fibrosis extends into and obliterates the submucosa, but usually giant-celled systems can be found in the related mesenteric lymph nodes. An experienced histologist can easily distinguish the condition from tuberculosis.

Clinical Features.—The disease, which is independent of age, sex, social and economic conditions, or geographical location, is increasing in frequency. To some extent it is familial.

Acute regional ileitis occurs only in 5 per cent. of cases. The symptoms and signs resemble those of acute appendicitis, with one exception, viz. diarrhoea almost invariably precedes the acute attack. Exceptionally, perforation of the intestine, resulting in local or diffuse peritonitis, occurs.

Chronic regional ileitis is the usual form of the disease. It can be divided into three stages, but sometimes the second stage is lacking.

First Stage.—There is a history of mild diarrhoea extending over months or years, occurring continuously or in bouts accompanied by intestinal colic, relieved by defæcation. Intermittent pyrexia, seldom more than 99° F. (37.2° C.) is usual, but some patients are afebrile throughout. As a rule a tender mass can be felt in the right iliac fossa, and frequently by a pelvic examination also. There is often a moderate secondary anæmia. Occult blood and some mucus is present in the stools; two-thirds of the patients have some degree of steatorrhœa. A perianal abscess is a frequent accompaniment of early Crohn's disease. The cause is probably an infected anal crypt associated with the concomitant diarrhoea. The high incidence and diagnostic significance of perianal and perirectal abscesses and fistulæ in patients with regional ileitis is generally accepted.

Second stage is characterised by symptoms of acute or chronic intestinal obstruction. Cicatrisation of the granulomatous area has progressed to such an extent that the lumen of the affected portion of the intestine is narrowed (fig. 1168).

Third stage is that of adhesions sometimes accompanied by slow perforation of the intestinal wall. Adhesions are dense, abscess formation is common, and fistulous tracts are wont to develop :—

(a) *Internally* into neighbouring hollow viscera, e.g. a redundant pelvic colon, or occasionally into the right side of the bladder. In all cases of enterocolic and vesico-intestinal fistulæ the possibility of regional ileitis should be considered.

(b) *Externally*, nearly always through the scar of a previous operation for the condition, e.g. appendicectomy.

Radiological Diagnosis.—X-ray examination after a barium meal often

shows lack of segmentation and feeble or absent peristalsis in the affected portion of the intestine, the lumen of which remains constant in diameter. Radiologically, cases can be divided into stenosing and non-stenosing. In



FIG. 1168.—Crohn's disease. Stage of cicatricial contracture.
(From the R.C.S. Museum, Professor G. Cunningham.)

the non-stenosing phase straightening of the valvulæ conniventes is characteristic. When ulceration has occurred multiple defects (cobblestone reticulation) can be seen after the barium has been evacuated from the segment in question. When cicatrization has occurred the radiograph is particularly characteristic; sometimes the terminal ileum is so constricted that the 'string' sign of Kantor (fig. 1169) is seen.



FIG. 1169.—The 'string' sign of Kantor.

Treatment:

Medical Treatment.—In the early stages medical treatment is given an extended trial. It consists of several weeks of rest in bed and a high protein diet with vitamin supplementation. Antibiotics should be used with caution—sulphaphthalidine is best. Most abdominal surgeons are also cautious about the use of steroids, especially if they are called on to operate on patients who have

bled or perforated during steroid therapy. Cortisone or ACTH sometimes brings about dramatic symptomatic improvement, but the effect on the long-term course of the disease is insignificant.

Indications for Surgery.—These are: (1) failure to arrest the course of

the disease by adequate medical treatment; (2) symptoms of chronic obstruction; (3) the presence of fistulæ.

Operation.—Should the abdomen be opened on the mistaken diagnosis of acute appendicitis and acute regional ileitis is found, the one thing *not* to do is to remove the appendix. Appendicectomy frequently determines the development of an external fistula. The correct procedure is to close the abdomen forthwith. Occasionally the condition resolves completely; more often chronic ileitis supervenes.

Surgeons differ as to whether the affected bowel should be resected or by-passed. Probably a by-pass operation is best for young people, especially if the disease is in a florid state, and resection for older patients and where the main trouble is in the cæcum.

On the whole, unless circumstances, such as an enterocolic fistula, leave no other alternative, more and more surgeons are following the advice of Crohn and his surgical colleagues, who for many years have recommended a more conservative course, i.e. division of ileum 6 inches (15 cm.) above the diseased portion, with closure of both ends, followed by ileo-transverse colostomy. The mortality of this operation is almost zero, and the results are at least as good as those following resection. In the cases that do not improve as a result of this short circuit, resection can still be undertaken.

TUBERCULOSIS OF THE INTESTINE

In Britain tuberculosis of the intestine has become infrequent. In countries where the Public Health control of tuberculosis is less strict, the disease is still common.

1. **Ulcerative type** is always secondary to pulmonary tuberculosis, and probably arises as the result of swallowing tubercle bacilli. It is characterised by the presence of multiple ulcers in the terminal ileum, the long axis of each ulcer lying transversely (fig. 1167B). The serous coat overlying the ulcerated segment is thickened, injected, and sparsely bespattered with tubercles. Perforation is rare, but in patients who overcome the infection subsequent stricture or strictures of the ileum are rather frequent.

Clinical Features.—Diarrhoea is the predominant symptom; there is also loss of weight. The stools have a foetid odour, and contain pus and occult blood. Often the patient has received, or is receiving, treatment for pulmonary tuberculosis; more rarely pulmonary tuberculosis is detected for the first time in the course of the investigations.

Radiology.—A barium meal often discloses complete absence of filling of the lower ileum, the cæcum, and most of the ascending colon, due to hypermotility of the ulcerated segment.

Treatment is sanatorium regimen and chemotherapy (p. 23), and provided the intestinal ulceration is not a terminal event of advanced pulmonary tuberculosis, healing often occurs. Operation is required in the rare event of perforation. Occasionally cicatrization causes intestinal obstruction, and calls for surgical intervention.

2. **Hyperplastic tuberculosis** occurs most commonly in the ileo-cæcal region, although solitary or multiple lesions of the lower ileum are met with occasionally. This form of intestinal tuberculosis is consequent upon the ingestion of *Mycobacterium tuberculosis* by a patient with a high resistance to the organism. In Western countries *mycobacterium tuberculosis bovis* is often the causative organism, while in the East the human variety is the culprit (Touffeeq). The infection establishes itself in lymphoid follicles, and spreads to the submucous and subserous planes. The resulting chronic inflammation causes much thickening of the intestinal wall, and consequent narrowing of its lumen. There is early involvement of regional lymph nodes, which may caseate. Unlike regional ileitis (which in many respects this disease simulates), abscess and fistula formation is rare.

Untreated, sooner or later subacute or acute intestinal obstruction supervenes, and in the East, not infrequently, impaction of an enterolith within the narrowed lumen is the precipitating cause.

Clinical Features.—Attacks of abdominal pain with intermittent diarrhoea are the premonitory symptoms. Frequently, the presenting picture is that of the 'blind loop' syndrome (p. 897). The ileum above the partial obstruction is distended and the stasis and consequent infection lead to steatorrhoea, anaemia and loss of weight. Sometimes the presenting picture is that of a mass in the right iliac fossa in a patient



FIG. 1170.—The difficulties in diagnosis of a mass connected with the caecum. In this case the lump outlined on the skin was thought at first to be an appendix abscess. After the caecum and ascending colon had been excised the specimen had many of the appearances of tuberculosis. Histologically the mass proved to be a carcinoma.

with vague ill health. This finding raises the possibility of an appendix mass, a carcinoma of the caecum, Crohn's disease or a tuberculous or actinomycotic granuloma of the caecum. The problem can be quite perplexing (fig. 1170).

Radiography.—In an established case a barium meal reveals a long, narrow filling defect consisting of the terminal ileum and ascending colon, lying vertically. The caecum may become subhepatic (fig. 1171).

Treatment.—When the diagnosis is certain, and the patient has not yet developed obstructive symptoms, sanatorium treatment with chemotherapy is advised and may completely cure the condition. If obstruction is present, operative treatment is required. Right hemicolectomy with removal of the diseased segment of ileum is the treatment of choice. In patients with intestinal obstruction, and those in poor general condition, a defunctioning ileocolostomy similar to that recommended for regional ileitis is advisable. If necessary resection can be undertaken later, for frequently striking improvement occurs after ileocolostomy and general treatment.



FIG. 1171.—Ileocaecal tuberculosis. The filling defect also occurs in carcinoma of the caecum. (Professor A. K. Toufeeq, Lahore, Pakistan.)

ACTINOMYCOSIS OF THE RIGHT ILIAC FOSSA

Actinomycosis develops when a breach of the mucous membrane, caused by disease or trauma of a foreign body, permits the entry of *Actinomyces israeli* (p. 22) into the deeper layers. Abdominal actinomycosis, which is comparatively rare, occurs with equal frequency in rural and urban communities. Unlike intestinal tuberculosis, cicatrization and consequent narrowing of the lumen of the intestine do not occur, neither do the mesenteric lymph nodes become involved. However, suppuration supervenes, and the disease spreads into the retroperitoneal tissues. Eventually, the abdominal wall becomes the seat of multiple indurated discharging sinuses, and the liver becomes involved by way of the portal vein.

Clinical Features.—The usual history is that appendicectomy has been performed for acute or subacute appendicitis. Possibly, had the appendix been subjected to histological scrutiny, actinomyces would have been found. More usually this examination is omitted or the organism is not discovered.

and about three weeks after the operation a mass forms in the right iliac fossa, and soon afterwards the wound commences to discharge. At first the purulent discharge is thin and watery; later, because of secondary infection, it becomes thicker and odorous. Other sinuses form, and fæcal fistulæ are liable to develop. At any stage of the disease, if pus is collected and allowed to trickle down the side of a test-tube, sulphur granules may be discovered. A swab is insufficient. The pus should be sent for immediate bacteriological examination.

Another clinical type is that of a patient, most usually a young adult male, who presents with vague abdominal pain. On examination a hard, slightly tender mass is found in the right iliac fossa. Extension of the disease to the psoas muscle sometimes causes flexion of the hip. Such a finding is characteristic, but is only present when the condition is moderately advanced. Little or no help is derived from radiology in distinguishing actinomycosis of the right iliac fossa from hypertrophic tuberculosis or carcinoma of the cæcum; a deformity of the cæcum is found in all these conditions. Actinomycosis rarely gives rise to obstructive symptoms. When, as is sometimes the case, there is a history of a more or less sudden onset of pain some weeks previously, a subsiding appendix abscess is probably diagnosed, but the mass does not resolve. There is loss of weight, anæmia, and occasional pyrexia. In most instances laparotomy is performed with one of the following findings: (a) an abscess is encountered and drained; (b) the mass is found to be densely adherent to the posterior abdominal wall, and irremovable. In such a case, a by-pass ileo-transverse colostomy as for Crohn's disease (p. 910) is wise; (c) the mass is sufficiently mobile for right hemi-colectomy to be carried out.

Treatment (p. 532).

PNEUMATOSIS CYSTOIDES INTESTINALES

Multiple gas cysts of the intestinal wall are pathological curiosities. Translucent, thin-walled cysts ranging from 1 to 2 or more centimetres in diameter, containing gas, mainly nitrogen, and having a lining of flattened cells of doubtful origin, occur in clusters under the serosa or in the mesenteries of the intestines. The condition nearly always affects the small intestine, but occasionally the colon, and even the rectum, are implicated. It is believed that air enters a breach in the mucosa, as would occur in the case, say, of a duodenal ulcer, and the air is driven onwards by peristalsis. The cysts, which are obvious on a plain radiograph, are usually symptomless, but occasionally they so occlude the lumen of the intestine that resection is necessary.

TUMOURS OF THE SMALL INTESTINE

Compared with the large intestine, the small intestine is rarely the seat of a neoplasm.

Benign.—Adenoma, submucous lipoma and leiomyoma occur from time to time, and sometimes reveal themselves by causing an intussusception. The second most common complication is intestinal bleeding from an adenoma, in which event the diagnosis is frequently long delayed because the tumour is overlooked at a radiological examination by barium meal, and often at operation as well, to be discovered perhaps only after a second or third operation.

Peutz Jegher's syndrome (small intestinal polyposis) consists of (a) familial intestinal adenomatous polyposis affecting mainly the jejunum, where it is a cause of hæmorrhage, and often of intussusception, and (b) melanosis of the oral mucous membrane and the lips. The melanosis takes the form of melanin spots which are

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Harold Jos Jeghers, Contemporary. Professor of Internal Medicine, New Jersey College of Medicine and Dentistry, Jersey City, U.S.A.

sometimes present on the digits and the peri-anal skin, but the pigmentation of the lips (fig. 1172) is the *sine qua non*.

Treatment.—As a malignant change never occurs, resection is necessary only for serious bleeding or intussusception. Large single polyps can be removed by enterotomy or short lengths of heavily involved intestine can be resected.



FIG. 1172.—Melanin spots on the lips of a patient afflicted with Peutz's syndrome.

Malignant :

Sarcoma (40 per cent.).—Lymphosarcoma and spindle-cell sarcoma—more usually the former—occur in the first five decades, the average age of the patient being thirty-five years. There are usually multiple lesions and the disease may follow idiopathic steatorrhœa. The neoplasm tends to convert the affected intestine into a rigid tube without much interference with the size of its lumen until the disease is advanced. Loss of weight and anæmia are the chief early symptoms. Perforation into the peritoneal cavity occurs more often than is the case with

other neoplasms of the small intestine.

Carcinoma (35 per cent.) occurs at the usual carcinoma age. The jejunum is affected three times more often than the ileum. The most frequent symptoms are melæna and those of intestinal obstruction, and in some cases a palpable tumour is present. It is unusual for malignant tumours to cause intussusception. Because the content of the small intestine is fluid, by the time intestinal obstruction has ensued metastases have occurred. Another train of symptoms is dyspepsia associated with melæna and increasing anæmia, in which case a tumour, often of the papilliferous variety, may be revealed radiologically at a barium meal.

Carcinoid (argentaffin) tumour (25 per cent.). The terminal ileum is the second most frequent site for this rare tumour. Unlike carcinoid tumour of the vermiform appendix (the most common site) the tumour metastasises to the regional lymph nodes and to the liver if it is not excised at a comparatively early stage. The tumour is composed of cells from the myenteric plexuses of the intestine. They have an affinity for silver stains (argentaffin) and secrete 5-hydroxytryptamine (5-H.T. or serotonin). This is normally destroyed by the liver but if hepatic metastases form, a high concentration of 5-H.T. occurs in the systemic blood. This causes pulmonary stenosis, diarrhœa, excessively loud and persistent borborygmi and attacks of flushing, especially after alcohol (fig. 1173).

Treatment.—In the case of a pedunculated benign tumour, sometimes extirpation can be effected by enterotomy. In all other instances wide resection of that portion of the intestine bearing the neoplasm, together with its mesentery, followed by end-to-end anastomosis, is indicated.

Prognosis.—Malignant tumours of the small intestine have an evil reputation. However, in the case of carcinoma of the lower ileum, as opposed to the jejunum, with wide resection the prognosis is fair, while the prognosis of carcinoid tumour is even better: should a solitary metastasis be present in the liver, it also should be resected (p. 816).



FIG. 1173.—Patient with secondary carcinoid tumour of the liver (primary in the terminal ileum) exhibiting flushing after a small dose of alcohol. (Dr. P. J. D. Snow, Manchester.)

TUMOURS OF THE LARGE INTESTINE

Benign

Adenoma.—Considerable confusion has arisen between the terms adenoma and adenomatous polypus, the more so because often the 'adenoma-

tous' is omitted, and the neoplasm is called a 'polypus', a term which, in point of fact, should be reserved for a chronic inflammatory condition (p. 903). So-called adenomatous polypi are adenomas with a well-developed stalk (fig. 1174), whereas an adenoma of the colon is sessile (fig. 1175). A



FIG. 1174.—Pedunculated adenomatous polyp of the large intestine. Longitudinal section. (J. H. Saint, F.R.C.S., Santa Barbara, California.)



FIG. 1175.—Adenoma of the colon. (Max Pemberton, F.R.C.S., Enfield Middlesex.)

solitary adenoma of the colon is acquired (see below for the multiple familial variety). It occurs in patients over forty years of age, and unless it is pedunculated, it cannot be distinguished macroscopically from a papilliferous carcinoma. In specimens of papilliferous carcinoma of the colon, often tiny adenomata are found adjacent to the parent tumour.

Treatment.—As a rule an adenoma of the colon should be treated by limited resection of that part of the intestine in which it is contained. Only in this way can recurrence and a carcinomatous change be prevented. On the other hand, if the adenoma is situated in a portion of the terminal colon it can be removed by fulguration through a sigmoidoscope. The patient should be kept under intermittent observation for many years.

Familial Adenomatous Polyposis of the Colon, which must be distinguished from Peutz's syndrome (p. 915), is transmitted from both sexes to both sexes, though males are affected more frequently than females. Lockhart-Mummery and Cuthbert Dukes studied 1069 members of 58 families; of these members 218 had polyposis, and 154 of them developed colonic carcinoma. The adenomatous polypi are most frequently situated in the sigmoid and the rectum. Often hundreds of tumours are present. The patient complains of attacks of lower abdominal pain associated with loss of weight, diarrhoea, and tenesmus, and the passage of blood and mucus, and sometimes pus—all symptoms very like those of ulcerative colitis. A rectal examination may reveal one or more adenomatous polypi. Sigmoidoscopy shows a variety of neoplasms ranging from small sessile pink elevations to pedunculated tumours. A barium enema, especially a contrast barium enema (p. 921), outlines the larger polypi.

Prevention.—(1) All members of the family should be examined at ten years. Repeat every two years. (2) Most of those who are going to get polypi will have them at twenty and these require operation. (3) If there are no polypi at twenty, continue with five-yearly examination till forty. If there are still no polypi there is

no inherited gene. No case of carcinomatous change before the age of twenty has been reported in this disease.

Treatment.—If operation is necessary, complete colectomy is advisable. A stump of rectum can remain and the terminal ileum anastomosed to it, the reasons being (a) the risk of carcinoma in the rectal stump is small if the patient is carefully followed-up; (b) an artificial anus is avoided; (c) other members of the family can be persuaded more easily to undergo the operation before late symptoms develop.

Hæmangioma.—A localised submucous telangiectasis is the cause of bleeding, which is often profuse. When the lesion is beyond the sigmoidoscopy field often the only method of detecting it is to operate while the bleeding is in progress. The distribution of blood within the intestine is noted; scrutiny of the blood-containing portion of the intestine usually reveals a dilated leash of veins in one portion of the mesocolon. At this site the intestine is opened and the tumour is resected.

Lipoma is less frequently encountered in the large than in the small intestine, and is almost confined to the cæcum. The tumour is submucous and in more than half the cases it is the cause of an intussusception. Other symptoms to which it gives rise are almost impossible to distinguish from those of a carcinoma; even macroscopical or microscopical blood is found in the stools. A barium meal may suggest a lipoma because of its smooth contour.

Malignant

CARCINOMA OF THE COLON

Pathology.—Microscopically, the neoplasm is a columnar-celled carcinoma originating in the epithelial cells that line the colon, or in the crypts of Lieberkühn. Macroscopically the growth takes one of four forms (fig. 1176):

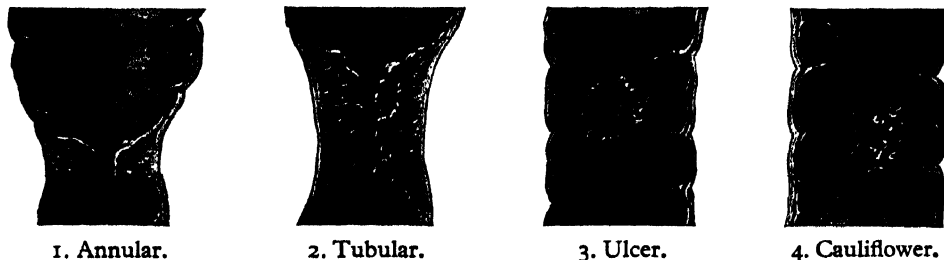


FIG. 1176.—The four macroscopical varieties of carcinoma of the colon.

Type 4 is the least malignant form, and this papilliferous carcinoma commences as a benign adenoma. The annular variety carries a relatively good prognosis, not because the growth is of low-grade malignancy, but because it gives rise to early obstructive symptoms, and therefore is often extirpated before metastases have occurred (Grey Turner).

Site.—The most frequent site is toward the termination of the colon, viz. the pelvic colon and the rectosigmoid junction (fig. 1177).

The Spread of Carcinoma of the Colon.—Generally speaking, it is a comparatively slowly growing neoplasm, and if extirpated thoroughly at a reasonably early period a cure can be hopefully anticipated.

Local Spread.—The growth is limited to the bowel for a considerable time. It spreads round the intestinal wall, and to a certain extent longitudinally, but usually it causes intestinal obstruction before it has penetrated adjacent structures. Particularly in the ulcerative variety, penetration of the serous coat is apt to occur and, according to the segment involved, adjacent structures then become invaded by the growth. When a hollow viscus is thus implicated an internal fistula results;

also the perforation may lead to the formation of a local abscess and an external fæcal fistula.

Lymphatic Spread.—The lymph nodes draining the colon are grouped as follows :

1. *The epicolic lymph nodes*, situated in the immediate vicinity of the bowel wall.

2. *The paracolic lymph nodes*, lying in relationship to the leash of blood-vessels proceeding to the colonic walls.

3. *The intermediate lymph nodes*, arranged along the ileo-colic, right colic, mid colic, left colic, and the sigmoid arteries. In the last instance the paracolic lymph nodes are often absent.

4. *The main lymph nodes*, aggregated around the superior and inferior mesenteric vessels, where they take origin from the abdominal aorta.

Spread by the blood-stream occurs late. Metastases are carried to the liver.

Clinical Features.—Carcinoma of the colon usually occurs in patients over fifty years of age, but it is not rare earlier in adult life. Exceptionally it appears in childhood, when, owing to delayed diagnosis, prognosis is poor. Twenty-five per cent. of cases present as emergencies with intestinal obstruction or peritonitis. It must be remembered that in any case of colonic bleeding in patients over forty years of age, complete investigation of the colon is required.

Men are attacked more frequently than women (3 : 2), although carcinoma of the ascending colon is encountered more often in women.

While certain outstanding symptoms frequently prevail in all types (fig. 1176) and at all sites (fig. 1177), it is instructive to contrast and to compare the symptoms produced by a carcinoma of the left side of the colon with that of the right, and to refer to the symptomatology of carcinoma of the sigmoid and that of the transverse colon.

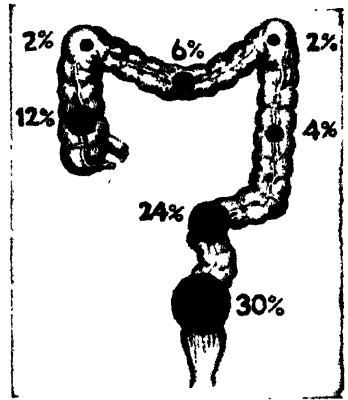


FIG. 1177.—Relative frequency of carcinoma in various portions of the large intestine omitting the rectum proper. (Mayo Clinic statistics.)

<i>Symptoms</i>	<i>Right Colon</i>	<i>Left Colon</i>	<i>Sigmoid</i>
Abdominal pain	78	68	51
Alteration of bowel habit . .	30	58	70
Loss of weight	50	15	20
Vomiting (frequently with colic)	32	15	0
Anorexia	18	9	0
Faintness, breathlessness . .	20	9	6
Bleeding per rectum	8	9	29
Lump present	67	46	39
Indigestion	8	0	0
Acute-on-chronic obstruction .	8	21	29

The duration of symptoms is extremely variable ; 5·5 months is the average.

(E. G. Muir's statistics—figures given are percentages.)

Carcinoma of the Left Side of the Colon.—As will be seen on reference to fig. 1177, in about 75 per cent. of cases the neoplasm is situated on the left side of the colon. Neoplasms in this situation usually are of the stenosing

variety (fig. 1176, 1 and 2) and as here the fæcal content is relatively solid, and the lumen of the bowel relatively narrow, the main symptoms are those of increasing intestinal obstruction in about 25 per cent. of cases.

Pain.—This is usually intestinal colic. If it becomes a constant ache, it suggests inoperability or pericolitis.

Alteration of Bowel Habit.—An adult who has had regular bowel movement all his life, in a short space of time develops irregularity. The patient often states that he has *increasing* difficulty in getting the bowels to move, and that he has to take *increasing* doses of purgatives. Because of the drastic purgation, or on account of irritation by the scybala causing excessive secretion of mucus above the constricting neoplasm, attacks of constipation are followed by diarrhœa.

Palpable Lump.—Very often the lump that is felt on abdominal, rectal, or abdomino-rectal examination, is not the growth itself, but impacted fæces above it (Oldham). When the carcinoma is situated in a pendulous pelvic colon a hard movable swelling is often felt in the rectovesical pouch.

Distension.—Lower abdominal distension is not uncommon, and, like the pain, is relieved by passing flatus.

Carcinoma of the sigmoid follows the general pattern of the above, but there are differences.

Pain, when it occurs, is usually colicky from the commencement.

Tenesmus.—Growths of papilliferous type situated low in the colon are inclined to give rise to a feeling of the need for evacuation, which may result in tenesmus accompanied by the passage of mucus and blood, especially in the early morning.

Bladder symptoms are not unusual, and in some instances they herald colo-vesical fistula (p. 1141).

Carcinoma of the transverse colon is frequently mistaken for a carcinoma of the stomach because of the position of the tumour together with the anæmia and lassitude that it sometimes engenders.

Carcinoma of the cæcum and ascending colon presents in several guises:

(a) Anæmia, severe and unyielding to treatment, is a frequent predominating feature. Should a palpable tumour be present, the diagnosis is, to some extent, simplified.

(b) The presence of a mass in the right iliac fossa often proves a diagnostic conundrum (fig. 1170).

(c) Cæcal carcinoma sometimes is discovered unexpectedly at operation for acute appendicitis or for an appendix abscess that 'fails to resolve'. On rare occasions the appendix is inflamed, or even gangrenous, from obstruction to its lumen by the carcinoma.

(d) Less commonly a papilliferous growth is the apex of an intussusception. A lump, present at one time and smaller or absent at another (owing to partial reduction), associated with attacks of acute abdominal pain, is characteristic of this complication.

METHODS OF INVESTIGATION

Sigmoidoscopy should be performed in cases where blood and mucus have been passed (fig. 1178) and also in suspected cases when a barium enema is negative, because early growths in the lower part of the pelvic colon are not always visualised by radiography.

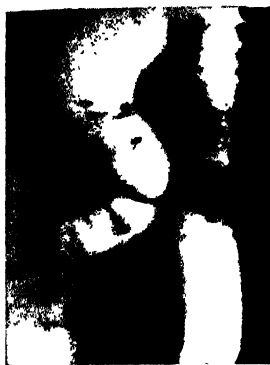


FIG. 1179.—Barium enema showing a carcinoma of the descending colon.

Radiography after a barium enema often shows a carcinoma of the colon as a constant, irregular filling defect (fig. 1179). On the other hand, negative radiography in comparatively early cases of carcinoma of the colon is not by any means conclusive evidence of the absence of a growth.

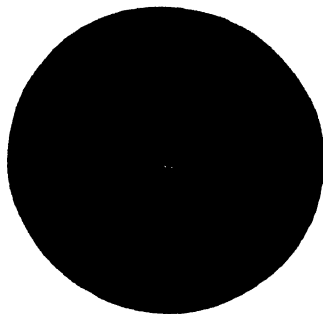


FIG. 1178.—An example of the inestimable value of sigmoidoscopy. The patient had been diagnosed and treated for some weeks for 'ulcerative colitis'. A barium enema was negative. Sigmoidoscopy showed a small cauliflower carcinoma giving rise to an intussusception.

In 75 cases of carcinoma

of the colon examined radiologically, no abnormality was detected in 8 per cent. This entailed considerable delay in diagnosis (Ramsay).

More refined methods in radiological technique are often rewarding, viz:

Contrast Enema.—In cases of a neoplasm involving only the mucous membrane, a contrast enema is very valuable. The barium emulsion is partly evacuated and air is injected into the colon. By this means the walls of the colon become delineated and a neoplasm that fails to alter the contour of the barium-filled colon may be demonstrated.

A tumour of the cæcum is more likely to be discovered by a barium meal than a barium enema. As a rule, in suspected cases of carcinoma of the colon, a barium meal is inadvisable because inspissated barium can precipitate intestinal obstruction if the lumen of the bowel is narrowed.

Exfoliative cytology.—In experienced hands colonic exfoliative cytology is a valuable adjunct in the diagnosis of obscure cases of carcinoma of the colon. Enemas are given until the return fluid is clear. The presence of malignant cells in smears of the washings is conclusive evidence of a malignant lesion of the colon. To obtain satisfactory results, the patient must be prepared carefully by a somewhat laborious procedure. After an interval of five to ten minutes, the returned fluid is collected and centrifuged. Films from the sediment are prepared and stained. Successful diagnoses have been made by this method in growths situated in all parts of the colon.

TREATMENT

Pre-operative Treatment.—When there is no intestinal obstruction, blood transfusion to correct anæmia, if present, enemas to cleanse the bowel, a high caloric and low residue diet, together with succinylsulphathiazole, 2 G. (30 grains) four-hourly for five days, are required. This sulphonamide

gives watery stools so that the bowel mucosa is clean for purposes of anastomosis and it effectively reduces the number of virulent organisms ordinarily present in the colon. This is probably all that is required. Other antibiotics such as neomycin may completely sterilise the bowel but the risk of staphylococcal enteritis (p. 3) after its use is very real.

When intestinal obstruction is present, preliminary drainage of the intestine proximal to the obstruction must be performed (p. 929).

Operation.—The Test of Operability.—The abdomen having been opened in the first place through a short paramedian incision, which is extended if the growth is removable: (1) the liver is palpated for secondary deposits, the presence of which is not necessarily a contraindication to resection, as the best palliative treatment for carcinoma of the colon is removal of the tumour; (2) the peritoneum, particularly the pelvic peritoneum, is inspected, if possible, and palpated for neoplastic implantations; (3) the various groups of lymph nodes that drain the involved segment are palpated. Their enlargement does not necessarily mean that they are invaded by metastases, for the enlargement may be inflammatory; (4) the neoplasm is examined with a view to ascertaining if it is fixed or free, and if it can be mobilised it is operable. "The whole colon above the last 3 inches (7.5 cm.) of the pelvic portion is either mobile or can be mobilised" (Ogilvie).

The operations to be described are designed to remove the lymph node fields which may be involved by metastases from the particular primary growth concerned. Lesser resections are indicated, however, should hepatic metastases render the condition incurable.

Carcinoma of the cæcum or the ascending colon is treated, when resectable, by right hemicolectomy (fig. 1180).

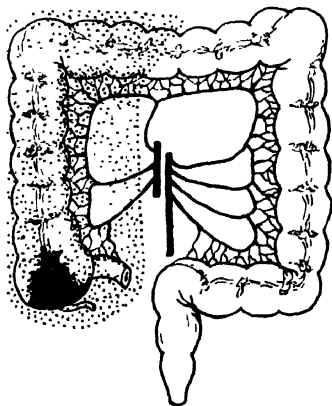


FIG. 1180.—Area to be resected when the growth is situated in the cæcum.

The abdomen is opened through a right paramedian incision. The technique of right colectomy should include at the outset ligatures placed around the bowel to prevent intraluminal spread. The peritoneum an inch (2.5 cm.) or more lateral to the ascending colon is incised and the incision is carried around the hepatic flexure. The right colon is elevated, with the leaf of peritoneum containing its vessels and lymph nodes, from the posterior abdominal wall, care being taken not to injure the ureter, spermatic vessels, or the duodenum. The peritoneum is separated medially to near the origin of the ileo-colic artery, which is divided between ligatures, as also is the right colic artery when that vessel has a separate origin from the superior mesenteric. The mesentery of the last foot (30 cm.) of ileum, and the leaf of raised peritoneum attached to the cæcum, ascending colon and hepatic flexure, after ligation of the blood-vessels contained therein, is divided as far as the proximal third of the transverse colon (fig. 1180). The surgeon, having verified that the blood supply to the proposed intestinal stumps is adequate, forthwith clamps and divides the ileum and the transverse colon at the levels of their respective severed mesenteries, and excises the free intestine. An end-to-end anastomosis between the ileum and transverse colon is then

carried out and their respective mesenteries approximated with catgut. The abdomen is then closed.

Carcinoma of the Hepatic Flexure.—When the hepatic flexure is the seat of the neoplasm the resection must be extended correspondingly (fig. 1181).

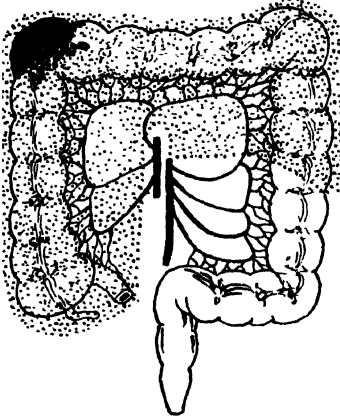


FIG. 1181.—Area to be resected when the growth is situated at the hepatic flexure.

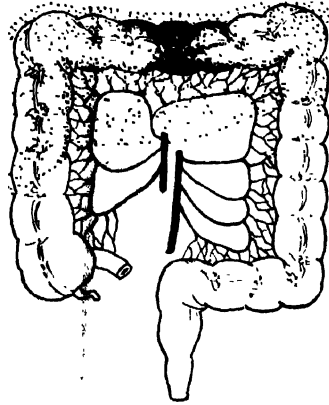


FIG. 1182.—Area to be resected when the neoplasm is situated in the transverse colon.

Carcinoma of the Transverse Colon.—When there is no obstruction, excision of the transverse colon and the two flexures (fig. 1182), together with the transverse mesocolon and the greater omentum, followed by end-to-end anastomosis, is a satisfactory procedure.

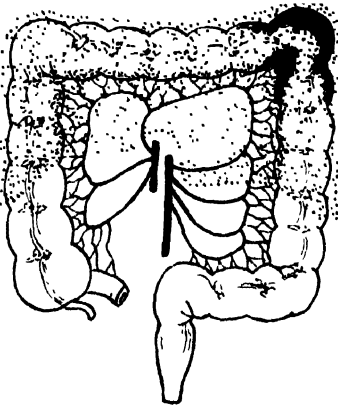


FIG. 1183.—Area to be resected when the neoplasm is situated at the splenic flexure, or the proximal part of the descending colon.

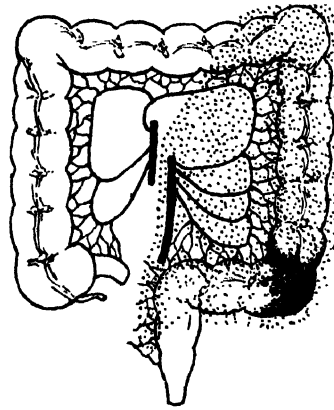


FIG. 1184.—Area to be resected in the case of a carcinoma of the pelvic colon.

Carcinoma of the Splenic Flexure or descending colon. The extent of the resection is shown in fig. 1183.

The phrenico-colic ligament is divided, and after incising the parietal peritoneum from the splenic flexure to the pelvic colon, the splenic flexure, descending colon, and pelvic colon are raised from the posterior abdominal wall in the same way as the

ascending colon is raised on the right side. When this has been achieved, the left branch of the middle colic and the left colic arteries are ligated near their origins, and the peritoneal leaf with its contained lymph nodes is divided. Excision with end-to-end anastomosis is then carried out.

Carcinoma of the Pelvic Colon.—A left paramedian incision gives a good approach. The left half of the colon is mobilised completely (fig. 1184).

In order that the operation be rendered radical, the inferior mesenteric artery below its left colic branch, together with the related paracolic lymph nodes, must be included in the resection. This entails carrying the dissection as far as the upper third of the rectum, and accordingly the incision in the lateral leaf of peritoneum is extended downward to an appropriate level.

In every case of resection of the colon with anastomosis, drainage down to the site of anastomosis should be provided.

Modern methods of bowel preparation render one-stage resection and anastomosis reasonably safe in *non-obstructed* cases of colonic carcinoma but if any doubt exists a protective colostomy must be added. The methods of dealing with large bowel obstruction due to colonic carcinoma are described on p. 929.

Post-operative Care following Colonic Resection.—Post-operative treatment includes the administration of antibiotics to guard against possible infection of the anastomotic area by *Cl. welchii*. Fluids are not given by mouth after anastomotic operations until flatus is passed (see Paralytic Ileus, p. 949).

When the Growth is found to be Inoperable.—If the growth is in the upper part of the left colon, transverse colostomy is performed. If it is in the pelvic colon, left iliac colostomy is preferable. When there is an inoperable growth in the ascending colon, ileo-colostomy is the best procedure. However, about 90 per cent. of colonic carcinomas can be resected, if necessary at a 'second look' operation, when infection will have subsided following the by-passing of fæces. As these are slow-growing neoplasms, after subsequent colostomy-closure, these patients often enjoy several years of trouble-free life.



FIG. 1185.—Fæcal fistula with prolapse of mucous membrane following acute appendicitis with gangrene of the cæcum. Aluminium paint is protecting the skin from excoriation.

FÆCAL FISTULÆ

An external fistula communicating with the cæcum sometimes follows an operation for gangrenous appendicitis (fig. 1185) or the opening of an appendix abscess. A fæcal fistula can occur from necrosis of a gangrenous patch of intestine after the relief of a strangulated hernia, or from a leak after an intestinal anastomosis. The opening of an abscess connected with chronic diverticulitis or carcinoma of the colon frequently results in a fæcal fistula. Tuberculous peritonitis, ileo-cæcal actinomycosis,

amoebiasis, and regional ileitis (in this instance nearly always following operation) are also causes of fæcal fistulæ, which may be multiple. About 4 per cent. of chronic fæcal fistulæ in hospital are kept open by patients pushing in knitting needles or other articles for reasons best known to themselves.

External fæcal fistulæ can be divided into three varieties :

1. A track lined by mucous membrane which protrudes above skin level.
2. A direct track lined by granulation tissue communicating with the exterior.

3. A long, tortuous track lined by fibrous tissue and partly epithelialised.

The discharge from a fistula connected with the duodenum or jejunum is bile-stained and causes severe excoriation of the skin. When the ileum or cæcum are concerned, the discharge is fluid fæcal matter ; when the distal colon is involved, it is solid or semi-solid fæcal matter. In some cases, when the leak from the small intestine or cæcum is small, it may be difficult to distinguish a fæcal discharge from fæculent pus. If methylene blue is administered by mouth and a fæcal leak is present, the blue colour will be distinguished easily in the discharge a few hours later. Often the site of the leak and the length of the fistula can be determined by radiography after a barium meal or barium enema. Should this fail to demonstrate the internal orifice, injection of lipiodol into the external opening will usually give the desired information.

Treatment.—Fæcal fistulæ, especially those in connection with the small intestine, tend to heal spontaneously, provided there is no obstruction beyond the fistulous opening. The abdominal wall must be protected from erosion by the use of a disposable ileostomy bag (p. 908).

The higher the fistula in the intestinal canal the more skin excoriation must be expected. This reaches its zenith in the case of a duodenal fistula (p. 735). A sump drain (p. 859) to remove the enzyme-laden discharge is a fundamental requirement. Such fistulæ cause dehydration and hypoproteinæmia and intravenous parenteral feeding will be required. If there is no prospect of rapid healing, a feeding jejunostomy is often a life-saving measure (p. 735).

Isogel or Pulv. cretæ aromat. ʒi (4 G.) q.i.d. will make the fæces more solid and avoidance of eggs and fish will help to reduce the malodour.

A fæcal fistula with mucosa visibly continuous with the skin edge will never close spontaneously. In some of these cases, where the opening is a large one, the intestine tends to prolapse upon the surface (fig. 1185).

The operative treatment for closure of a fæcal fistula consists in making an incision encircling the fistula and dissecting up the tract through the abdominal wall and the peritoneum. The base of the fistula, now free, is crushed, ligated, and oversewn. The abdominal wall is then closed in layers. In the case of a colo-cutaneous fistula connected with colonic diverticulitis, if the fistula fails to heal after a few weeks, resection of the affected segment is usually advisable (p. 902). Should the mass from which the fistula arises prove to be an inoperable carcinoma, a defunctioning colostomy should be performed at a higher level. When there is no obvious cause for the fistula, the discharge must be examined on several occasions for *actinomyces* or *mycobacterium tuberculosis*. The demonstration in a plain X-ray of calcified tuberculous mesenteric lymph nodes favours tuberculosis as the cause. In complicated fistulæ, an operation to defunction the loop of intestine involved, is probably the best procedure.

CHAPTER 39

INTESTINAL OBSTRUCTION

INTESTINAL obstruction is a common surgical emergency and because of its serious nature it demands early diagnosis and speedy relief. It may be classified into two types, according to whether peristalsis is present or absent.

1. **Dynamic.**—Here there is increasing peristalsis working against an obstructing agent, which may be in the *lumen*, such as a bolus of incompletely digested material, inspissated fæces, or a gall stone; *in the wall*, such as an inflammatory or malignant stricture; or *outside the wall*, as in herniæ or adhesions.

2. **Adynamic** (p. 949).—In this condition, peristalsis ceases and no true propulsive waves occur—as in paralytic ileus or mesenteric vascular occlusion.

DYNAMIC OBSTRUCTION

These are classified clinically into three types:

1. *Acute obstruction* confined to the small gut with central abdominal pain, early vomiting, abdominal distension, and constipation as a late feature.

2. *Chronic obstruction* confined to the large bowel with abdominal colic at first and absolute constipation and distension later.

3. *Acute-on-chronic obstruction* which spreads from the large bowel to involve the small intestine and gives early pain and constipation, followed by general distension and vomiting.

PATHOLOGY

At the outset, the intestine *above* the point of obstruction endeavours to overcome the obstruction by vigorous peristalsis. Increased peristalsis continues for a period of from forty-eight hours to several days; the more distal the point of obstruction, the longer does it remain vigorous. If the obstruction is not relieved, a time is reached when increasing distension causes peristalsis to become less and less; finally, peristalsis ceases, and the obstructed intestine becomes flaccid and paralysed.

For two or three hours following the obstruction, the intestine *below* the point of obstruction exhibits normal peristalsis, and absorption from it continues, until the residue of its contents has been passed onwards. Then the empty intestine becomes immobile, contracted, and pale, and so it remains, until the obstruction has been overcome, or death ensues.

Distension.—This occurs proximal to the obstruction, and begins immediately after the obstruction occurs. The cause of distension is two-fold:

(a) **Gas.**—This consists of swallowed atmospheric air (68 per cent.), diffusion from blood into the bowel lumen (22 per cent.), and the products of digestion and bacterial activity (10 per cent.). When the oxygen has been absorbed into the blood-stream.

he resultant mixture is made up of nitrogen (90 per cent.), carbon dioxide, and hydrogen sulphide.

(b) **Fluid.**—This is made up by the various digestive juices—about 8,000 ml. per twenty-four hours.

<i>Above pylorus.</i>	4,000 ml.	Saliva 1,500 ml.
		Gastric 2,500 ml.
<i>Below pylorus.</i>	4,000 ml.	Bile and pancreatic 1,000 ml.
		Succus entericus 3,000 ml.

In obstruction, absorption from the gut is retarded but excretion of water and electrolytes into the lumen persists and may even be increased. Deprivation of water and electrolytes, therefore, is due to:

- (a) Vomiting.
- (b) Defective absorption.
- (c) Sequestration in lumen of bowel.

The severity of depletion and speed with which it becomes manifest is dependent upon the level of obstruction. It is most severe and occurs early in high small intestinal obstruction, later in ileal obstruction, and is slow to appear in colon obstruction. (For effects of fluid and electrolyte loss, see Chapter 6.)

Intestinal Toxins.—It is well known that release of intestinal obstruction may be followed by death, particularly in cases of strangulation. In unrelieved strangulation, toxic substances appear in the peritoneal cavity only when the viability of the bowel wall is affected. However, when obstruction is relieved, these toxins may pass on to the bowel where absorption can occur. It is probable that the substances involved are endotoxins of gram-negative bacilli. This factor stresses the need for intestinal decompression before and during operation.

Strangulation of the bowel occurs when it is trapped by a hernia or a band, or involved in a volvulus or intussusception in such a way that its *blood supply is progressively interfered with*. It is a very dangerous condition and demands early treatment. The first effect of strangulation is to compress the veins so as to cause the strangulated bowel, and its involved mesentery, to become blue and congested. Mesenteric vascular occlusion alone gives rise to gangrene without mechanical obstruction.

The Onset of Gangrene.—Much depends on the tightness of the constricting agent. When the venous return is completely occluded, the colour of the intestine turns from purple to black. About this time, in many instances, owing to increased œdema at the point of obstruction, the arterial supply is jeopardised. Then the peritoneal coat loses its glistening appearance, the mucous membrane becomes ulcerated, and moist gangrene is imminent.

Loss of blood volume into the congested segment is proportional to the length of that segment. When, as is often the case in strangulated external hernia, only a few inches are involved, the amount of blood thus imprisoned is inconsequential; on the other hand, when a large coil of intestine becomes strangulated the loss of blood is sufficient to render the patient oligæmic; when several feet of small intestine are involved the volume of circulating blood is so reduced as to imperil the patient's life.

Distension.—For a considerable time the strangulated segment (fig. 1186 (B)) alone distends, the greatest distension occurring when the venous return is completely obstructed while the arterial supply remains unimpaired. Unlike non-strangulating obstruction, early distension of the proximal intestine (fig. 1186 (A)) is absent; indeed, for a time varying from

a few minutes to several hours the proximal intestine contracts. After this varying interval, vigorous peristalsis occurs in the proximal segment, but is still unaccompanied by distension. By the time gangrene of the strangulated segment is imminent, retrograde thrombosis is proceeding along the related tributaries of the mesenteric vein. Distension then appears both on the proximal and distal sides of the strangulation (fig. 1186 (A) and (C)) (Chesterman).

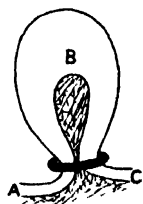


FIG. 1186.

Transmigration of Bacteria and Toxins.—When the wall of the intestine becomes partly devitalised, both bacterial toxins and the products of tissue autolysis pass into the peritoneal cavity, there to be absorbed into the circulation. This is followed by the migration of bacteria, and peritonitis follows. So it comes about that strangulation in an *external* hernia is far less dangerous than intraperitoneal strangulation, for in the former the transudate containing lethal toxins and bacteria is confined to the comparatively small absorptive area of the hernial sac.

Closed-loop obstruction is present in the majority of cases of intestinal strangulation. In its typical form it is seen in carcinomatous stricture of the colon (fig. 1187). Distally the colon is occluded by the neoplasm, while in one-third of cases the ileo-cæcal valve prevents regurgitation of the contents of the large intestine into the ileum, and consequently that part of the colon proximal to the neoplasm is closed at both ends (fig. 1187). As a result of anti-peristalsis the pressure within the cæcum becomes so high as to compress the blood-vessels within its walls.

If the obstruction is unrelieved, stercoral ulceration, gangrene, and perforation of the cæcum will eventually occur.



FIG. 1187.—Carcinomatous stricture of the hepatic flexure: closed-loop obstruction.

CLINICAL FEATURES OF ACUTE INTESTINAL OBSTRUCTION

There are four important symptoms and signs: pain, vomiting, distension, and constipation. These must be carefully looked for in each case.

Acute Obstruction.

1. *Abdominal pain* is the first symptom: it commences suddenly, and often without warning. It becomes increasingly severe, then passes off gradually, only to return at intervals of a few minutes. These attacks of intestinal colic, which last from three to five minutes, spread all over the abdomen, but are localised mainly at the umbilicus. In between attacks the patient is often quite free from pain. Recurring attacks of severe abdominal pain are a leading feature of all varieties of acute intestinal obstruction, with the sole exception of paralytic ileus (p. 949).

2. *Vomiting*.—When the jejunum is mechanically obstructed, vomiting occurs with the first and each succeeding attack of pain. In the much more common obstruction of the ileum the patient may vomit once, following which there is an interval of several hours during which time the attacks of pain

occur without vomiting. Ultimately copious, forcible, oft-repeated vomiting sets in. As acute intestinal obstruction progresses, the character of the vomitus alters. Initially it contains partly digested food; next it consists entirely of mucoid fluid; thereafter the vomitus becomes yellow or green from regurgitation of bile; finally, it is feculent.

3. *Distension*.—In early cases of obstruction of the small intestine abdominal distension is often slight, or even absent. Centrally placed distension is present in fully established cases of obstruction to the ileum. *Visible peristalsis* may be present (fig. 1188). In order to observe it the abdomen must be watched for several minutes. *Borborygmi* are sometimes loud enough to be heard by the unaided ear. More often auscultation is necessary. The sound of turbulent peristalsis coinciding with an attack of colic is valuable evidence of intestinal obstruction. In all cases of suspected intestinal obstruction it is essential to *examine the common hernial sites*. An irreducible external hernia may be present although the patient is entirely unaware of it.



FIG. 1188.—Visible peristalsis. Intestinal obstruction due to the strangulated right femoral hernia, to which the arrow points.

4. *Constipation*.—In complete intestinal obstruction, after the contents of the bowel below the obstruction have been evacuated, there is constipation, and usually neither feces nor flatus is passed, i.e. *absolute* constipation. It should be noted that there may be a natural action of the bowels after the onset of the attack, and that it is commonplace for an enema to yield a small fecal result.

The rule that constipation is present in intestinal obstruction does not apply in cases of Richter's hernia (p. 1034), gall stone obturation (p. 947), mesenteric vascular occlusion (p. 948), and intestinal obstruction associated with a pelvic abscess (p. 873).

Dehydration.—Repeated vomiting and also loss of absorptive power by the distended intestine leads to dehydration, and when the patient is first examined obvious signs of dehydration—a dry skin, a dry tongue, and sunken eyes—may be present. The output of urine is small; it is concentrated, and contains little or no chlorides.

Chronic Obstruction.—This is commonly due to carcinoma or diverticulitis of the colon. Here *constipation* appears first, may last for days or weeks and finally becomes absolute when the passage of flatus ceases. Abdominal *distension* then occurs, especially in the flanks. No matter where the obstruction lies, whether in the ascending, transverse, or descending portions of the colon or even in the rectum, the brunt of the obstruction is borne by the cæcum (fig. 1189). This has been likened to a gun back-

firing into its breach, and the cæcum becomes ballooned. Pain accompanies the distension and there are regular bouts of colic, usually in the hypogastrium. Soon after the onset of intestinal colic, if the abdomen is watched, the cæcum sometimes can be seen to distend with each attack of colic, and subside as the colic passes off. It is not uncommon for vomiting to be delayed for two or three days, and on this account signs of dehydration are exceptional. On abdominal palpation, a neoplasm occasionally can be felt in the line of the colon; but more usually the relatively small lump, occasioned by a cicatrising neoplasm is obscured by the distension. A rectal examination will enable a carcinoma of the rectum, or a mass of impacted fæces, to be felt, and it may be possible to feel a neoplasm in the pelvic colon occupying the recto-vesical pouch. An enema is

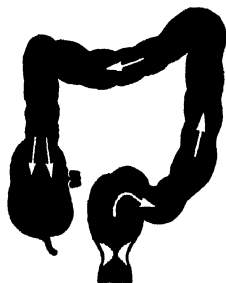


FIG. 1189.—Wherever the large intestine is obstructed the cæcum bears the brunt.

usually given but, in the presence of absolute obstruction is returned without force, fæces, or flatus.

Acute-on-Chronic Obstruction.—This starts with all the features of chronic obstruction but after a few days the pain, vomiting, and central distension betoken the fact that the ileo-cæcal valve is open and fæculent fluid has passed up into the ileum where it is rapidly vomited.

Strangulation.—It is of the highest importance to distinguish strangulating from non-strangulating intestinal obstruction, because if the former is not relieved by an urgent operation, gangrene follows quickly. The diagnosis is made entirely by clinical methods; the picture is usually that of an obstruction together with a degree of shock which is sometimes severe. In some instances the differentiation is not difficult, for when the strangulation is a tight one, unlike non-strangulating obstruction, pain is never completely absent. In strangulation, the symptoms usually commence very suddenly and spasms of intestinal colic recur three or four times a minute. Generalised tenderness and sometimes rigidity are indicative that an early laparotomy should be performed. Much more difficulty is encountered when the strangulation commences as a mild venous occlusion. In such circumstances the presence and character of local tenderness is of great significance, and frequently two or more clinical examinations at half-hourly intervals are required to collect sufficient data to make a confident diagnosis of strangulation. In non-strangulating obstruction there may be an area of localised tenderness at the site of obstruction; in strangulation there is always tenderness over an intra-abdominal strangulated coil and the pain is made worse if the palpating hand is suddenly removed. *This rebound tenderness is a distinctive sign of strangulation.* Cases of intestinal obstruction in which pain persists in spite of one, or at the most two, hours of treatment by gastro-duodenal aspiration, even in the absence of any of the above signs, should be diagnosed as intestinal strangulation. Where the strangulation occurs in an external hernia the lump is *tense, tender*, there is *no cough impulse* and it has *recently increased in size*. These four points are most valuable in diagnosis.

X-rays.—In all cases, where the diagnosis is not clear, a 'scout' film of the abdomen, with the patient standing and lying down, must be taken in the X-ray department as soon as possible.

Gas Shadows.—When the jejunum, the ileum, or the colon is distended with gas, each has a characteristic appearance that allows it to be distinguished radiologically. The diameter of the viscus is no criterion as to whether it is small or large intestine. Obstructed small intestine is revealed by relatively straight segments that generally lie more or less transversely; obstructed large intestine is disclosed by its haustration markings; a distended cæcum is shown by a rounded gas shadow.

Jejunum is characterised by its valvulæ conniventes that pass from the antimesenteric to the mesenteric border, spaced regularly, giving rise to a concertina effect (fig. 1190 (a)).

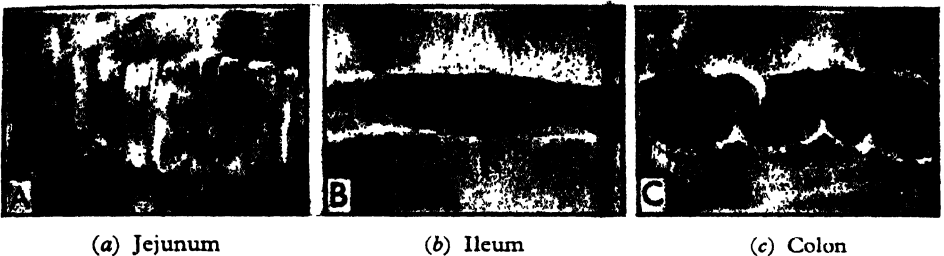


FIG. 1190.—Gas shadows characteristic of various parts of the intestine.

Ileum.—The distal ileum is piquantly described by Wangensteen as being 'characterless' (fig. 1190 (b)).

Large intestine (the cæcum excepted) shows haustral folds. Haustral folds, unlike the valvulæ conniventes, are spaced irregularly and the indentations are not placed opposite one another (fig. 1190 (c)).

Fluid Levels.—In infants under the age of two years a few fluid levels in the small intestine are a normal occurrence. In adults, two inconstant fluid levels must be regarded as physiological. One is at the duodenal cap; the other, which is more infrequent, is within the terminal ileum. In intestinal obstruction it takes a little time for the gas to separate from the fluid; consequently fluid levels appear later than gas shadows. When paralysis of the intestine has occurred, fluid levels become more conspicuous and more numerous. By the time fluid levels are pronounced, obstruction is advanced. The number of fluid levels is proportionate to the degree of obstruction and to its site in the small intestine; the nearer the obstruction is to the ileo-cæcal valve, the larger the number of fluid levels (fig. 1191). Obstruction *low* in



FIG. 1191.—Fluid levels; 'step-ladder' pattern; sub-acute intestinal obstruction by bands; patient erect.

the colon does not commonly give rise to fluid levels in the small intestine but in the case of obstruction *high* in the large intestine, this phenomenon is not unusual, because in many individuals the ileo-cæcal valve is incompetent. The commonest cause of false fluid levels is an incompletely evacuated enema.

In obstruction of the large intestine, a plain radiograph always shows a large amount of gas in the cæcum. In most cases a barium enema is contra-indicated during an attack of intestinal obstruction.

TREATMENT OF ACUTE INTESTINAL OBSTRUCTION

There are three measures for combating and overcoming the effects of intestinal obstruction. They are: (1) gastro-duodenal or, when possible, gastro-intestinal suction drainage; (2) replacement and maintenance of fluid and electrolytic balance; (3) relief of the obstruction, usually by operation.

The first two are always necessary preliminaries to the relief of obstruction by operation, and they are also the mainstays of post-operative treatment. In some cases, as will be shown, they are used exclusively.

In every case of acute intestinal obstruction the first step is to empty the stomach by a transnasal aspirating tube and to keep the stomach empty by withdrawing the contents with a syringe or by continuous suction. The second step is to correct the fluid and electrolytic balance (Chapter 6). When, on clinical examination, the cause of the obstruction is not obvious, radiographs of the abdomen are taken and the clinical and radiographic data are correlated. The main indications for early operation (as soon as the fluid and electrolytic depletion has been corrected) are:

1. Obstructed or strangulated external hernia (Chapter 43).
2. Internal intestinal strangulation.
3. Acute or acute-on-chronic obstruction of the large intestine.

The most urgent of these is intestinal strangulation. Gastro-duodenal aspiration should be continued throughout the operation, and also in most

instances the intravenous infusion, which, in cases of strangulation, should be supplemented by blood transfusion.

Relief of Obstruction by Operation.—When the cause of the obstruction lies within the abdomen but its site is doubtful, a right lower paramedian incision is employed.

When the Obstruction lies in the Small Intestine.—The hand is passed to the cæcum.



FIG. 1192.—Acute intestinal obstruction: tracing a collapsed coil to the site of obstruction.

In obstruction of the small intestine the cæcum is collapsed. The site of obstruction may be obscured by dilated coils of intestine, in which event an

unobstructed contracted coil of ileum is sought (fig. 1192) and followed upwards. This will guide the fingers to the site of obstruction which, if deeply placed, is exposed by displacing distended coils away from the site with warm, moist abdominal packs. Occasionally it is necessary to withdraw several coils of distended intestine before the site of obstruction can be displayed satisfactorily. Eviscerated coils must be kept covered by abdominal packs. The obstruction is relieved by one of the various methods described under special forms of intestinal obstruction.

Emptying the Intestine at Operation.—If there is much distension, it is wise to deflate the bowel using Savage's intestinal decompressor or, better, a plastic tube made on the same principles (fig. 1193), which is attached to the sucker. The tube is introduced into the lumen through a purse-string suture of the sero-muscular coat of the bowel at its most distended part. The bowel is progressively emptied by threading it over the instrument for its full length (fig. 1194).

If this, or a similar instrument, is not available, an ordinary sucker or a rubber catheter attached to a sucker is an adequate substitute. By these means closure of the abdominal wall is facilitated and intestinal toxins mostly eliminated. The amount of fluid removed should be taken into account in the fluid-balance chart, but in practice it is mostly gas which is aspirated.

Measures to be taken when the small Intestine is Strangulated.—If, as is frequently the case in intra-abdominal strangulation, blood-stained fluid is present

in the peritoneal cavity, the fluid should be removed by suction or mopped up as completely as possible, for it is toxic and infected. After the relief of strangulation a decision must be reached as to whether the segment that was strangulated is viable. When it is black and the peritoneal coat has lost its sheen, when the mesentery shows a lack of arterial pulsation, or thrombosis of its veins, it is non-viable, if not already gangrenous, and, if practicable, resection followed by anastomosis is carried out. In doubtful cases when the intestine is blue, purple, or dark red, the effect of wrapping it in a warm moist abdominal pack is noted. At the same time the anæsthetist administers pure oxygen for three minutes. By these means viable is differentiated from non-viable intestine thus :



FIG. 1193.—Plastic tube for intestinal decompression. (After Savage.)

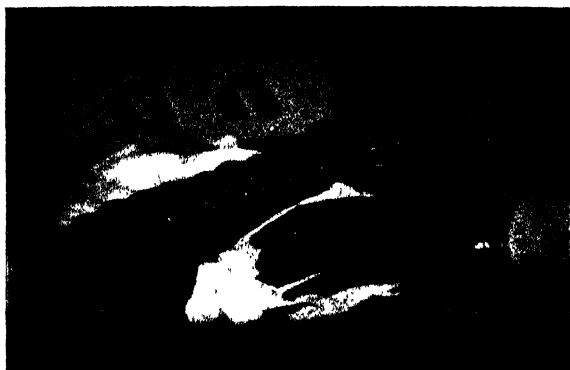


FIG. 1194.—Savage's decompressor introduced into the small intestine which is plicated over it.

<i>Intestine</i>	<i>Viable</i>	<i>Non-viable</i>
Circulation	Dark colour becomes lighter; mesentery bleeds, if pricked.	Dark colour remains; no bleeding if mesentery is pricked.
Peritoneum	Shiny.	Dull and lustreless.
Intestinal musculature	Firm. Pressure rings may or may not disappear. Peristalsis may be observed.	Flabby, thin, and friable. Pressure rings persist. No peristalsis.

Special attention should always be paid to the sites of constriction ('pressure rings') at each end of the segment, which if of doubtful viability, should be enfolded by passing sutures through the sero-muscular coats and covering the area with omentum.

Pressure rings having received attention, viable intestine is returned to the abdominal cavity and the laparotomy incision is closed. When the strangulated intestine is deemed non-viable, it is resected and the continuity of the alimentary canal restored by end-to-end anastomosis.

When the obstruction occurs in the large intestine it is usually due to a carcinoma, or occasionally, in the case of the pelvic colon, to its imitator, diverticulitis, and the obstruction is of the acute-on-chronic variety. Acute-on-chronic obstruction of the large intestine should always be treated by early operation. If the patient's condition is good, laparotomy is performed through a right or left paramedian incision, according to the site of the obstruction. If the site is unknown, a right lower paramedian incision is employed. Distension of the cæcum at once confirms that the obstruction lies in the large intestine. Palpation of the pelvic colon and, if that be collapsed, the transverse colon, will readily lead to the obstruction. When removable obstruction is present in the cæcum, ascending colon, at the hepatic flexure, or in the proximal part of the transverse colon, emergency *right hemicolectomy* (p. 922) is the correct procedure. If the growth is irremovable or the patient extremely ill, an ileo-transverse colostomy will relieve the obstruction. If the obstruction lies at the splenic flexure, in the descending colon, the pelvic colon, or in the rectum, *transverse colostomy* is performed (fig. 1195). The obstructing growth is then removed at a second operation and the colostomy closed.

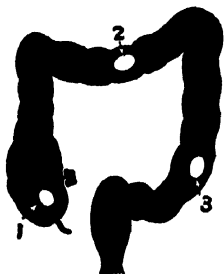


FIG. 1195.—The sites for an artificial anus in acute-on-chronic intestinal obstruction. 1. Cæcostomy. 2. Transverse colostomy. 3. Left iliac colostomy.

In very old or enfeebled patients when a carcinoma of the rectum is fixed and probably irremovable, left iliac colostomy is the best site for a permanent artificial anus.

By placing the colostomy a few inches proximal to the growth and excising it together with the growth at a second-stage operation, the number of operations and the length of hospitalisation is reduced.

Blind cæcostomy or transverse colostomy should be avoided, except in desperate cases, as the obstruction may be due to some condition other than the growth, e.g. adhesion of a loop of small bowel to the growth, in which case draining the large bowel is useless. Modern methods of fluid replacement and anaesthesia render an expeditiously performed laparotomy nearly as safe as a blind procedure on a poor-risk patient.

GASTRO-INTESTINAL SUCTION DRAINAGE

When intestinal strangulation can be ruled out and the obstruction lies in the small intestine, a period of a few hours' gastro-intestinal suction drainage is the best form of preliminary treatment.¹ Suction drainage should also be employed almost exclusively in paralytic ileus. In mechanical obstruction the combined effects of relieving distension by suction instillation of neomycin at the lowest possible level and the administration of fluid intravenously greatly improve the general condition of the patient for operation. Locally, diminution in size of the distended coils of intestine facilitates the operation and closure of the wound. Another most important consideration is that if highly toxic intestinal contents are aspirated before operation, it spares the patient the danger of absorbing this material after the obstruction has been relieved.

There are two methods of suction drainage—

1. **The Miller-Abbott Double Lumen Tube** (fig. 1196).—This has a distal balloon which is inflated as soon as it is through the pylorus and from there is propelled

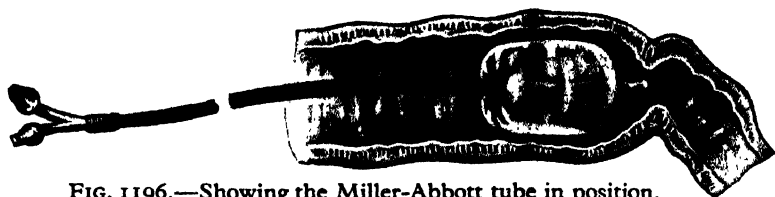


FIG. 1196.—Showing the Miller-Abbott tube in position.

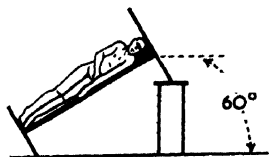
by peristalsis, thus enabling coil after coil of distended intestine to be decompressed, the onward progress of the tube being arrested only at the site of mechanical obstruction. One third of the lumen is used for inflation of the balloon which is attached to the distal extremity of the tube; two-thirds of the lumen are devoted to suction drainage viz.

The tube is 3·2 metres in length and is calibrated in centimetres. It has been largely replaced by a weighted tube as it is so difficult to get through the pylorus.

2. **A Mercury Weighted Tube.**—The exact details of its construction and method of introduction are important (Kilby). The tube can be easily put together by the surgeon. It should be of rubber and approximately 3 metres long. The bag, a finger cot containing 0·5 ml. of mercury is tied on the end and also acts as a radio-opaque marker. The tube $\frac{3}{8}$ -inch (0·5 cm.) in diameter, marked with indelible ink at 45 cm. (cardia), 60 cm. (antrum) and 75 cm. (mid duodenum). A series of holes for suction 1·5 cm. apart are placed in the distal 45 cm. of the tube. It is passed in stages each of which takes three to five minutes.

Stage 1.—The tube is swallowed until the bag is at the cardia (45 cm.).

Stage 2.—The patient lies on the left side in the head up position at 60°. The tube is slowly passed along the greater curve to the antral mark at 60 cm., and the stomach aspirated. A drink of warm water or tea (1 oz. (30 ml.)), may help the pylorus to relax and give the patient confidence to swallow more of the tube.



¹ Even when strangulation can be ruled out, not more than six or eight hours should be expended in this form of treatment. If operation is delayed over twenty-four hours, in spite of suction drainage, the mortality is nearly doubled.

gether with the whole of the small intestine, which has a narrow attachment, revolves. Broadly speaking, the clinical features are similar to, and the radiological findings are identical with, those of arrested rotation which, indeed, is present. The onset, however, is more catastrophic, and dehydration occurs more rapidly than in arrested rotation *per se*. In addition, abdominal distension is often evident.

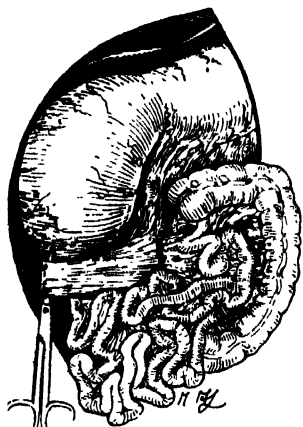


FIG. 1202.—Incomplete rotation of the cæcum and Ladd's transduodenal band compressing the duodenum. (V. Swain, F.R.C.S., London.)

Treatment.—When the abdomen is opened, only distended coils of small intestine (which may or may not be cyanotic) and the stomach are seen. The whole of the midgut must be delivered on to the surface, where the intestine is protected with warm, moist abdominal packs. Only after this step has been taken is it possible to see the volvulus (fig. 1203) which usually takes place in a clockwise direction. Untwisting is only half the operation; of equal importance is to divide the second obstructive lesion—the transduodenal band of Ladd—which is often present.



FIG. 1203.—Volvulus of the midgut.

Meconium ileus is the neonatal manifestation of mucoviscidosis (fibrocystic disease of the pancreas, p. 847). The terminal ileum becomes filled with meconium mixed with viscid mucus, notably from the pancreas, and during the latter months of foetal life this mixture becomes progressively inspissated (fig. 1204). The infant is born with intestinal obstruction. At times the coil filled with inspissated meconium can be felt as a rubbery mass. A typical radiograph shows distended small intestine, some of which is mottled. Unlike ileal atresia, there is no abrupt termination of the gas-filled intestine.

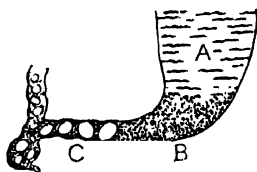


FIG. 1204.—Operative findings in meconium ileus: (a) Treacly fluid; (b) Putty-like material; (c) Meconium pellets.

Pathognomonic Test.—Into a bowl of vomitus is placed a piece of exposed X-ray film, and there it is left for half an hour. If trypsin is present, the gelatine that constitutes the sensitised coat of the film will be digested. In meconium ileus the film is merely rendered a little soft.

Treatment.—Laparotomy with the usual pre-operative preparation is essential. The only condition with which meconium ileus can be confounded is Hirschsprung's disease (p. 893) affecting the whole colon, but in the latter condition (b) and (c) of fig. 1204 are lacking. A freshly made solution of 3 per cent. hydrogen peroxide 1 part, in water 3 parts, should be available. The coil containing the putty-like material is isolated with abdominal packs, and an incision $\frac{1}{4}$ inch (1.3 cm.) is made into the antimesenteric border of the intestine. Through the opening a catheter is inserted, at first in a proximal direction, and 10 ml. of hydrogen peroxide are injected. After waiting for several minutes, an endeavour is made to milk the viscid contents through the opening. By repeating this process a number of times the obstructing material can be expressed both from above and below the opening. This accomplished, fluid meconium is aspirated with a sucker. No solution other than hydrogen peroxide has the property of loosening this extremely viscid material, which adheres to everything with which it comes in contact even more tenaciously than glue. When the operator is satisfied that the obstruction has been relieved, the opening in the intestine is sutured. The coil, mechanically cleansed, is replaced, after which the abdomen is closed. Sometimes this method fails and it then becomes necessary to resect the affected bowel including the dilated segment (A, fig. 1204), continuity being restored by end-to-back anastomosis (fig. 1201).

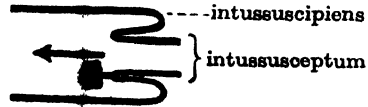
Post-operative Complications.—In the section dealing with fibrocystic disease of the pancreas (p. 847), attention has been directed to the susceptibility of infants with mucoviscidosis to pulmonary complications. Oxygen and antibiotic therapy, in addition to the usual post-operative regimen for intestinal obstruction, are therefore mandatory.

ACUTE INTUSSUSCEPTION

One portion of the gut becomes invaginated into another immediately adjacent ; almost always it is the proximal into the distal. Very rarely indeed is an intussusception retrograde.

Ætiology.—In a few cases there is some obvious cause, e.g. a polypus, a papilliferous carcinoma, a submucous lipoma (fig. 57), or an inverted Meckel's

FIG. 1205.—The mechanism of the production of an intussusception.



diverticulum (p. 899). Obviously such a protrusion invites intussusception (fig. 1205). In intussusception of infants it is generally agreed that:

1. Idiopathic intussusception occurs most often between the sixth and ninth months.
2. Between the sixth and ninth months there is a change in the infant's diet—it is weaned.
3. An idiopathic intussusception usually commences in some part of the last 2 feet (50 cm.) of the small intestine.
4. The maximum aggregation of Peyer's patches is in the lower ileum.

Theory

1. Change of diet brings about a change of intestinal flora.
2. This predisposes to inflammation of the intestinal tract.

3. Which in turn causes inflammation and swelling of Peyer's patches.

4. A swollen Peyer's patch produces an elevation protruding into the lumen of the gut comparable to one of the known causes of intussusception.

Another theory:

Intussusception often shows a seasonal incidence related to attacks of upper respiratory infection in children. In many children, after an operation for intussusception antibodies to certain viruses have been isolated and it is presumed that these cause swelling of Peyer's patches.

Pathology.—An intussusception is composed of three parts:

1. The entering, or inner, tube.
2. The returning, or middle, tube.
3. The sheath, or outer tube.

The outer tube is called the *intussusciens*. The inner and middle tubes together form the *intussusceptum*. The neck is the junction

of the entering layer with the mass. That part which advances is the apex, and the mass which constitutes the intussusception (fig. 1206) increases as it advances.

The blood supply of the inner layers of the intussusception is liable to be impaired. The onset of early gangrene is dependent upon the tightness of



FIG. 1206. — An intussusception dissected to show its constituent parts.

the invagination. Because of the great pressure exerted upon it by passing through the ileo-cæcal valve, an ileo-colic intussusception provides most examples of early gangrene.

Varieties (fig. 1207).—The following is a simple classification :

1. <i>Ileo-ileal</i> . Ileum is invaginated into ileum . . .	Approx.	8 per cent
2. <i>Ileo-cæcal</i> . The ileo-cæcal valve is the apex of the intussusception . . .	"	46 "
3. <i>Ileo-colic</i> . An ileo-ileal intussusception which has passed through the ileo-cæcal valve into the colon . . .	"	36 "
4. <i>Cæcal</i> . The caput cæci becomes invaginated . . .	"	2 "
5. <i>Colo-colic</i> . The colon is invaginated into the colon . . .	"	8 "

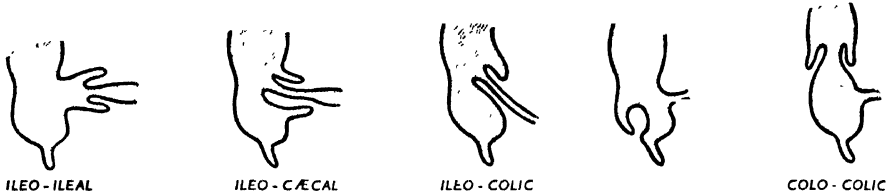


FIG. 1207.—Varieties of intussusception.

Clinical Features of Intussusception in Infants.—Usually the patient is a fine, lusty male child between six and nine months of age. The onset is sudden. The child has a paroxysm of abdominal pain, draws up his legs, and screams. He may vomit shortly after the onset of the attack, but this is not a constant occurrence; not until the intussusception has been present for about twenty-four hours is vomiting a conspicuous feature. The attacks, which last a few minutes and recur about every fifteen minutes, are accompanied by facial pallor. They become progressively more severe. In between the attacks he lies listless and looks somewhat drawn. In the early stages a normal stool is frequently passed; later blood and mucus are evacuated—the well-known ‘red-currant jelly’ stool.

On Examination.—The abdomen is not distended. The abdomen should be palpated, between the spasms, with a warm hand slipped beneath the bed-clothes, care being taken not to arouse the dozing infant. Typically a lump, is felt which may harden on palpation (fig. 1208), but if the lump is lying



FIG. 1208.—The physical signs recorded in a typical case of intussusception in an infant.

under the right or left costal margins, it may not be possible to feel it, even under an anæsthetic. There is said to be a feeling of emptiness in the right iliac fossa (*signe de Dance*). On rectal examination, if the intussusception has travelled far enough, its apex, a conical mass which is aptly likened to the cervix uteri, will be felt, especially on bimanual examination. In the large majority of cases the intussusception has not advanced far enough for

the apex to be felt per rectum, but in most cases blood-stained mucus will be found on the examining finger.

In a few instances the intussusception actually protrudes through the anus. This does not necessarily imply that the intussusception is of long standing, but rather that the patient is possessed of a long mesentery, rendering the small intestine unduly mobile.

Unrelieved, the pain becomes continuous. After twenty-four to thirty-six hours the abdomen commences to distend, and vomiting becomes copious. Absolute intestinal obstruction follows, and death from this cause, or from peritonitis secondary to the gangrene, is probable. Once in a while a natural cure occurs due to sloughing of the intussusceptum.

Radiography.—Plain films of the abdomen usually reveal increased gas shadows in the small intestine, and at times an absence of the cæcal gas shadow. Radiography following a barium enema (fig. 1209) gives positive evidence of the presence of an ileo-colic intussusception, but if the intussusception is ileo-ileal, and the ileo-cæcal valve is competent, this form of examination is negative.



FIG. 1209.—Barium enema showing ileo-colic intussusception.

Differential Diagnosis

1. *From Acute Entero-colitis.*—Unlike intussusception, diarrhoea is a leading symptom. As in intussusception, abdominal pain and vomiting often occur; likewise blood and mucus may be passed, but in entero-colitis faecal matter or bile is always present in the stools.
2. *From Purpura with Intestinal Symptoms (syn. Henoch's Purpura).*—There is likely to be the characteristic rash, which might be mistaken for flea-bites. Intussusception is a not uncommon accompaniment of this form of purpura, consequently the differential diagnosis is not of vital importance, for exploratory laparotomy must be performed in suspicious cases.
3. *From Prolapse of the Rectum.*—This can be eliminated readily. In prolapse the projecting mucosa can be felt continuous with the perianal skin. In intussusception protruding from the anus, the finger passes indefinitely into the depths of a sulcus (figs. 1291 and 1292).

Intussusception in adolescence is nearly always caused by an inverted Meckel's diverticulum (p. 899).

Intussusception in adults is most often due to a papilliferous carcinoma; consequently, the colo-colic type is frequent. Rarer causes are a papilloma or a submucous lipoma. Idiopathic intussusception is extremely uncommon, except in Egypt immediately after the Mohammedan fasting seasons (Mooro).

Preliminary Treatment.—If necessary, the general condition of the patient is improved by the administration of dextrose-saline solution. Especially in cases of more than twelve hours' duration, gastric aspiration should be carried out and continued during and after the operation.

Reduction by Hydrostatic Pressure.—In early cases, Australasian surgeons¹ in particular favour reduction by hydrostatic pressure. In the operating theatre an unlubricated catheter is passed into the rectum of the anaesthetised infant. The catheter is connected to the tubing of a reservoir filled with saline solution, and elevated to the height of 3½ feet (1 metre). The solution is allowed to run into the bowel for four minutes, while the buttocks are pressed together to prevent escape of

¹ Intussusception is common in the urban districts of Australia.

the fluid. The catheter is then removed, and the fluid is allowed to escape into a bowl. Several such injections are made. After the first injection the fluid returned is blood-stained; subsequently, if reduction of the intussusception is effected, flatus and fæcal matter are passed. If there is the *slightest* doubt as to whether the intussusception has been reduced completely, laparotomy is performed but, as a result of the hydrostatic pressure, in most instances the intussusception is so reduced in size and is so near its place of origin, that a grid-iron incision in the right iliac fossa is all that is necessary. Scandinavian surgeons favour the use of a barium enema to accomplish the reduction (fig. 1209). The more usual method of treatment is to proceed with the operation, without resorting to these preliminary measures.

Operative Reduction.—The abdomen is opened through a right lower paramedian incision. The first part of the reduction is accomplished by squeezing the lowest part of the sausage-like mass, if possible without exteriorising the bowel, and little by little the intussusception is reduced (fig. 1210). The last part of the intussusception is the most difficult to reduce and should be withdrawn and gently compressed in a warm, saline-soaked pack, to lessen the œdema. In the majority of instances

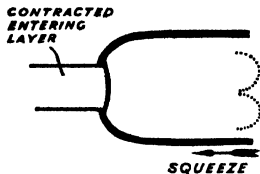


FIG. 1210.—Diagram showing the method of reducing an intussusception.

reduction is completed easily by squeezing the apex of the intussusception, as shown in fig. 1211. After reduction is completed, the terminal portion of small intestine, the caput cæci, and the appendix will be seen reddened and stiffened with œdema.

Owing to earlier diagnosis, the number of irreducible intussusceptions is decreasing; and the mortality is correspondingly lower.



FIG. 1211.—Reducing the terminal part of the intussusception. (After R. E. Gross.)

Methods of effecting Reduction in Difficult Cases

1. The little finger is inserted into the neck of the intussusception and an endeavour is made to separate adhesions between the intussusciens and intussusceptum, after which reduction is re-attempted (Cope's method).

2. The thumb and forefinger are placed as shown in fig. 1211, and gentle pressure is exerted. Gradually the pressure is increased. In this way œdema is squeezed from the region of the ileo-cæcal valve.

Resection of an Irreducible or Gangrenous Intussusception.—Previous methods consisted of exteriorising the ileum and colon by various manœuvres. With exact methods of electrolyte balance and of fluid replacement, these have been abandoned and resection with end-to-end anastomosis is the rule.

After-treatment.—When reduction is effected easily, gastric aspiration should be continued for twelve to twenty-four hours and dextrose-saline administered intravenously, or subcutaneously with hyaluronidase. In infants, nepenthe minims 1 (0.06 ml.) is given by mouth every three or four hours for three doses. On the second day the gastric tube is removed and sips of water are given. A few hours later, feeding is commenced with the

nother's milk if the infant is breast-fed. In cases where resection has been necessary, intravenous alimentation is required.

Recurrent intussusception occurs in only 2 per cent. of cases of idiopathic intussusception. If a second operation is necessary, the last few inches of the ileum should be anchored to the ascending colon by sutures in order to avoid still further recurrence.

VOLVULUS

A volvulus results from axial rotation of a portion of the alimentary tract.

(a) **Volvulus Neonatorum** (p. 937).

(b) **Volvulus of the small intestine**, other than the above, usually occurs in the lower ileum, and is favoured by the presence of an adhesion passing from the antimesenteric border of an intestinal loop (fig. 1212) to the parietes or to the female pelvic organs. In Africans, volvulus involving many feet of small intestine without causative adhesions occurs rather commonly. The consumption of a large meal of maize and vegetables seems to predispose to the condition.

Treatment is to untwist the loop, if possible. A causative adhesion should be divided and the stump of its intestinal attachment buried in the intestinal wall by a purse-string suture.

(c) **Volvulus of the cæcum** occurs occasionally, especially when the right half of the colon is lax and mobile. Volvulus of this part of the large intestine occurs nearly always in a clockwise direction. The first twist obstructs the ascending colon; if a second twist occurs, it obstructs the ileum also. The highest incidence is between twenty-five and thirty years of age. The symptoms are those of acute obstruction of the small intestine. In about 25 per cent. of cases there is a palpable tympanitic swelling not, as a rule, in the right iliac fossa, for in process of torsion the mobile cæcum moves out of the right iliac fossa into the mid-abdomen, or even to the left side. A plain radiograph shows loops of gas-filled ileum, and sometimes an especially large gas shadow which can be recognised as the cæcum. At first, the obstruction is not absolute; fæces and flatus may be expelled after an enema, but unless spontaneous untwisting occurs, the attacks of intestinal colic continue.

Treatment.—Laparotomy is performed, and in early cases it is usually possible to untwist the organ. Sometimes before untwisting can be accomplished, it is necessary to deflate the ballooned cæcum by the insertion of a needle. Untwisting should be followed by cæcostomy, which serves two purposes—it relieves distension and it fixes the organ to the abdominal wall, thereby preventing a recurrence. If the cæcum is gangrenous or its viability is not assured, right hemicolectomy (p. 922) is carried out.

(d) **Volvulus of the pelvic colon** is common in Eastern Europe, India, Scandinavia, and Peru. The predisposing causes are indicated in fig. 1213. The loop may rotate half a turn, in which event spontaneous rectification sometimes occurs. After the loop has rotated $1\frac{1}{2}$ turns the veins involved in the torsion are compressed, and the loop becomes greatly congested. If, as is sometimes the case, it rotates more than $1\frac{1}{2}$ turns, the blood supply is cut off entirely and the loop becomes gangrenous. The rotation nearly always occurs in an *anti-clockwise* direction.

FIG. 1213.—The predisposing causes of volvulus of the sigmoid colon.

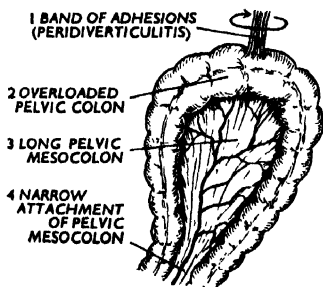


FIG. 1212.

Clinical Features.—Males are more commonly affected than females, and the sufferers are usually middle-aged or elderly. There is often a history of acute attacks of left-sided abdominal pain, probably due to a partial volvulus, that untwists itself and is followed by the passage of large quantities of flatus and fæces. As a rule the onset of volvulus of the pelvic colon is sudden and is characterised by severe abdominal pain, often coming on while the patient is straining at stool. Abdominal distension soon follows; in no other condition does extreme abdominal distension, partly due to the diffusion of CO₂ from distended veins, occur so quickly. If

the patient is examined two or three hours after the commencement of the attack the distension is mainly left-sided. In a matter of six hours the whole abdomen becomes distended. Hiccough and retching occur early; vomiting is late. Constipation is absolute, but an enema may be returned blood stained. A plain X-ray film of the abdomen shows massive distension of the colon with gas.

Treatment.—Sigmoidoscopy should be carried out and when the obstruction is reached an attempt is made to coax a soft rectal tube into the twisted gut. This will immediately deflate the gut and operation can be delayed for a few days until the patient is more fit. If deflation does not succeed, laparotomy must be performed immediately. An attempt is made to untwist the gas filled viscus; meanwhile a rectal tube is passed by a nurse and guided into position by the surgeon with his hand inside the abdomen. When the colon is deflated, a resection and end-to-end anastomosis can be carried out. Some surgeons prefer to exteriorise and resect the volvulus by the Paul-Mikulicz procedure. This method is specially valuable if there is any suggestion of impending gangrene. Redundancy of the colon makes the performance of this procedure simple (fig. 1214).

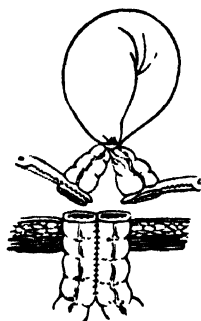


FIG. 1214.—The Paul-Mikulicz operation applied to volvulus of the pelvic colon.

OBSTRUCTION BY ADHESIONS AND BANDS

By Adhesions

Type 1.—Post-operative *fibrinous* ('bread and butter') adhesions are unlikely to produce complete intestinal obstruction unless paralytic ileus is also present. This type of obstruction commences between the third and sixth post-operative days. After some months fibrinous adhesions completely disappear.

Type 2.—Post-operative *fibrous* adhesions are strong bands and occur at a site where an abdominal organ is deficient of blood supply and becomes adherent to the omentum or parietal peritoneum in order to gain an additional blood supply. They can give rise to intestinal obstruction at any time after an abdominal operation.

Type 3.—Adherence of a loop of intestine to an inflamed intraperitoneal structure, e.g. a tuberculous mesenteric lymph node (fig. 1130). The adhesive area occurs at the site of previous granulation tissue.

Type 4 follows chemical irritation from materials such as talc glove powder entering the peritoneal cavity by accident, or sulphonamide or penicillin placed there by design.

Post-operative adhesions giving rise to intestinal obstruction usually involve the lower ileum. Operations for acute appendicitis and gynaecological procedures are the most common precursors of this condition and demand early re-operation.

Treatment.—Gastro-intestinal suction drainage (fig. 1215), combined with intravenous fluid therapy, is extremely beneficial; occasionally it is curative, but only in *Type 1*. On this account especial vigilance is necessary, for strangulation, if not present initially, is liable to develop during the course of such treatment. When, as is usually the case, operation is required, although many adhesions are often present, frequently only one of them is found to be the cause of the obstruction, in which case the condition can easily be remedied by dividing this adhesion. At other times the intestine is angulated by adherence to the parietes, to the

mesentery, to another coil of intestine (fig. 1216) or, in the female, to the uterus or adnexa. In these circumstances it is sometimes possible to free the obstructed intestine by dissection. In order to prevent recurrence the bare areas should be covered with omental grafts.

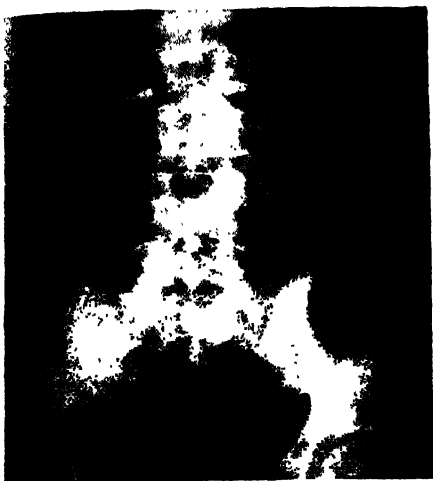


FIG. 1215.—Tip of a Miller-Abbott tube arrested in the ileum. Case of obstruction to the small intestine due to adherence of the ileum to the transverse colon after resection of a carcinoma of the transverse colon. The condition of the patient was much improved by suction drainage, after which the coil was freed by operation.

In *Type 1* the adhesions can usually be separated with a finger, thereby releasing the obstruction. When adhesions are widespread and the small intestine so matted that a definite point of obstruction cannot be found, it is best to do a Noble plication (p. 951) or the intestinal intubation procedure (p. 951).

By a Band

A band (usually one band only is culpable) is occasionally the cause of acute obstruction to the small intestine. Such a band may be:

- (a) Congenital: often an obliterated vitello-intestinal duct (p. 1026).
- (b) A string-like, frequently thin and fragile, band (fig. 1217) following previous bacterial peritonitis.



FIG. 1226.—Adhesions causing intestinal obstruction by angulation.

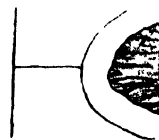
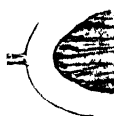


FIG. 1217.—Metamorphosis of an inflammatory peritoneal band from an adhesion.

- (c) A portion of greater omentum, adherent usually to the parietes, constitutes an obstructing band of relative stoutness (fig. 1218).

Treatment.—Provided the involved intestine is viable, division of the band and release of the obstructed loop is the simplest and one of the most satisfactory operations in surgery. Entrapment of a coil of small intestine beneath a string-like band is notorious for producing early gangrene. Even when the imprisoned intestine, after release, regains its pristine hue, if the narrow areas that have suffered direct compression by the band show any colour



FIG. 1218.—Intestinal obstruction occurring under a portion of greater omentum adherent to the parietal peritoneum.

kneading. Should the bolus be impacted so firmly that this manœuvre prove impracticable—and such is likely only in cases of obstruction by dried fruit or when stricture of the small intestine is present—enterotomy must be performed. After the obstructing material has been removed the opening in the intestine is sutured, the coecum mechanically cleansed before returning it to the abdomen, which is then closed.

Obstruction by stercolith gives rise to symptoms identical with those of obstruction by a gall-stone, for a stercolith, contrary to what might be expected, is usually formed and found in the small intestine, particularly in cases where a jejunal diverticulum or a stricture in the ileum is present. On careful dissection of a stercolith it is not unusual to find a nucleus of recognisable material, e.g. tomato skins.

Obstruction due to Worms.—An aggregation of *ascaris lumbricoides* is sometimes the cause of low small intestinal obstruction in children, usually under ten years of age, and especially those living in or near the tropics. There is debility out of proportion to that produced by the obstruction. The obturation is inclined to follow the ingestion of an anti-helminthic. If it is not known that the patient is suffering from ascariis infestation, a worm in the vomitus or the presence of eosinophilia may be the means of making a correct pre-operative diagnosis. In this form of intestinal obstruction laparotomy must be performed, and if possible the tangled mass should be kneaded along the ileum into the cæcum.

EMBOLISM AND THROMBOSIS OF THE MESENTERIC VESSELS

Arterial embolism is more common than spontaneous thrombosis, and the superior mesenteric vessels are implicated far more frequently than the inferior; the latter is often silent due to a better collateral circulation.

Embolism of the Superior Mesenteric Artery.—The embolus is derived from a vegetation on the mitral valve, the left auricular appendage (especially in mitral stenosis), an atheromatous plaque arising from the aorta, or from a pyæmic infarct of the lung which has led to thrombosis of the pulmonary vein.

Primary thrombosis of the superior mesenteric artery is the result either of arteriosclerosis or thrombo-angiitis obliterans.

Primary thrombosis of the superior mesenteric veins occurs occasionally in portal hypertension and in pyelephlebitis.

No matter whether the occlusion is arterial or venous, hæmorrhagic infarction occurs although, in the case of embolism, a short-lived pallor has been observed at early laparotomy. The intestine and its mesentery become swollen and œdematous, demarcation between infarcted and healthy intestine being gradual. Blood-stained fluid is exuded into the peritoneal cavity and the lumen of the infarcted intestine becomes filled with blood. When the main trunk of the superior mesenteric artery becomes occluded, infarction of nearly the whole of the small intestine, the cæcum,

and part of the ascending colon occurs (fig. 1223). More frequently a branch of the main vessel is implicated and the area of infarction is proportionately less.

Clinical Features.—The most important clue in early diagnosis (which is essential to success) is continuous severe mid line abdominal pain in a patient with auricular fibrillation or arteriosclerosis.

Repeated vomiting and persisting *pain* ushers in this catastrophe.

Facial pallor and a *fall in systolic pressure* closely resembles an internal hæmorrhage; indeed, the volume of blood lost to the circulation is a serious internal hæmorrhage.

Abdominal rigidity is a late sign, and is absent in early cases.

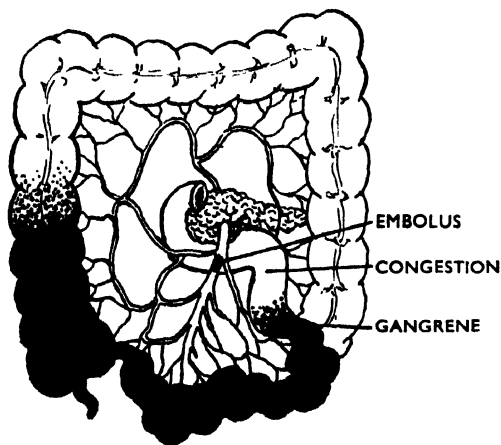


FIG. 1223.—Embolus lodged in the main trunk of the superior mesenteric artery. Showing the widespread gangrene that results.

Rebound tenderness may be present. In late cases the patient passes blood, often decomposed, per rectum. On X-ray, gas is usually completely absent from the small intestine. When it is known that the patient is suffering from one of the conditions that predispose to mesenteric thrombosis and embolism, the correct diagnosis is not difficult.

Treatment.—In the early case, when the diagnosis has been confirmed at laparotomy, superior mesenteric embolectomy should be attempted. In the later cases the affected bowel should be resected. If the surgeon is in doubt whether the infarcted bowel will survive, the abdomen should be closed and re-opened in twenty-four hours when a firm decision can be made. In all cases, blood transfusion is required and anticoagulant treatment should be commenced twelve to twenty-four hours after operation. In patients who recover, intestinal obstruction is liable to occur later from fibrosis of an area of small intestine.

Infarction of the large intestine is rare. Lodgment of an embolus in the middle colic artery should be treated by resection of the transverse colon with exteriorisation of both ends. If the patient survives and is deemed fit for a further operation, the bowel ends can be joined; in other circumstances the patient is left with a permanent colostomy.

Mesenteric vascular occlusion carries a higher mortality than any other type of intestinal obstruction. This is not surprising when one considers the type of patient prone to this disaster.

Ischaemic Colitis is the term given to describe the structural changes in the colon as a result of deprivation of blood. Such episodes are similar to those in the heart or brain and occur specially in the region of the splenic flexure whose blood supply is particularly precarious. They are classified into gangrenous (see above) stricturing and transient forms (Marston). The presenting syndrome is lower abdominal pain of abrupt onset, vomiting, fever and the passage of bright red blood per rectum.

FIG. 1224.—Ischaemic colitis—site of lesion in a woman of 67 with six months' rectal bleeding; occasional colicky pain and vomiting. Lesion resected with recovery.
(Adrian Marston.)



The age is that expected in degenerative vascular disease and at operation the length of colon has the appearance of a hæmorrhagic infarct which may require immediate resection. In the cases where no operation is performed, a stricture may appear on a barium enema as a tubular narrowing over several inches of bowel most often in the region of the splenic flexure. The differential diagnosis is from carcinoma, Crohn's disease (p. 912) and ulcerative colitis. An aortogram is usually conclusive.

ADYNAMIC OBSTRUCTION (*syn.* PARALYTIC ILEUS)

This has already been defined as a state in which the intestine fails to transmit peristaltic waves, and is due to failure in the neuromuscular mechanism—i.e. in the myenteric plexus (Auerbach) and the submucous plexus (Meissner). This results in a collection of fluid and gas in the intestine, with resulting distension, vomiting, absent or high 'tinkling' bowel sounds, and failure to pass flatus.

The following varieties are recognised:

1. *Post-operative.*—Some degree of ileus, either local or general, may follow any abdominal operation. In the absence of infection, it is rare and not serious. Indeed, it has been shown that intestinal motility and absorption commonly returns to normal in about sixteen hours—well ahead of gastric and colonic activity. However, it is important to stress that post-operative ileus may be prolonged if there is hypoproteinæmia, or latent renal failure (*vide infra*).

Jeffrey Adrian Priestley Marston, *Contemporary.* Senior Lecturer in Surgery, Middlesex Hospital Medical School, London.

Leopold Auerbach, 1828–1897. Professor of Neuropathology, Breslau.

Georg Meissner, 1829–1905. Professor of Physiology, Göttingen.

2. *Infective*.—Peritonitis gives rise to prolonged ileus, but several factors may be involved. At the outset, peristalsis ceases as a normal response to prevent dissemination, but afterwards, bacterial toxins prevent the normal activity of the nerve plexuses. When the bowel begins to recover, the early, feeble peristaltic waves may not be able to overcome the obstructive effect of the newly formed slender adhesions between loops of intestines, and so further quiescence of activity follows. In this form of ileus, there are therefore mechanical as well as neurogenic factors to be considered.

3. *Reflex*.—This form of ileus may occur following fractures of the spine or ribs, retroperitoneal hæmorrhage, or even the application of a plaster jacket.

4. *Uræmia*.—This type, with distension, vomiting, and hiccoughs is well recognised, and is seen in renal failure. It may follow prostatectomy.

5. *Hypokalæmia*.—A low serum potassium may cause ileus.

Clinical Features.—Ileus should be suspected if, forty-eight hours after laparotomy:

- (a) There has been no passage of flatus.
- (b) There is no result from an enema.
- (c) There is no return of normal bowel sounds on auscultation.

Abdominal distension becomes more marked and drum-like (tympanic); in the absence of gastric aspiration, there is effortless vomiting of large volumes of dirty fluid. There is no colic, and often no abdominal pain at all. There may be respiratory distress from the abdominal distension and the pulse-rate increases. Prolonged distension increases the risk of wound dehiscence. Radiologically, the abdomen shows distended loops of intestine with multiple fluid levels.

It is important to recognise three types of bowel sounds:

- (1) The *normal* low-pitched borborygmi each lasting about a second and occurring every twenty seconds or so.
- (2) The prolonged, rapidly recurring and noisy borborygmi of *dynamic obstruction*.
- (3) The high-pitched tinkling note 'like bells at evening pealing', which occurs every ten to thirty seconds, and is distinctive of *paralytic ileus*. It is due, not to peristalsis, but to the overflow of fluid from one distended loop to another.

Management.—The essence of this is prevention and the incidence has been greatly reduced by withholding fluids by mouth after laparotomy until normal bowel sounds return and/or the passage of flatus. Further, electrolyte balance should be achieved before, and maintained after operation. Specific treatment is directed to the cause, but there are some principles which have general application:

- (1) The primary cause must be removed.
- (2) Normal bowel activity will return if distension is removed by adequate gastro-intestinal decompression. It is probably the distension of the gut (possibly exacerbated by the air swallowing of anxiety), that causes the ileus, rather than the ileus that causes the distension. Thorough decompression is therefore essential (p. 933). If a post-operative indwelling gastric tube is used, it must not be spigoted but be kept open to permit swallowed air to be evacuated.

(3) Morphine or pethidine in adequate doses is a well proved and valuable sedative in these cases.

(4) Close attention to the fluid and electrolyte balance, especially the serum potassium and the blood urea, is essential (Chapter 6).

(5) Peristaltic stimulants have no place in treatment. The object is to rest the bowel, not to stimulate it.

Measurement of abdominal girth at the umbilicus is the only way of being certain of increase or decrease of distension. This should be done four- or six-hourly. A well-greased rectal flatus tube should be passed twice a day with the patient lying on his side in such a position that the anus is the highest point of the body. As recovery occurs, segmentation returns before peristalsis. This may cause 'wind pains', but is soon followed by the passage of flatus.

If paralytic ileus is prolonged and threatens life, and if a tube (p. 935) cannot be persuaded to pass the pylorus, the operation of intestinal intubation should be carried out (*vide infra*).

RECURRENT ATTACKS OF ACUTE INTESTINAL OBSTRUCTION

Recurrent attacks of intestinal obstruction due to adhesions are usually the aftermath of peritonitis. The difficult problem of preventing further attacks has, to a large extent, been solved by a plication operation. When a patient has had two or more attacks of intestinal obstruction due to adhesions necessitating operation for their relief, the arguments in favour of performing a plication operation or intestinal intubation are weighty.

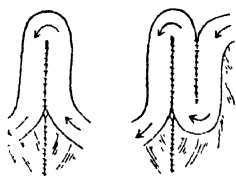


FIG. 1225.—Noble's plication operation.

Noble's plication operation (fig. 1225) is useful though time-consuming. After the abdomen has been opened by an adequate incision, all involved intestine is freed by dissection, which may include the whole of the small bowel. Adjacent coils (average length 6 to 8 inches (15 to 20 cm.)) are sutured together along their mesenteric borders. If only a portion of the small intestine is plicated, the resulting folds of mesentery must be united to prevent internal herniæ. This accomplished, the abdomen is closed.

Intestinal Intubation

Intestinal intubation is valuable especially where chronic gross distension of the small intestine is encountered in the presence of massive adhesions. Before operation, a long mercury weighted tube (p. 935) is passed into the duodenum. At operation the intestine is everywhere freed and the tube is slowly advanced till the end of the ileum is reached. It may be necessary to open the lower ileum and pull the tube through as in the Savage manoeuvre, (p. 933). The tube is left in position with suction for ten to fourteen days. By this means:

1. The intestine is completely decompressed and normal bowel diameter and peristalsis is regained and maintained.

2. The tube 'splints' the bowel so that where adhesions reform they are not so likely to cause angulation and further obstruction.

With this object in view, it is important, when returning the intestine to the abdomen, to see that the whole of the intestine is lying satisfactorily. A moderately rigid tube will then maintain this position.

CHRONIC INTESTINAL OBSTRUCTION

The various abnormalities and diseases giving rise to chronic intestinal obstruction are described in Chapters 38 and 41. There remains one condition to be considered here.

Fæcal impaction occurs principally in elderly, and often bed-ridden patients. It is all too often overlooked, in which case the patient suffers unnecessary distress. The fæces usually accumulate in the upper part of the rectum.

The symptoms are those of chronic intestinal obstruction, and attacks of spurious diarrhœa are a common accompaniment. A fæcal accumulation may form a palpable abdominal mass which can be indented. When the mass is situated in the rectum, it can be indented by the palpating finger.

Treatment.—Enemas are usually insufficient. Repeated bowel wash-outs and the use of various proprietary fæcal plasticisers (e.g. Dioctyl) by mouth or by enema, may result in disimpaction. Otherwise the anal sphincter must be dilated under general anæsthesia and the mass removed with the aid of fingers and tablespoon.

CHAPTER 40

THE VERMIFORM APPENDIX

SURGICAL ANATOMY

The vermiform appendix is present only in man, certain anthropoid apes, and the wombat.¹ Morphologically, it is the undeveloped distal end of the large cæcum found in many lower animals. It is true that many herbivores are provided with a wide-lumened cæcal diverticulum in which bacteriolytic breakdown of cellulose takes place. However, the walls of this diverticulum lack the heavy deposition of lymphoid tissue that characterises the *vermiform* appendix (Last).

The appendix varies considerably in length and circumference. The average length is between 3 and 4 inches (7·5 to 10 cm.), but variations from $\frac{1}{2}$ inch to 8 inches are not unusual: specimens of over 1 foot (30 cm.) in length have been recorded. In all large series of measurements, the appendix averages 0·5 cm. longer in the male than in the female. The lumen, which should admit a matchstick, is irregular, being encroached upon by the multiple longitudinal folds of mucous membrane.

From without inwards, the structure of the appendix is as follows. There is a peritoneal coat which completely invests it, except along the narrow line of attachment of the mesoappendix. The muscular coat resembles that of the small intestine. There is a well-developed submucous coat containing, especially in childhood and youth, a large number of lymphoid follicles. The mucous membrane resembles that of the large intestine, but there are fewer crypts of Lieberkühn.

The **mesoappendix** which springs from the lower surface of the mesentery is subject to great variations. Sometimes as much as the distal one-third of the appendix is bereft of mesoappendix. Especially in childhood, the mesoappendix is so transparent that the contained blood-vessels can be seen. In many adults it becomes laden with fat, which obscures these vessels.

The **appendicular artery**, a branch of the lower division of the ileo-colic artery, passes behind the terminal ileum to enter the mesoappendix a short distance from the base of the appendix. It then comes to lie in the free border of the mesoappendix; but for a variable distance from the tip, where the mesoappendix is lacking, the artery lies directly on the muscle wall beneath the peritoneal coat.

An **accessory appendicular artery** (fig. 1226), when present, requires independent ligation during appendicectomy.

The **appendicular vein** is a radicle of the ileo-colic vein, which drains into the portal system.

Lymphatic Vessels.—Four, six, or more lymphatic channels traverse the mesoappendix to empty into the ileo-cæcal lymph nodes.

McBurney's point lies at the junction of the lateral third with the medial two-thirds of a line joining the anterior superior iliac spine and the umbilicus (fig. 1227). McBurney's point is the classical site of greatest tenderness in appendicitis, and also a most useful point to have in mind when a grid-iron incision is made.²

Inconstancy of Position.—The vermiform appendix is the only organ in the body which has no normal position. The relative frequency of the more usual

¹ Wombat—a nocturnal, burrowing Australian marsupial.

² Appendicectomy is the most frequently performed abdominal operation; 8·4 per cent. of recruits for the Royal Air Force in 1941 bore the scar of an appendicectomy.

Raymond Jack Last, *Contemporary. Professor of Applied Anatomy, Royal College of Surgeons of England*
Johann Nathanael Lieberkühn, 1711–1766. Anatomist, Berlin. He demonstrated his anatomical Preparations in
London, and was awarded the F.R.S.
Charles McBurney, 1846–1913. Surgeon, Roosevelt Hospital, New York.

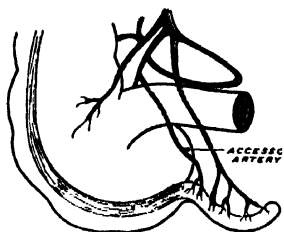


FIG. 1226.—In nearly 50 per cent. of cases there is an accessory appendicular artery, a branch of the posterior caecal. (After T. Seshachalam.)



FIG. 1227.—McBurney's point, the surface marking of the base of the appendix.

positions occupied by the organ is depicted in fig. 1228. In addition, the appendix must necessarily share in abnormalities in position of the caecum. The most frequent of these is failure of the caecum to descend, which results in the base of the appendix being situated in the right hypochondrium. Very occasionally the caecum and appendix are situated in the left iliac fossa. This is due either to abnormal rotation of the gut during embryonic life or to transposition of viscera, in which case the apex beat of the heart is usually on the right side.

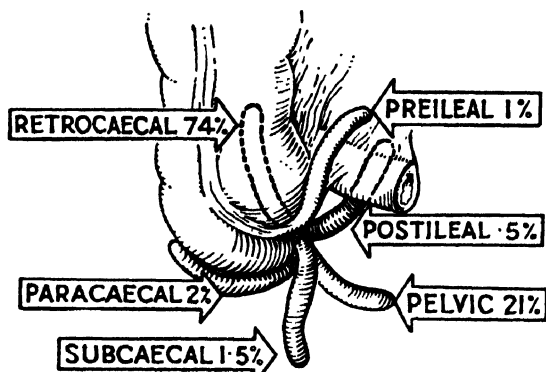


FIG. 1228.—The various positions of the appendix. (After Sir Cecil Wakeley, F.R.C.S., London.)

Locating the Appendix.—Even when the caecum is in full view it is not always an easy matter to find the appendix. The traditional method consists of following one of the tæniæ coli downwards till the appendix is reached. If the organ is still not visible and it is certain that it has not been removed, it will probably be found buried in the posterior caecal wall, and will be discovered by palpation and dissection.

CONGENITAL ABNORMALITIES

Agensis.—Once in 100,000 persons the vermiform appendix is absent; possibly some of these are congenital, but most are due to sloughing of an appendix that had intussuscepted previously (p. 973).

Duplication.—A few cases of double appendix have been reported; in some instances one of the twin appendices has been found acutely inflamed and the other uninvolved.

Left-sided Appendix.—*Situs inversus viscerum*, a congenital abnormality where there is complete transposition of thoracic and abdominal viscera, occurs once in 35,000 individuals, and is more common in males. In such cases, of course, the vermiform appendix is situated on the left as it is also in some cases of non-rotation of the mid-gut (fig. 1229).

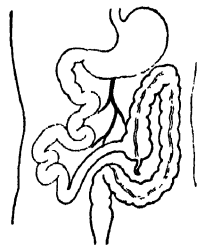


FIG. 1229.—Left-sided caecum and appendix due to incomplete intestinal rotation. (After Findlay and Humphreys.)

ACUTE APPENDICITIS

During recent years the mortality from acute appendicitis has been falling. Earlier diagnosis and appendicectomy while the inflammation is still confined

to the appendix, the cognisance of the general public that it is dangerous to take or give to a child a purgative in the presence of undiagnosed abdominal pain, more discrimination in performing immediate appendicectomy in late cases, a greater appreciation of the importance of accurate fluid and electrolytic balance, better anæsthesia, and the control of infection by antibiotics have all played a part in bringing about this improvement. Nevertheless, hospital statistics¹ show that in cases where the inflammation is no longer limited to the appendix, the mortality rate is 5 per cent. in males and 6 per cent. in females. In men over sixty-five, the mortality may be as much as 25 per cent.

All are agreed that early diagnosis with prompt appendicectomy is the goal. On the other hand, for one reason or another, numbers of patients fail to seek medical advice until a late hour. In such cases there must be no rule of thumb, and when to operate and when not to operate becomes a matter of refined judgment, helped considerably by the hour at which the aid of the surgeon is sought. Although not universally accepted, statistics indicate that after the attack has been present for some days and if there is a lump, it is wise not to operate immediately but to adopt the Ochsner-Sherren (delayed) treatment (p. 968).

Ætiology

Until the close of the nineteenth century appendicitis remained unrecognised. Unquestionably, before this time it was a comparatively rare disease, but there can be no doubt that it existed even in remote times, for an acutely inflamed, perforated appendix was found preserved in the mummy of a young royal princess of Egypt (Spencer).

The riddle of appendicitis—its actual cause and its meteoric rise from an insignificant disease to the most common serious intra-abdominal inflammatory affection of Western civilised races—has been a matter for much speculation. So far no satisfactory explanation has been forthcoming. The following ætiological factors are important, but for the most part they are purely contributory.

Race and Diet.—Appendicitis is particularly common in the highly civilised European, American, and Australasian countries, while it is rare in Asiatics, Africans, and Polynesians. Rendle Short showed that if individuals from the latter races migrate to the countries where appendicitis is common, they soon acquire the local susceptibility to the disease. Even apes in captivity appear to acquire the human liability to appendicitis. These significant facts satisfy many that the rise of appendicitis amongst the highly civilised is due to departure from a simple diet rich in cellulose to one relatively rich in meat. But this cannot be the whole explanation, for acute appendicitis occurs in lifelong vegetarians and even in babes at the breast.

Social Status.—In England, acute appendicitis is more common among the upper and middle classes than in those belonging to the so-called working class. Thus the mortality from acute appendicitis is about 20 per cent. higher in men of social classes I and II (professional and managerial workers) than it is in social class V (unskilled labourers) (Registrar-General, 1954).

Familial Susceptibility.—That there is sometimes a familial tendency to the disease cannot be disputed. This generally accepted fact can be accounted for by an hereditary abnormality in position of the organ, which pre-



FIG. 1230.—Long retro-cæcal appendix. This type tends to be familial.

¹ Statistics from the Social Medical Research Unit of the Medical Research Council, The London Hospital, 1957.

Arthur Morgan Spencer, *Contemporary. Medical Superintendent, Powick Hospital, nr. Worcester, England.*
Arthur Rendle Short, 1880–1953. *Professor of Surgery, University of Bristol.*

disposes to infection. Thus the whole family may have long retrocæcal appendicæ (fig. 1230) with comparatively poor blood supply, and many of its members fall victim to appendicitis in one form or another.

Obstruction of the Lumen of the Appendix.—When an acutely inflamed appendix has been removed, some form of obstruction to its lumen can be demonstrated in a large percentage of cases. The obstructing agent is usually a fæcolith or a stricture; exceptionally, a foreign body or a round worm or threadworms are found.



FIG. 1231.—Fæcoliths. X-ray of an appendix after removal.

Fæcoliths (fig. 1231) vary in size and have a laminated structure. They are composed of inspissated fæcal material, calcium and magnesium phosphates and carbonates, bacteria and epithelial debris; rarely, a foreign body is incorporated in the mass. The presence of a fæcolith or fæcoliths postulates some form of appendicular stasis.

Worms.—Worms (fig. 1232) and other intestinal parasites can injure the appendicular mucous membrane and occasionally block its lumen.



FIG. 1232.—An appendix filled with oxyuria vermicularis.

The Abuse of Purgatives.—It is abundantly clear that the ingestion of purgatives, particularly castor oil, by patients with 'stomach ache', and the violent peristaltic action which results, favours, and often determines, perforation of an inflamed appendix. 'Purgation means perforation' is a wise adage.

Epidemic Form.—From time to time acute appendicitis occurs as an epidemic. In this instance the infection is streptococcal, and the portal of entry almost certainly the naso-pharynx.

Bacteriology

There is no one organism mainly responsible for appendicitis. Cultures from inflamed appendices usually reveal that the infection is mixed and there is hardly a pyogenic organism which has not been isolated from such specimens. The most common organisms present are a mixture of *Esch. coli* (found in 85 per cent. of cases), enterococci (30 per cent.), non-hæmolytic streptococci, anaërobic¹ streptococci, together with *Cl. welchii* (30 per cent.) and bacteroides (p. 868). Sometimes there is synergistic action among these organisms that renders each species more pathogenic than it would be in pure culture. In most instances the infecting organisms are normal inhabitants of the lumen of the appendix.

Pathology

The menace of acute appendicitis lies in the frequency with which the peritoneal cavity is infected from this focus:

1. By perforation.
2. By transmigration of bacteria through the appendicular wall.

The greater omentum, the 'abdominal policeman', attempts to arrest the spread of peritoneal invasion, whilst violent peristalsis from ingested purgatives tends to spread it. Obviously, if the inflamed appendix lies dangling amidst coils of small intestine (fig. 1233), the threat of peritonitis is increased; should early perforation occur, diffusing peritonitis is inevitable.

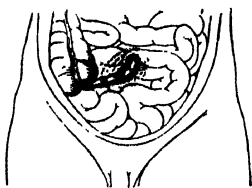


FIG. 1233.—When an inflamed appendix lies amidst coils of small intestine the risk of peritonitis is high.

¹ The foul odour of exudates connected with appendicitis with perforation is caused by anaërobic streptococci or anaërobic bacilli, and not by *Esch. coli*, as is so commonly believed.

It is of great importance to recognise two types of acute appendicitis.

(a) **Non-obstructive Acute Appendicitis.**—The inflammation usually commences in the mucous membrane; less often in the lymph follicles. Like any inflammatory process, it terminates in one of the following ways: (1) Resolution; (2) Ulceration; (3) Suppuration; (4) Fibrosis; (5) Gangrene. Non-obstructive acute appendicitis is less serious than the obstructive variety in that the mucopurulent products of inflammation have an opportunity of escaping along the lumen into the cæcum. Nevertheless, all grades of inflammation occur and once infection reaches the loose submucous tissues it progresses rapidly. The organ becomes turgid, dusky red, and hæmorrhages occur into the mucous membrane. The vascular supply of the distal part of the appendix is often in jeopardy because it is intramural and liable to occlusion by inflammation or thrombosis. This may lead to gangrene of the tip. As a rule, in non-obstructive appendicitis the inflammation progresses sufficiently slowly for protective adhesions to form, and the resulting peritonitis is localised. In many instances the infection never progresses beyond the mucous lining (i.e. catarrhal inflammation) but although the attack passes off, it is unlikely that a *status quo ante* is ever regained. Because the tip suffers most, fibrosis usually occurs therein and this is a classical finding in recurrent appendicitis (fig. 1250).

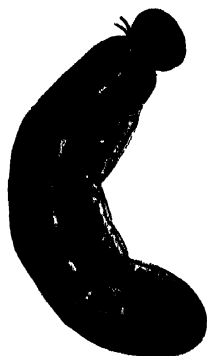


FIG. 1234.—Acute appendicitis. Perforation imminent.

(b) **Obstructive Acute Appendicitis.**—About one-third of cases of acute appendicitis belong to this group. The obstruction can be in the lumen (fæcolith, foreign body, or parasites); in the wall (almost invariably inflammatory, but exceptionally by a carcinoma of the cæcum); outside the wall (adhesions and kinking). Of these, much the most common is a fæcolith. On occasions the appendix becomes strangulated in an inguinal or femoral hernia. A kink is unlikely to cause complete obstruction.

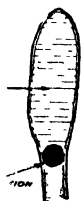


FIG. 1235.—Acute obstructive appendicitis.

In obstructive appendicitis the products of inflammation become pent up (fig. 1235) so that the inflammation proceeds more rapidly and more certainly to gangrene or perforation. Often within twelve to eighteen hours the appendix distal to the obstruction becomes gangrenous (fig. 1236). Close examination of gangrenous appendices directly after their removal shows conclusively that they usually belong to the obstructive group (Wilkie).



FIG. 1236.—Acute obstructive appendicitis with gangrene. There is a large fæcal concretion impacted in the proximal end of the lumen of the organ.

Perforation occurs most often at the site of an impacted fæcolith before protective adhesions have had time to form. The escaping purulent and gaseous contents are under high pressure and early widespread peritonitis is liable to ensue.

Clinical Features

Age Incidence.—Rare before the age of two, acute appendicitis becomes increasingly common during childhood and adolescence. The maximum incidence is between the ages of twenty and thirty; thereafter there is a gradual decline, but no age is exempt.

The patient often gives a history of similar slight attacks. The attack can commence at any time, but frequently it does so in the early hours of the morning, awakening the patient from sleep. Recent constipation is usual.

Non-obstructive Acute Appendicitis.—Typically there are three main features:

1. **Abdominal Pain which Shifts.**—Usually the first symptom is pain around the umbilicus, in the epigastrium, or it may be generalised. This is *visceral* pain and is therefore somewhat vague (p. 892). It is due to distension of the appendix. In non-obstructive cases the pain is constant but in obstructive cases it is colicky. After a few hours the pain shifts to the point where the inflamed appendix irritates the parietal peritoneum, which is very sensitive. This pain is *somatic* or *peritoneal*, accurately localised and constant.

2. **Upset of Gastric Function.**—Protective pylorospasm occurs and this may be manifested by anorexia, nausea, vomiting a brown-furred tongue, and a foul breath. Typically the vomiting is of short duration and stops as soon as the stomach is empty. In the majority of instances the patient is constipated but occasionally diarrhoea occurs, especially in the very young or when the appendix lies in the post-ileal position.

3. **Localised Tenderness at the Site of the Appendix.**—As soon as the pain has shifted, there is localised tenderness either at McBurney's point or elsewhere, as determined by the site of the appendix (fig. 1228). This tenderness may be confined to the pelvis and thus rectal examination must be done in every case of lower abdominal pain.¹ The identification of the site of the appendix is important; it will determine the operative approach. With the passage of time, accurate localisation becomes more difficult as muscular rigidity becomes evident in addition to the tenderness.

General Features.—During the first six hours there is rarely any alteration in the temperature or pulse-rate; after that time slight pyrexia² (99° to 100° F. (37.2° to 37.7° C.)) with corresponding increase in the pulse-rate to 80 or 90, is usual. In severe cases, as time passes the temperature rises to about 101° F. (38.3° C.) but seldom more, and the pulse-rate becomes correspondingly elevated. In 90 per cent. of cases the white cell count is greater than 10,000 cells per c.mm.

In Obstructive Appendicitis the sequence of clinical events occur much

¹ "If you don't put your finger in the rectum, you may put your foot in it."

² In 20 per cent. of cases the temperature is 98.6° F. (37° C.) or less.

more quickly and early diagnosis and treatment are accordingly much more urgent. The onset is abrupt and there may be severe generalised abdominal colic from the start. The temperature is often normal, but vomiting is common, so that the clinical picture mimics acute intestinal obstruction. In a few hours, however, tenderness appears in the right iliac fossa and the diagnosis will become clear.

The special features which may be encountered when the appendix occupies one of its more secluded positions are:

Retrocæcal.—Rigidity is often absent, and even on deep pressure tenderness may be lacking, the reason being that the cæcum, distended with gas, prevents the pressure exerted by the hand from reaching the inflamed structure, and gurgling may even be elicited. However, deep tenderness is often present in the loin, and rigidity of the quadratus lumborum may be in evidence. Psoas spasm, due to the inflamed appendix being in contact with that muscle, may be sufficient to cause flexion of the hip joint; to extend the joint causes abdominal pain. Hyperextension of the hip joint may induce abdominal pain when the degree of psoas spasm is insufficient to cause flexion of the hip.

Pelvic.—When the appendix lies entirely within the pelvis there is usually complete absence of abdominal rigidity, and often tenderness over McBurney's point is lacking as well. In some instances deep tenderness can be made out just above and to the right of the symphysis pubis. In either event a rectal examination reveals tenderness in the recto-vesical pouch or the pouch of Douglas, especially on the right side. Psoas spasm may also be present when the appendix is in this position; alternatively, spasm of the obturator internus is sometimes demonstrable when the hip is flexed and internally rotated. If an inflamed appendix is in contact with the obturator internus, this manoeuvre will cause pain in the hypogastrium (Zachary Cope). An inflamed appendix in contact with the bladder may cause frequency of micturition. Very occasionally early diarrhoea results from an inflamed appendix being in contact with the rectum.

Post-ileal.—Although this is rare, it accounts for some of the cases of 'missed appendix'. Here the inflamed appendix lies behind the terminal ileum. It presents the greatest difficulty in diagnosis because the pain may not shift, diarrhoea is a marked feature, retching may occur and tenderness, if any, is ill-defined, though it may be present immediately to the right of the umbilicus. As the appendix irritates the lower ileum, the patient usually passes small loose stools soon after eating or drinking. The temperature may be around 102°F. (39°C.).

Maldescended (subhepatic).—The tenderness is in the subhepatic region (p. 961). It is sometimes mistaken for acute cholecystitis.

ACUTE APPENDICITIS IN INFANTS, DURING PREGNANCY, AND IN THE ELDERLY

Acute Appendicitis in Infants.—In infants under thirty-six months of age the incidence of perforation is over 80 per cent. (Fields), and the mortality

*James Douglas, 1675-1742. Physician to Queen Caroline, wife of King George II.
Sir Zachary Cope, Contemporary. Consulting Surgeon, St. Mary's Hospital, London.
Irving A. Fields, Contemporary. Associate Professor of Surgery, Loma Linda University, Los Angeles, U.S.A.*

is considerably higher than the general mortality; indeed, when acute appendicitis occurs during the first year of life, only 50 per cent. of the patients reach their first birthday. One of the reasons for the rapid onset of diffuse peritonitis is that the greater omentum, being comparatively short and undeveloped, is unable to give much assistance in localising the infection. Even more important is the difficulty in arriving at an early diagnosis, and particularly in differentiating the condition from enteritis; also acute appendicitis can complicate enteritis. In addition, acute appendicitis may be associated with acute respiratory infection or one of the exanthemas.

Acute Appendicitis in Children.—It is rare to find a child with appendicitis who has not vomited and they usually have complete aversion to food. In addition, they do not sleep during the attack and very often bowel sounds are completely absent in the early stages.

Acute Appendicitis in Pregnancy.—Pregnancy with its shift of the vermiform appendix to the central or upper abdomen favours peritonitis; the nearer to term, the greater the danger, even in cases of appendicitis without perforation. After the sixth month acute appendicitis carries a maternal mortality of 20 per cent.—ten times greater than in the first three months (Parker). As pregnancy advances the pain becomes higher and more lateral. When it is necessary to exclude pyelonephritis, microscopical examination of specimens of urine, obtained from the right ureter by cystoscopy and catheterisation, will help to settle this important question. In doubtful cases it is best to perform early appendicectomy. The pregnant patient with acute perforated appendicitis aborts or goes into premature labour in 50 per cent of cases, while in acute non-perforated appendicitis this figure is reduced to 30 per cent.

Acute Appendicitis in the Aged.—Gangrene and perforation occur much more frequently in elderly patients, because, on account of arteriosclerosis, vascular occlusion of the appendicular artery occurs more readily. Elderly patients with lax abdominal walls or obesity may harbour a gangrenous appendix with little evidence of it. In addition, the picture may simulate sub-acute intestinal obstruction and if enemas are given, peritonitis may be spread more widely.

THE DIFFERENTIAL DIAGNOSIS OF ACUTE APPENDICITIS

Although acute appendicitis is the commonest abdominal emergency the diagnosis at times can be extremely difficult. It is wise to consider carefully possible diseases of the chest, the abdomen, the pelvis, the genito-urinary system, the central nervous system, and the spine¹. For these purposes it is helpful to visualise the body as a house (fig. 1237) and compare six parts of the house to the appropriate anatomical regions.

¹ "Distension, rigidity, vomiting, pain,
Are actors abdominal which often deign
To act on behalf of the chest, spine or brain,
Or general ills of which typhoid's the main."
From "*The Acute Abdomen in Rhyme*"
by 'Zeta' (Zachary Cope)

1. The Attic (i.e. *The Thorax*)

PNEUMONIA AND PLEURISY, especially when unilateral, give rise to referred abdominal pain. These conditions, however, are associated with an increased respiration rate and pain prevents deep inspiration. Pleural friction or altered breath-sounds occur on auscultation, and a chest X-ray may be helpful.

2. The Upper Storey (i.e. *Diaphragm to the level of the Umbilicus*)

PERFORATED PEPTIC ULCER (notably a perforated duodenal ulcer with duodenal contents passing along the paracolic gutter to the right iliac fossa).—As a rule there is a history of dyspepsia and a very sudden onset of pain which starts in the epigastrium and passes down the right side of the abdomen. In appendicitis the pain starts in the umbilical region. Rigidity and tenderness in the right iliac fossa are present in both conditions, but in perforated duodenal ulcer the rigidity is usually greater in the right hypochondrium. X-ray may show gas under the diaphragm (fig. 976, p. 752).

ACUTE CHOLECYSTITIS.—Murphy's sign and especially the radiation of pain through to the right scapula are important features (Boas' sign, p. 830). Retching and noisy vomiting are common. Jaundice may be present. In cases of difficulty in differentiating acute cholecystitis from acute appendicitis, urgent intravenous (biligrafin) cholecystography may be of value (p. 819).

CYCLICAL VOMITING.—The patient is an infant or a young child, and there is a history of previous similar attacks. Rigidity is absent and acetone is found in the urine, but acetonuria may accompany starvation.

3. The Ground Floor (i.e. *Umbilicus to the Brim of the Pelvis*)

ENTERO-COLITIS.—In this condition there is intestinal colic together with diarrhoea and vomiting but localised tenderness does not usually occur. There is often a history of a

local epidemic. Post-ileal appendicitis may mimic this condition almost completely and it is better to look and see than to wait and see.

NON-SPECIFIC MESENTERIC LYMPHADENITIS.—The patient, usually a child, is completely free from pain in between attacks, which last a few minutes. Cervical lymph nodes may be enlarged. Shifting tenderness when the child turns on to his left side, if present, is convincing evidence. This condition is a common difficulty in children and if doubt exists a laparotomy is advisable. Some hold the view that appendicectomy may help resolution of the lymph nodes.

INTESTINAL OBSTRUCTION.—Here there is persisting colicky pain around the umbilicus with vomiting first of the stomach, then of the intestinal contents. The bowel-sounds are noisy and a plain X-ray shows fluid levels.

REGIONAL ILEITIS in its acute form may be indistinguishable from acute appendicitis unless a doughy mass of inflamed ileum can be felt. A history of diarrhoea suggests regional ileitis rather than appendicitis.

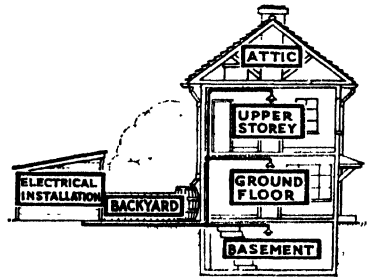


FIG. 1237.

CARCINOMA OF THE CAECUM (p. 965), when obstructed, may mimic appendicitis in patients in the carcinoma age-group.

MECKEL'S DIVERTICULITIS (p. 899).

4. The Basement (i.e. *The Pelvis*)

It is in women of the child-bearing age that pelvic disease so often mimics acute appendicitis:

SALPINGITIS.—Unlike early acute appendicitis, early salpingitis should be treated by non-operative measures. The history of a vaginal discharge, of menstrual irregularities and dysmenorrhœa, or burning pain on micturition, are all helpful differential diagnostic points. The onset of symptoms usually follows a period and the pain *starts* low down and remains there (fig. 1238). On rectal or vaginal examination the enlarged tender Fallopian tubes can be palpated. Only rarely does the cervical smear provide additional help; culture is wise and will provide information about the sensitivity of the organism. In a few cases when the condition is mainly right-sided, the differential diagnosis is so difficult that it is wiser to explore the abdomen.



FIG. 1238.—Typical distribution of abdominal tenderness in acute salpingitis.

ECTOPIC GESTATION.—It is unlikely that a *ruptured* ectopic pregnancy, with its well-defined signs of hæmoperitoneum, will be mistaken for acute appendicitis, but the same cannot be said for a right-sided tubal abortion, or still more for a right-sided unruptured tubal pregnancy. In the latter, the signs are very similar to those of acute appendicitis, except that the pain *commences* on the right side and stays there. The pain is severe and continues unabated till operation. Usually there is a history of a missed period. The cervix is softened and often severe pain is felt when it is moved. In addition, in tubal abortion, the signs of intraperitoneal bleeding usually becomes obvious after a while. The patient should be questioned specifically regarding referred pain in the shoulder, especially a quarter of an hour after the foot of the bed has been raised on blocks. When the internal bleeding has not been excessive the differential diagnosis between acute appendicitis and tubal abortion is difficult, especially when the history of a missed period is lacking. The abdomen moves well on respiration, there is deep tenderness in the iliac fossa, but seldom rigidity. It is important therefore to be able to recognise the softened cervix and the tenderness in the right fornix.

RUPTURED OVARIAN FOLLICLE (*syn.* apoplectic ovary) usually occurs half-way between periods—i.e. about the fourteenth to the sixteenth day, especially in early womanhood. The patient is usually unmarried, or recently married and childless. The signs are similar to those of very early tubal abortion, but of course the history of a missed period is absent, as also is the sign of a soft cervix. In many of these cases it is practically impossible to exclude acute appendicitis.

TUBO-OVARIAN PAIN when it presents on the right side mimics the so called 'chronic appendicitis'. It occurs in post-pubertal females aged about 14 to

5. The pain is localised to one inch (2.5 cm.) above to about one inch below the inner quarter of the inguinal ligament. The pain is described as a 'twinge' and lasts anything from a few seconds to several minutes. It may occur several times a day and ceases at the onset of pregnancy. The patient may have recently started amorous adventures and be engaged to be married, or married and childless. It has been appropriately called the pain of 'ovarian longing'. A right ureteric stone must be born in mind but the brevity of the pain and the fact that it passes on to or below the inguinal ligament is usually sufficient to exclude appendicitis.

TWISTED RIGHT OVARIAN CYST.—Here the pain is severe, is often referred to the loin, and is made worse when the patient rolls over. The pulse-rate progressively rises while the temperature remains normal. Sometimes the cyst is not easy to feel. If examination of the pelvis under anæsthesia is practised preparatory to laparotomy, a mistake will seldom be made.

5. The Backyard (i.e. *The Retroperitoneal Structures*)

RIGHT URETERIC COLIC.—In typical ureteric colic, pain commences in the loin and passes to the groin; this symptom, combined with the presence of urinary symptoms, serves to distinguish many cases from acute appendicitis. When ureteric colic is due to a stone in the right ureter there is often considerable tenderness in the right iliac fossa. Coughing causes pain in acute appendicitis, but not with a ureteric calculus. A plain X-ray may show a stone in the line of the right ureter and microscopy of the urine will reveal red cells. If an intravenous pyelogram is done, it will probably show a non-functioning kidney. However, if early acute retrocæcal appendicitis cannot be ruled out, it is safer to perform appendicectomy.

RIGHT-SIDED ACUTE PYELONEPHRITIS is accompanied and often preceded by increased frequency of micturition. It may cause difficulty in diagnosis, especially in women. The leading features are tenderness confined to the loin, fever (temperature 102° F. (39° C.)) and possibly rigors and pyuria. It presents special difficulty in pregnancy.

Acute seminal vesiculitis is sometimes confused with acute appendicitis. Often the presence of a urethral discharge is lacking, especially if antibiotic treatment has been given. Should the history of dysuria be concealed by the patient, the differentiation from pelvic appendicitis is difficult unless the clinician, when the rectum is examined, realises that it is the seminal vesical which is acutely tender.

6. The Electrical Installation (i.e. *Central Nervous System*)

Pre-herpetic pain of the right tenth and eleventh dorsal nerves is localised over the same area as that of appendicitis. It does not shift and is associated with marked hyperæsthesia. There is no intestinal upset nor rigidity. The herpetic eruption may be delayed for thirty-six to forty-eight hours.

Tabetic crises are now rare. Severe abdominal pain and vomiting usher in the crisis. Other signs of tabes confirm the diagnosis.

Spinal conditions are sometimes associated with acute abdominal pain especially in children and the elderly. These may include Pott's disease of the spine, secondary carcinomatous deposits, senile osteoporosis, and myelomatosis. The pain is essentially due to compression of nerve roots and may be precipitated by movement. There is rigidity of the lumbar spine and intestinal symptoms are absent. In a doubtful case spinal X-rays will often be a guide.

Percival Pott, 1714-1788. Surgeon, St. Bartholomew's Hospital, London.

Other conditions to be remembered are:

The abdominal crises of porphyria which may simulate appendicular colic (p. 958) are characterised by violent intestinal colic with constipation, and are liable to be precipitated by the administration of barbiturates, the symptoms being produced by areas of intestinal spasm. The urine of these patients is usually orange in colour, which is often dismissed as 'concentrated'. If the specimen of urine is left exposed to daylight for even a short space of time, it becomes amber in colour, particularly near the surface. There are several conclusive laboratory tests for porphyrinuria. A plain X-ray of the abdomen often displays short segments of intestinal spasm with related gaseous distension of the small and large intestine. In obscure cases of intestinal colic it is well to remember this condition, especially when it is associated with mental or neurological symptoms.¹

'**Diabetic abdomen**' denotes the severe abdominal pain and vomiting which occasionally precedes coma. The urine should be tested in every abdominal emergency.

PERFORATION AND GANGRENE

When perforation or gangrene occurs within twelve to twenty-four hours after the commencement of the attack, as is sometimes the case in acute appendicular obstruction, diffuse peritonitis is liable to occur. In non-obstructive appendicitis particularly, and in obstructive appendicitis when perforation or gangrene develops after a period of twenty-four hours, the resulting peritonitis often becomes localised, especially when the appendix lies in a relatively secluded portion of the peritoneal cavity.

LOCAL, DIFFUSING AND DIFFUSE PERITONITIS are discussed in Chapter 37.

THE APPENDIX MASS (*syn.* PERI-APPENDICULAR PHLEGMON)

On the third day (rarely sooner) after the commencement of an attack of acute appendicitis, a tender mass can frequently be felt in the right iliac fossa beneath some rigidity of the overlying musculature, the other quadrants of the abdomen being free from rigidity or tenderness. Alternatively, the mass is situated within the pelvis. The mass, which at this time is not yet an appendix abscess, and may never become one, is composed mainly of the greater omentum, œdematous cæcal wall, and œdematous portions of the small intestine. In its midst is a perforated or otherwise inflamed vermiform appendix. By the fourth or fifth day the mass becomes more circumscribed. As the rigidity passes off its periphery can be defined clearly and should be outlined with a skin pencil. During the ensuing days (fifth to tenth day) the swelling either becomes larger, and an appendix abscess results, or it becomes smaller, and subsides slowly as the inflammation resolves.

Appendix Abscess.—Accompanying an abscess there is variable pyrexia, but the pulse-rate is usually under 100. There is an increased leucocyte count with a relative increase of polymorphonuclear cells. To a great extent the location of the abscess is governed by the position of the appendix. Thus the commonest site of the abscess is in the lateral part of the iliac fossa (extension of retrocæcal suppuration) (fig. 1239) and the second most common is in the pelvis (fig. 1240). Notwithstanding, an abscess centred beneath McBurney's point is not so unusual as the percentages of the anatomical

¹ It is conjectured that the bouts of mental instability from which King George III (who died in 1820) suffered were due to porphyria.

positions of the appendix would indicate. This is because perforation often complicates the proximal half of an inflamed appendix.

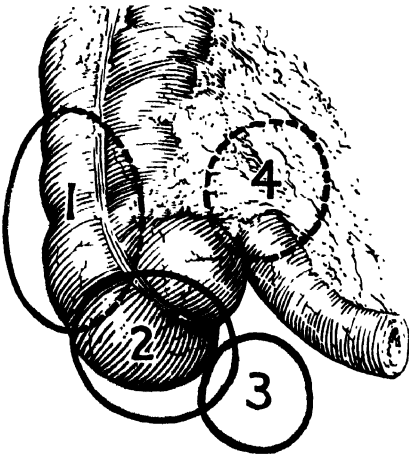


FIG. 1239.—Positions of an appendix abscess palpable from the abdomen. 1. Retrocaecal. 2. Subcaecal. 3. Retrorectus (behind the rectus abdominis muscle). 4. Post-ileal (pre-ileal occupies the same position as 4, but lies in front of the ileum).

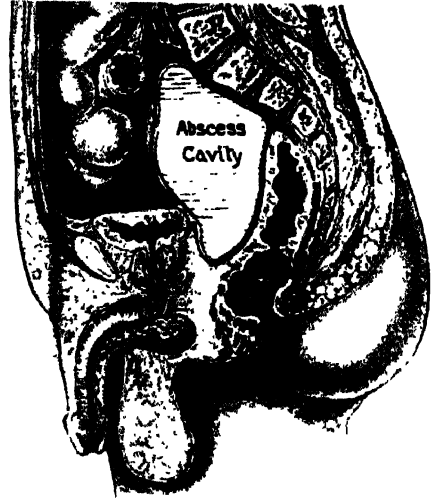


FIG. 1240.—Appendix abscess invading the pelvis. Note the relationship to the rectum.

Differential Diagnosis of an Appendix Mass

Carcinoma of the Cæcum.—Here the lump is rarely tender, it has appeared slowly and there is usually a secondary anæmia with occult blood in the stools. There is often progressive deterioration in health over months.

Crohn's Disease.—The diarrhœa, loss of weight, and a lump nearer the midline merits careful barium studies of the small bowel in order to make a firm diagnosis which may otherwise be difficult. Occult blood in the stools and a raised erythrocyte sedimentation rate may help, especially in a young adult.

Parametritis.—Here there is usually a history of recent parturition, the lump is over the medial third of the inguinal ligament and appears to be continuous with it.

Actinomycosis of the Right Iliac Fossa (p. 914).

Tuberculosis of the Cæcum (p. 913).

Twisted Ovarian Cyst (p. 963).—The essential step in diagnosis is the bimanual examination.

Iliac Lymphadenitis.—When right-sided, this condition sometimes simulates an appendix abscess. In the early stage psoas spasm is often in evidence. There is tenderness, some rigidity, and a palpable swelling above the inguinal ligament (fig. 1241). Often the inguinal lymph nodes are unaffected. Suppuration of the iliac



FIG. 1241.—Tender mass connected with suppurating deep iliac lymph nodes.

lymph nodes leads to an extraperitoneal abscess. There is usually a focus of infection, e.g. a scratch or a blister on the skin of the lower limb.

TREATMENT OF ACUTE APPENDICITIS

The treatment of acute appendicitis is appendicectomy.¹ If the diagnosis is made at an early stage in the attack, and particularly in the absence of a localised mass, all are agreed that the appendix should be removed urgently.

Appendicectomy.—When the diagnosis is certain the grid-iron incision is the best one to be employed. When the diagnosis is in doubt the lower right paramedian incision is preferable because it gives good access to the pelvic organs in the female and, if necessary, it can be readily extended to deal with a perforated duodenal ulcer upwards.

The Grid-iron² Incision.—An adequate incision, according to the age, musculature, or obesity of the patient, is made with its centre over McBurney's point, at right angles to a line joining the anterior superior iliac spine to the umbilicus. In the subcutaneous tissues an arterial twig from the superficial circumflex iliac artery usually requires ligation. The external oblique is incised in the length of the incision. The fibres of the internal oblique and transversus abdominis are separated, and after suitable retraction the peritoneum is opened. If it is found that more room is required, the sheath of the rectus muscle can be incised and the rectus muscle retracted medially. The lowest mortality following appendicectomy for acute appendicitis is associated with the grid-iron incision.



FIG. 1242. — Grid-iron incision and paramedian incision.

The paramedian incision is a vertical incision lying parallel to and $\frac{1}{2}$ to 1 inch (1.25 to 2.5 cm.) to the right of the middle line. It commences 1 inch (2.5 cm.) below the level of the umbilicus and ends just above the pubis. The anterior rectus sheath is incised in the line of the incision and the rectus muscle is retracted laterally. Branches of the inferior epigastric vessels may require ligation. The transversalis fascia and the peritoneum are incised together, the peritoneal cavity being opened through the length of the incision. The advantages of the incision have been referred to already. Its disadvantages are (a) that it gives poor access to a retrocæcal appendix (it should be possible

to diagnose retrocæcal appendicitis pre-operatively); (b) if the incision becomes infected its 'trap-door' nature harbours infection.

Rutherford Morison's incision is useful if the appendix is para- or retrocæcal and fixed. It is essentially an oblique muscle-cutting incision with its lower end over McBurney's point and extending obliquely upwards and laterally as necessary. All layers are divided in the line of the incision.

Removal of the Appendix.—It will be assumed that the abdomen has been opened by a grid-iron incision. A retractor is placed under the medial side of the peritoneum and the abdominal wall is lifted up. After removing any serous exudate with a mechanical sucker, packing is inserted into the wound on the medial side. Using a swab, the cæcum is withdrawn. A finger may be inserted into the wound to aid delivery of the appendix. Once the appendix has been delivered the cæcum is grasped by an assistant.

¹ The first surgeon to perform deliberate appendicectomy for acute appendicitis was Lawson Tait, in May 1880. The patient recovered. It is recorded that in 1715 Claudius Amyand successfully removed an acutely inflamed appendix from the hernial sac of a boy.

² Grid-iron = a frame of cross-beams to support a ship during repairs. The grid-iron incision was described first by McArthur.

James Rutherford Morison, 1853–1939. Professor of Surgery, University of Durham.
Robert Lawson Tait, 1846–1899. Surgeon, Hospital for Diseases of Women, Birmingham, England.
Claudius Amyand, 1685–1740. Surgeon, St. George's Hospital, London.
Lewis Linn McArthur, 1858–1934. Surgeon, St. Luke's Hospital, Chicago.

tissue-holding forceps (e.g. Lane's) are applied around the appendix in such a way as to encircle the organ and yet not damage it (fig. 1243). The mesoappendix is clamped in a hæmostat and severed. Sometimes only one such manœuvre frees the whole of the mesoappendix. When the mesoappendix is broad, the procedure must be repeated with a second, or rarely, a third hæmostat. The appendix, now completely freed from its mesoappendix, is crushed near its junction with the cæcum in a hæmostat, which is removed



FIG. 1243.—Showing the appendix delivered and the mesoappendix displayed.



FIG. 1244.—Appendicectomy. (Inset) Morrant Baker forceps in use.

and reapplied just distal to the crushed portion (fig. 1244). A ligature is tied around the crushed portion close to the cæcum and a purse-string suture is inserted into the cæcum about $\frac{1}{2}$ inch (1.25 cm.) from the base. This stitch passes through the muscle coat, especially picking up the tæniæ coli. It is left untied until the appendix has been amputated with a scalpel close to the hæmostat, which is still applied to it. The stump is invaginated (fig. 1245) while the purse-string suture is tied, thus burying the appendix stump. The meso-appendix is then ligated and if it curtains much fat a transfixion suture en-



FIG. 1245.—Appendicectomy. Inverting the stump of the appendix.

Methods to be Adopted in Special Circumstances.—When the cæcal wall is œdematous, the purse-string suture is in danger of cutting out. If the œdema is of limited extent, this can be overcome by inserting the purse-string suture into more healthy cæcal wall at a greater distance from the base of the appendix. Occasions may arise when, because of extensive œdema of the cæcal wall, it is better not to attempt invagination, in which case the stump of the appendix should be ligated and the cut surface covered by stitching a detached portion of greater omentum over it.

When the base of the appendix is inflamed, it should not be crushed, for fear of distributing infection by way of the lymphatics or blood-stream. It should be ligated close to the cæcal wall just tightly enough to occlude the lumen, after which the appendix is amputated and the stump invaginated.

Should the base of the appendix be gangrenous, neither crushing nor ligation must be attempted. Two stitches are placed through the cæcal wall close to the base of the gangrenous appendix, which is amputated flush with the cæcal wall, after which these stitches are tied. Further closure is effected by means of a second layer of interrupted sero-muscular sutures.

Retrograde Appendicectomy.—When the appendix is retrocæcal and adherent it is an advantage to divide the base of the organ between hæmostats. After the stump has been ligated and invaginated gentle traction on the cæcum will enable the surgeon to deliver the organ which is removed from base to tip.

Drainage of the Peritoneal Cavity.—This is usually unnecessary provided adequate peritoneal toilet has been done. If, however, there is considerable purulent fluid in the retrocæcal space or the pelvis, or if there is persistent oozing, it is wise to bring out a Penrose drain¹ through a separate stab incision.

Drainage of the Parietes.—This is indicated if there is any soiling of the wound, especially in the obese and in children.

The rule is: 'If in doubt drain and, especially, the parietes'.

THE MANAGEMENT OF AN APPENDIX MASS

If an appendix mass is present, the standard modern treatment is conservative, i.e. the *Ochsner-Sherren* regimen. This decision is based on the fact that Nature has already localised the lesion and it is foolish to disturb these barriers. Inadvertent surgery at this time is difficult, bloody, and dangerous. It may be impossible to find the appendix and, occasionally, a fæcal fistula may form. For these reasons it is wise to observe a rigid non-operative programme, but to be prepared to operate at any time should Nature fail to control the infection.

The treatment is not merely a postponement of operation; it is not a substitute for operation, but a preparation for it—essentially a surgeon's treatment, to be undertaken only in a hospital, or a correspondingly equipped nursing home. Although the treatment should be conducted on the threshold of an operating theatre, there are circumstances—for instance, in a ship at sea—where conservative treatment would be less dangerous than to attempt operation.

MANAGEMENT OF CASES BY THE 'DELAYED' TREATMENT

The history is taken, and particular note is made of the number of hours since the onset. The history begins 'ten, twenty-six, fifty-five hours ago', not 'last Thursday' or 'three days ago'. The physical signs are then recorded in diagrammatic form. The extent of the rigidity is marked by shading; the presence of a lump is drawn as near as possible to scale.

In this connection it should be noted especially that sometimes when a patient is first admitted overlying muscular rigidity renders an appendix mass indefinite, or even impalpable. In the majority of such instances, should a lump be present, if the patient is re-examined in two hours' time, lessened apprehension and the warmth of being in bed will reduce guarding of the abdominal wall sufficiently to permit the lump to be felt. This was the case in the patient whose chart is reproduced in fig. 1246.

In the absence of complications or intercurrent disease countering immediate operation, the prime requirement necessary before the delayed

¹ Penrose drain. Extremely thin (latex) rubber tubing with a gauze wick.

Charles Bingham Penrose, 1862–1925. Professor of Gynæcology, University of Pennsylvania, Philadelphia.
Albert John Ochsner, 1858–1926. Professor of Surgery, University of Illinois, Chicago, U.S.A.
James Sherren, 1872–1946. Surgeon, The London Hospital.

method of treatment can be contemplated is the presence of a localised mass or a palpable swelling.¹

TECHNIQUE OF THE 'DELAYED' TREATMENT

Charts.—As a routine, the pulse is recorded every two hours on a special chart. Temperature is relatively unimportant, and is recorded every four hours. Instructions are given to the nurse to report if the patient vomits and to save the specimen for inspection. Unless the vomitus is a small quantity of clear fluid, no time should be lost in passing and retaining a trans-nasal gastric aspiration tube, in order to keep the stomach empty.

Diet.—Water, 1 ounce (30 c.c.) hourly, may be given by mouth. Mouth-washes are given frequently. Desire for food, usually about the fourth or fifth day, is an indication that satisfactory progress is being made and that oral feeding may be started. progression to solid food can take place over

the next few days.

Intravenous fluids with fluid balance chart and daily assay of electrolytes are instituted.

Application of Local Heat.—The best form is an electrically heated pad. Failing that, the patient is given a well-covered hot-water bottle to apply to the abdomen.

Drugs.—It should be particularly noted that no morphine or its derivatives are given in border-line cases that are being watched closely for a few hours in order to observe whether the pulse-rate and other signs are tending to settle. Once it has been decided definitely to treat the patient by conservative measures, omnopon may be given. Pain, as opposed to tenderness, is very seldom complained of after the first twelve hours of the treatment.

Antibiotic therapy is, of course, employed by both schools. Penicillin, 500,000 units, and streptomycin, 0.5 G., are given intramuscularly twelve-hourly. Alternatively, by adding a total of 500 to 1,000 mg. of oxytetracycline (terramycin) to the contents of the flasks of intravenous fluid given over a period of each twelve hours, not only is the blood level of antibiotic kept constant, but the patient is spared the pain of repeated intramuscular injections. Two days after the patient is permitted to receive nourishment by mouth, the antibiotic therapy is changed to the oral administration of a tetracycline, e.g. aureomycin 0.5 G. six-hourly for an adult.

Bowels.—If the bowels are not opened naturally by the fourth or fifth day, a glycerine suppository will encourage normal evacuation. No purgatives of any kind are given until resolution is complete—that is, until the temperature and pulse have been normal for a week and pain and physical signs are absent—then liquid paraffin, or some similar preparation, is prescribed.

CRITERIA FOR STOPPING 'DELAYED' TREATMENT

(1) a rising pulse-rate, (2) vomiting or copious gastric aspirate, (3) increasing pain, and, in the later stage of the treatment, (4) diarrhoea or the passage of mucus in the stools (pelvic abscess).

A rising pulse-rate in the early stages is the most reliable single sign that it is dangerous to proceed with the delayed method. If the pulse-rate has increased, or even if it is stationary towards the end of the first twenty-four hours of expectant treatment, operation is indicated.

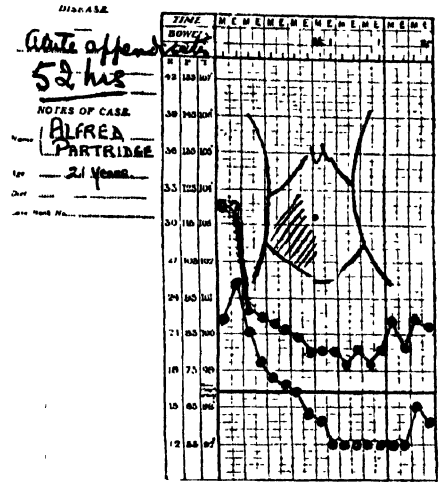


FIG. 1246.—The chart of a patient with late acute appendicitis treated by the Ochner-Sherren method.

The first feeds should be fluids only and the next few days.

¹ From time to time an appendix mass is felt for the first time when the patient has been anaesthetised. It then requires considerable strength of mind to return the patient to bed without opening the abdomen, but in some instances it is in the patient's interest so to do.

Vomiting (or copious gastric aspirate) after the first few hours should always be regarded seriously, and this by itself may be a sufficient indication to operate.

A patient undergoing delayed treatment should not complain of pain, as opposed to tenderness, after the first six hours of such treatment. If he does, there is usually something amiss, and there is a strong indication for operation.

CONTRAINDICATIONS TO THE 'DELAYED' TREATMENT

1. The diagnosis cannot be made between acute appendicitis and some other intra-abdominal catastrophe normally requiring immediate operation.
2. The signs indicate that the inflammation is still confined to the appendix.
3. When the patient is under ten years of age (poor development of the greater omentum and early free perforation of the appendix).
4. When the patient is over the age of sixty-five years more than ordinary bias is directed towards immediate operation, because of the frequency of diffusing peritonitis with minimum clinical signs. However, we have treated successfully a large number of patients in the evening of life who had an un-

mistakable localised mass by the Ochsner-Sherren regimen, and the results have not differed greatly from those of less advanced years.



FIG. 1247.—This fourteen-year-old boy had symptoms for three days. The confines of the mass are outlined. He responded to conservative treatment and returned three months later for appendicectomy.

THE OUTCOME

Under the delayed treatment about 90 per cent. of cases resolve without incident. The appendix, however, must be removed to avoid further attacks. The originators of this treatment suggested that this should be done after an interval of three months. Present-day practice, however, favours appendicectomy as soon as convenient after complete resolution of the mass.

THE TREATMENT OF APPENDIX ABSCESS

Failure of resolution of an appendix mass usually indicates that there is pus within the mass (p. 964).

Indications for Opening an Appendix Abscess.¹—(1) When the swelling is not diminishing in size after the fifth day of treatment; (2) when the temperature is swinging above 100° F. (37·8° C.) on several successive days; (3) a pelvic abscess seldom resolves. Repeated rectal examinations are required to determine when it is ready for opening into the rectum (p. 873).

Opening an Appendix Abscess.—The swelling is palpated under the anaesthetic.

A retrocaecal appendix abscess can be opened extraperitoneally. An incision from 1 to 2 inches (2·5 to 5 cm.) long, depending on the thickness of the abdominal wall, is made over the centre of the swelling, rather nearer the lateral than the medial aspect. The external oblique is incised and the fibres of the deeper muscles are divided, instead of being separated, so as to give freer exit to the contents of the abscess. When the peritoneum has been reached the extraperitoneal tissues are separated in an outward and backward direction, until the abscess cavity is entered. In cases where the abscess cavity lies at some distance from the incision, more direct drainage is afforded by a counter-incision in the flank, in which case the original incision is closed.

¹ The first recorded operation for an appendix abscess was by Thomas Henry Hancock of Charing Cross Hospital, London, in 1846.

COMPLICATIONS ARISING AFTER APPENDICECTOMY FOR ACUTE APPENDICITIS

The complications following appendicectomy for acute appendicitis vary with the degree of peritonitis that was present, and with the resistance of the patient to the infection. These complications include:

Early

Ileus (p. 949), Wound sepsis, Residual abscess (local, fig. 1249, pelvic paracolic, subphrenic, pp. 873 and 874), Intestinal obstruction from adhesions (p. 944), Fæcal fistula (p. 924), Pylephlebitis (p. 796), Post-operative thrombosis and embolism (pp. 142 and 796), Actinomycosis (p. 914), Pulmonary complications (pulmonary collapse or pneumonitis) (p. 678).



FIG. 1249.—Following appendicectomy a sinus persisted for 6 months until this faecolith appeared on the dressing. (Roger Mitchell, F.R.C.S.).

Late

Intestinal obstruction from adhesions (p. 944), Incisional hernia, Right inguinal hernia following the grid-iron incision (especially if a drain is brought through the wound) (p. 1054).

★ ★ ★

It is advisable to include the following practical problem :

After an operation for acute appendicitis the condition of the patient is unsatisfactory. The temperature is swinging and the pulse rate is elevated—signs which foretell pocketing of pus. How would you investigate the case ?

1. *Examine the wound and the abdominal wall* for an abscess.
2. *Consider the possibility of a pelvic abscess* (p. 873).
3. *Palpate the left iliac fossa* for an abscess in this situation.
4. *Examine the loin* for retrocæcal swelling and tenderness.
5. *Examine the legs*—to exclude the possibility of phlebitis.
6. *Examine the conjunctivæ* for an icteric tinge and the liver for enlargement, and enquire if the patient has had rigors—pylephlebitis.
7. *Examine the lungs*—pneumonitis or collapse.
8. *Examine the urine for organisms* (pyelonephritis).
9. *In children consider tonsillitis and otitis media.*
10. Lastly, suspect the possibility of a subdiaphragmatic abscess (p. 873).

SUBACUTE APPENDICITIS

Subacute appendicitis is but a mild form of acute appendicitis, and requires no detailed consideration.

RECURRENT APPENDICITIS

Appendicitis is notoriously recurrent. This is perhaps the commonest form of appendicitis—mild subacute attacks which are so often attributed to 'biliousness' or a 'chill on the liver'. The attacks vary in intensity, may occur every few months, and the majority of cases ultimately culminate in severe acute appendicitis. If careful histories are taken from patients with acute appendicitis, over two-thirds remember having had milder but similar attacks of pain.



FIG. 1250.—Recurrent attacks of appendicitis lead to fibrosis spreading from the tip with gradual obliteration of the lumen.

The appendix in these cases shows obliterative appendicitis (fig. 1250). In thin patients a fibrotic appendix can sometimes be rolled under the palpating fingers, and the patient is aware of the 'snick' as it slips away from the applied pressure.

CHRONIC APPENDICITIS

Chronic appendicitis, *per se*, does not exist. Patients labelled thus are usually examples of the recurrent form of the disease.

APPENDICULAR DYSPEPSIA

This is an important form of presentation of recurrent appendicitis in which the upset of gastric function predominates over the local signs. These patients are often thought to have a duodenal ulcer because of the reflex pylorospasm. They complain of attacks of anorexia, dirty tongue, flatulence, and a dyspepsia which gives little response to alkalis. They do not notice any pain in the right iliac fossa but on careful and repeated examination localised tenderness will be found. Appendicectomy should be done through a right paramedian incision so that other organs can be examined.

Radiology as an Aid to Diagnosis.—In the case of the vermiform appendix, radiology is not a great diagnostic aid. If the appendix cannot be visualised after an opaque meal, it suggests that its lumen is obstructed; certainly such an appendix should be removed. If it fills and empties, it is indicative that at least part of the organ is healthy—but as nobody can tell the length of a given appendix until the organ has been displayed, there must always be uncertainty in the radiological diagnosis of appendicitis.

LESS COMMON PATHOLOGICAL CONDITIONS OF THE APPENDIX

Mucocele of the appendix may occur when the proximal end of the lumen slowly becomes completely occluded, usually by a fibrous stricture, and the pent-up secretion remains sterile. The appendix is greatly enlarged; sometimes it contains several ounces of mucus. The symptoms produced are those of mild subacute appendicitis unless infection supervenes, when the mucocele is converted into an empyema. Rupture of a mucocele of the appendix is a cause of pseudomyxoma peritonei (p. 883).

Diverticula of the Appendix.—Diverticulosis occurs once in about 200 appendices removed by operation. It is clear that these diverticula are not merely extensions of diverticulosis of the colon; some are congenital (all coats); most are acquired (no muscularis layer). Diverticula of the appendix can occur in conjunction with a mucocele. The intramural pressure rises sufficiently to cause herniation of the mucous membrane through the muscle coat at several points. More often diverticula (fig. 1251) are not found in association with a mucocele, and often there is no demonstrable obstruction to the lumen. Usually the patient gives a history of previous recurrent attacks of appendicitis. It is probable that each diverticulum is the result of damage to the muscle coat by a previous intramural abscess which discharged into the lumen. If encountered during the course of an operation for another condition, a diverticula-bearing appendix should always be removed, because if perchance such an appendix becomes the seat of inflammation, perforation will occur very easily.



FIG. 1251.—Appendicular diverticulosis.

Intussusception of the appendix is a very rare condition, occurring most often in childhood. It can be diagnosed only at operation. The symptoms usually are not acute, and are often present for weeks or months. Untreated, the condition may pass on to an appendiculo-colic intussusception. The appendix may slough, and this accounts for most of the very rare cases in which the appendix is absent (p. 154).

The treatment is appendectomy, and if the intussusception cannot be reduced, caecotomy is necessary.

Endometriosis of the Appendix.—About 150 cases have been reported. The tumour—a miniature uterus in so far as endometrium is concerned—gives rise to monthly melæna. Occasionally the loss of blood per rectum is sufficient to endanger the patient's life.

Neoplasms of the Appendix

Carcinoid tumour (*syn.* Argentaffinoma), which arises in argentaffin tissue (Kulschitzky cells of the crypts of Lieberkühn) can occur anywhere in the gastro-intestinal tract, but most commonly it is situated in the vermiform appendix; indeed, the tumour is found once in about 300 to 400 appendices subjected to histological examination. Carcinoid tumours are distributed evenly among appendices removed from patients between the ages of sixteen and sixty years. Most of the patients are females (80 per cent.).

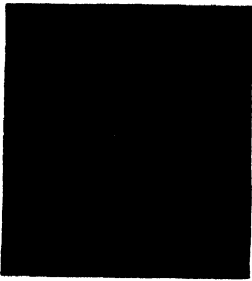


FIG. 1252. — Carcinoid tumour of the distal third of appendix. The solid bulbous tip is characteristic. (Dr. L. C. D. Hermitte, Sheffield.)

In many instances the appendix is removed because of symptoms of subacute or recurrent appendicitis. The tumour can occur in any part of the appendix, but it frequently does so in the distal third of the organ (fig. 1252). The neoplasm feels moderately hard, and on slitting up the appendix it can be seen between the intact mucosa and the peritoneum, replacing the muscular coats. There is no mistaking it, because it is of a bright yellow colour, due to contained lipid. Microscopically the

chief cells are spheroidal in type and contain granules that stain with ammoniacal silver salts. Unlike carcinoid tumours arising in other parts of the intestinal tract, in only 4 per cent. of cases does the tumour give rise to metastases, and it is most exceptional for metastases from an argentaffin carcinoma of the vermiform appendix to secrete sufficient hormone to produce the characteristic symptomatology described on p. 916. Carcinoid tumour is ten times more common than other forms of carcinoma of the vermiform appendix.

Columnar-cell Carcinoma.—This rare lesion usually presents like acute appendicitis with or without peritonitis. The occurrence of neoplasm in this organ stresses the need for routine histology in all cases of appendectomy. Pathologically the lesion resembles carcinoma of the colon. Initially it is confined to the appendix and may obstruct the lumen. Later, it spreads to the cæcum.

Colloid Carcinoma.—The whole appendix is involved and greatly enlarged. At first sight it may appear like a mucocele, but it is firm and solid. The growth is of average grade malignancy.

Treatment of Neoplasms of the Appendix.—Appendectomy suffices in a carcinoid which does not involve the cæcum. If the lesion is recognised as carcinoma at the time of operation, a right hemicolectomy is the wise procedure. The real difficulty arises when the pathologist reports carcinoma following appendectomy. Because of the frequency of metastases the correct treatment is to perform right hemicolectomy.

CHAPTER 41

THE RECTUM AND ANAL CANAL

SURGICAL ANATOMY OF RECTUM AND ANAL CANAL

The **anatomical anal canal** extends from the anal valves to the anal verge¹.

The **surgical anal canal** commences at the level where the rectum passes through the pelvic diaphragm (fig. 1253) and ends at the anal verge.

Anal Canal Musculature:

The **internal sphincter** is a thickened continuation of the circular muscle coat of the rectum. This involuntary muscle commences where the rectum passes through the pelvic diaphragm, and ends just within the anal orifice, where its lower border can be felt. The internal anal sphincter is 1 inch (2.5 cm.) long and 2 to 4 mm. thick. When exposed during life, it is pearly-white in colour, and its individual transversely placed fibres can be seen clearly. Spasm and contraction of this muscle play a major part in fissure and other anal affections.

The **longitudinal muscle** is a continuation of the longitudinal muscle coat of the rectum intermingled with fibres from the pubo-rectalis. Its fibres fan out through the lowest part of the external sphincter, to be inserted into the true anal and perianal skin, thus constituting the **corrugator cutis ani** of Ellis.

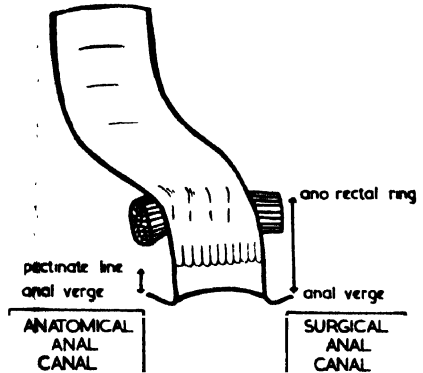


FIG. 1253.—Difference between anatomical and surgical anal canal. The dentate line is clearly shown. (E. S. R. Hughes, F.R.C.S., Melbourne.)

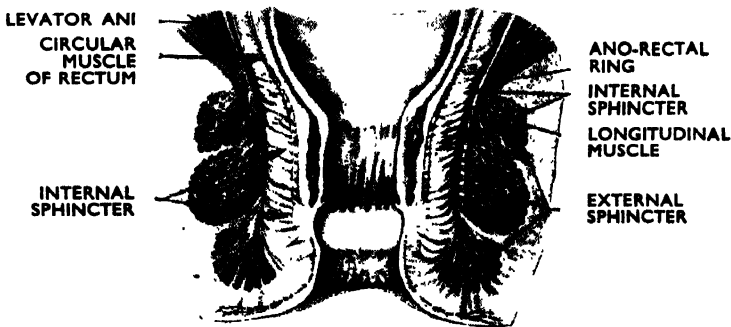


FIG. 1254.—The musculature of the anal canal. (After Naunton Morgan, F.R.C.S., London.)

The **external sphincter**, formerly subdivided into a deep, superficial, and subcutaneous portion is now considered to be one muscle (Goligher). Some of its fibres are attached posteriorly to the coccyx, while anteriorly they are inserted into the mid-perineal point in the male, whereas in the female they fuse with the sphincter vaginae. In life the external sphincter is pink in colour, and homogeneous.

The longitudinal muscles, by traversing the internal and external sphincters to each their insertions, serve to brace these sphincters.

¹ Anal verge = the external or distal boundary of the anal canal.

The Mucous Membrane.—The *pink* columnar epithelium lining the rectum extends through the ano-rectal ring into the surgical anal canal. The mucosa of the

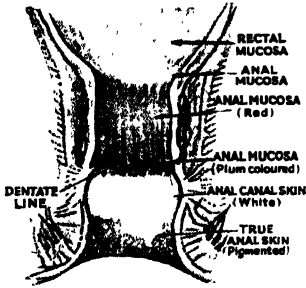


FIG. 1255.—The lining membrane of the anal canal. (After Naunton Morgan, F.R.C.S., London.)

surgical anal canal is attached loosely to the underlying structures, and covers the internal rectal plexus. Passing downwards where it clothes the series of 8 to 10 longitudinal folds known as the columns of Morgagni the mucous membrane becomes cubical and *red* in colour (fig. 1255); above the anal valves the mucous membrane becomes *plum coloured*. Just below the level of the anal valves there is an abrupt, albeit wavy, transition to squamous epithelium, which is *parchment colour*. This wavy junction constitutes the dentate line. The squamous epithelium lining the anatomical anal canal is thin and shiny, and is known as the anoderm. The anoderm passes imperceptibly into the pigmented skin of the anus. Below the dentate line the anoderm is attached very firmly indeed to deeper structures.

The dentate line is a most important landmark both morphologically and surgically. It represents (1) the site of fusion of the proctodæum and post-allantoic gut, and (2) the position of the anal membrane, remnants of which may frequently be seen as anal papillæ situated on the free margin of the anal valves. The dentate line separates :

Above

Cubical epithelium
Autonomic nerves (insensitive)
Portal venous system

Below

from squamous epithelium
from spinal nerves (very sensitive)
from systemic venous system

The anal valves of Ball are a series of transversely placed semilunar folds linking the columns of Morgagni. They lie along and actually constitute the waviness of the dentate line. They are functionless remnants of the fusion of the post-allantoic gut with the proctodæum.

The crypts of Morgagni (*syn.* anal crypts) are small pockets between the inferior extremities of the columns of Morgagni. Into several of these crypts, mostly those situated posteriorly, opens one **anal gland** by a narrow duct. This duct bifurcates, and the branches pass outward to enter the internal sphincter muscle, where often there is situated an ampulla (fig. 1256). Issuing from this ampulla there are three to six tubular sub-branches that extend into the inter-muscular connective tissue, where they end blindly. In some lower animals these glands secrete an odori-

FIG. 1256.—Anal gland with duct opening into a crypt of Morgagni.



FIG. 1257.—Houston's valves as seen through a sigmoidoscope.

ferous substance during the rutting season; in man their function, if any, is obscure. Some of their cells have been shown to give a positive staining reaction for mucin, but as the lining epithelium is mainly cubical, the mucus-secreting propensity of the anal glands must be extremely small. Infection of an anal gland can give rise to an abscess, and in the opinion of a number of surgeons, infection of an anal gland is the most common cause of ano-rectal abscesses and fistulae.

The Rectum¹.—The rectum extends from the third sacral vertebra to the ano-rectal ring. It describes three lateral curves, two concave to the left (hence the *left lateral* position for sigmoidoscopy) and one concave to the right. The relative shortness of the longitudinal muscle coat forms the **valves of Houston** that are so much in evidence in sigmoidoscopy (fig. 1257).

¹ The word rectum means 'straight'. It is straight only in infants and the elderly.

The **ano-rectal ring** marks the junction between the rectum and the anal canal. It is formed by the fusion of the pubo-rectalis muscle (fig. 1258), external sphincter, joined longitudinal muscle, and internal sphincter. The ano-rectal ring can be easily felt digitally, especially on its posterior and lateral aspects. Division of the ano-rectal ring results in permanent incontinence of faeces.

THE BLOOD SUPPLY OF RECTUM AND ANAL CANAL

The **superior rectal artery** (fig. 1259) is the direct continuation of the inferior mesenteric artery and constitutes the chief arterial supply to the rectum. Opposite the third sacral vertebra the artery divides into a right and a left branch. About half-way down the rectum the right branch subdivides into an anterior and posterior branch. The terminal branches run straight downwards, each in a column of Morgagni.

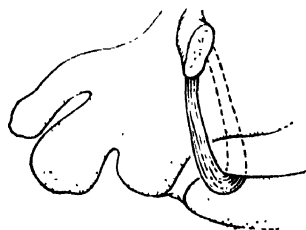


FIG. 1258.—The disposition of the pubo-rectalis muscle.

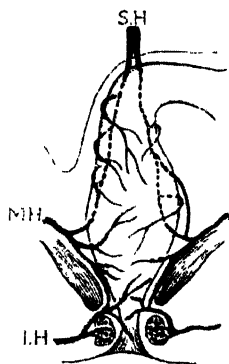


FIG. 1259.—The arterial supply of the ano-rectum. S.H., superior rectal artery; M.H., middle rectal artery; I.H., inferior rectal artery.

The **middle rectal artery** arises on each side from the internal iliac artery and passes in close proximity to the lateral ligament of the rectum to supply the muscle coat and mucosa of the mid-rectum. Often it is a comparatively small vessel. After division of the inferior mesenteric artery, in the operation of anterior resection, the middle and inferior rectal arteries can maintain an adequate blood supply as high as the recto-sigmoid junction.

The **inferior rectal artery** arises on each side as a branch of the internal pudendal artery, as this artery enters Alcock's canal. Crossing the upper part of the ischio-rectal fossa, it breaks up into branches which supply the anal sphincters, anal canal, and the skin of the anal margin.

The **internal rectal venous plexus** lies in the loose submucosa of the anal canal and extends from the level of the dentate line to that of the ano-rectal ring. The plexus drains into about six collecting veins which are situated in the submucosa of the rectum. About half-way up the rectum these branches pass through the rectal wall, and having reached the outside of the rectum, they unite to form the **superior rectal vein**, an important tributary of the portal vein. The **middle rectal veins**

are small and drain into the internal iliac veins.

The **external rectal venous plexus** lies under the skin of the anal canal below the dentate line and beneath the skin of the anal margin. Communicating veins pass from the external rectal plexus to the internal rectal plexus beneath the anoderm. The lower part of the external rectal plexus drains into the internal pudendal veins and thence into the internal iliac veins, thus providing a link between the portal and systemic venous systems.

LYMPHATIC DRAINAGE OF RECTUM AND ANAL CANAL

The lymphatics of the mucocutaneous lining intercommunicate freely with those of the muscle coats. This free anastomosis accounts for the occasional spread of malignant cells of the rectum into the inguinal lymph nodes. There are, however, three main sets of lymph nodes:

1. **Superior Rectal Lymph Nodes.**—The lymph vessels and nodes keep close to the superior rectal vessels. A special group lie just above the levator ani and close to the rectal wall in the region of the ampulla—they are the pararectal lymph nodes of Gerota. There are larger lymph nodes at the bifurcation of the superior rectal artery and so on up to the origin of the inferior mesenteric artery from the aorta.

2. **Middle Rectal Lymph Nodes.**—Along the lateral ligaments of the rectum lie lymph vessels and nodes close to the middle rectal vessels. From here they pass to the

Benjamin Alcock published the details of his canal in 1836. He was dismissed from his post as Professor of Anatomy at Cork in 1855 (for breach of Anatomy Acts), and disappeared in America.
Dimitrie Gerota, 1867–1939. Professor of Surgery, Bucharest, Rumania.

lymph nodes around the internal iliac vessels, which are frequently involved in malignant lesions so that wide excision of the lateral ligaments is indicated.

3. Inguinal Lymph Nodes.—The anus and lower portion of the anal canal are drained by lymphatics which pass up to the inguinal lymph nodes (fig. 1260).

There is seldom, if ever, metastasis along the lateral or inferior lymphatic pathways from a carcinoma situated *above* the ampulla of the rectum. Hence, in general it can be stated that the higher the growth the more confined are its metastases. This justifies restorative resection in cases of carcinoma high in the rectum.

SYMPTOMS OF ANO-RECTAL DISEASE

In every case, careful enquiry should be made for pain, prolapse, pruritus, bleeding, discharge, and alteration in bowel habit.

EXAMINATION OF THE RECTUM

Rectal examination should be conducted systematically as follows:

Inspection, e.g. prolapsed hæmorrhoids, sentinel pile, fistula, pruritus ani.

Digital examination.—Intraluminal, e.g. polyp, fæcal impaction.

Intramural, e.g. carcinoma.

Extramural $\left\{ \begin{array}{l} \text{Male, e.g. prostate.} \\ \text{Female, e.g. contents of pouch of Douglas.} \end{array} \right.$

Discharge.—On wiping the fingerstall mucus, pus, or blood may be seen.

Examination with a proctoscope (fig. 1261) is of great importance and value. The key to good visualisation is to examine the patient in the knee-elbow position (fig. 1262). This permits air to distend the rectum and causes the pelvic

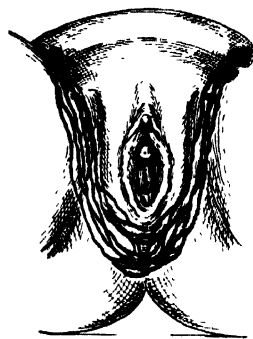


FIG. 1260.—Lymphatic trunks from the anus and the lower part of the anal canal draining into the inguinal lymph nodes. (After M. P. C. Sappey.)

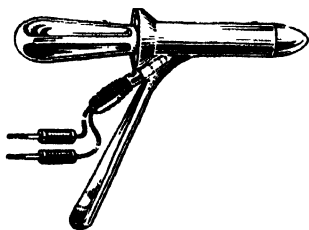


FIG. 1261.—An illuminated proctoscope.

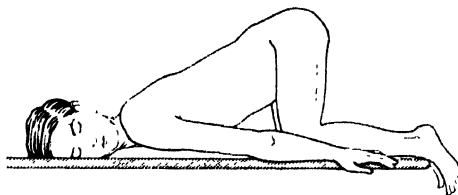


FIG. 1262.—Knee-elbow position for proctoscopy.

colon to drop away from the proctoscope so that a good view can be obtained. If the recto-sigmoid junction cannot be seen, a sigmoidoscopy must be done.

EMBRYOLOGY

Early in embryonic life there is a common chamber—the cloaca—into which open the hind gut and the allantois. The cloaca becomes separated into the bladder and post-allantoic gut (rectum) by the down-growth of a septum (fig. 1263). About this time an epiblastic bud, the proctodæum, grows in towards the rectum. Normally fusion between these two structures occurs during the third month of intrauterine life.

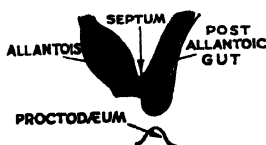


FIG. 1263.

CONGENITAL ABNORMALITIES

Imperforate Anus¹.—One infant in 4,500 is born with an imperforate anus, or with imperfect fusion of the post-allantoic gut with the proctodæum. The condition is divided into two main groups—the high and the low, depending on whether the termination of the bowel is above or below the pelvic floor. The low varieties are easy to diagnose, simple to treat, and the outlook is good. The high varieties often have a fistula into the urinary tract together with a deficient pelvic floor and the prognosis is not good.

Low Abnormalities (fig. 1264).—(1) The *covered anus*: the underlying anal canal is covered by a bar of skin with a track running forward to the perineal raphe. The track should be opened with scissors, followed by routine dilatation of the anus. (2) The *ectopic anus*: the anus is situated anteriorly and may open in the perineum in boys or more commonly in the vulva in girls, or

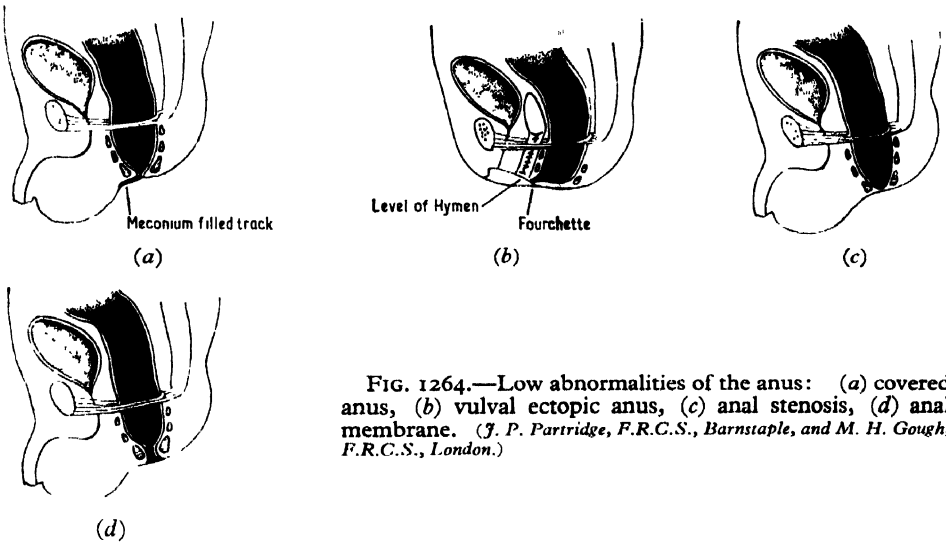


FIG. 1264.—Low abnormalities of the anus: (a) covered anus, (b) vulval ectopic anus, (c) anal stenosis, (d) anal membrane. (J. P. Partridge, F.R.C.S., Barnstable, and M. H. Gough, F.R.C.S., London.)

rarely into the vagina. A plastic 'cut-back' operation is required. (3) The *stenosed anus*: the anus is microscopic but careful examination usually reveals a minute opening which responds to regular dilatation. (4) *Membranous stenosis*: here the anus is normally sited but is covered with a thin membrane which bulges with retained meconium. It is rare, and an incision will cure the condition.

High Abnormalities (fig. 1265).—(1) *Ano-rectal Agenesis*.—A blind rectal pouch lies just above the pelvic floor—its anterior aspect in the male is attached to the bladder and often there is a recto-vesical fistula manifested by the passage of gas or meconium in the urine. In the female the fistula is usually into the posterior fornix. (2) *Rectal Atresia*.—The anal canal is normal but ends blindly at the level of the pelvic floor. The bowel also ends blindly above the pelvic floor without a fistulous opening. This anomaly is rare but must be treated by mobilisation of the rectum and excision of the stricture. After

¹ The term imperforate anus is used as a well-recognised description. Strictly it should be 'agenesis' and 'atresia' of the rectum and anus.

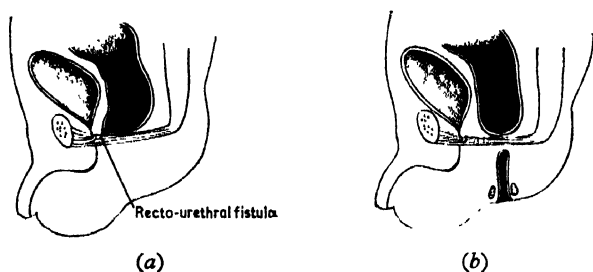


FIG. 1265.—High congenital anomalies: (a) ano-rectal agenesis with recto-urethral fistula, (b) rectal atresia. (J. P. Partridge, F.R.C. Barnstaple, and M. H. Gould F.R.C.S., London.)

that, end-to-end anastomosis of the anus and rectum must be attempted. More conservative measures are followed by an intractable stricture. (5) *Cloaca*.—This occurs only in females and here the bowel, urinary and genital tracts all open into a common wide cavity (fig. 1263). Commonly there are severe malformations of the area associated with other developmental abnormalities elsewhere.

Clinical Management.—As congenital abnormalities are frequently multiple, very careful general examination of the baby must be made to exclude any other anomalies. It is urgent and important to determine whether the abnormality is high or low and an X-ray will help.

X-ray Examination.—Six hours after birth sufficient air may have collected in the large intestine to cast an X-ray shadow. With a metal button or a coin strapped to the site of the anus, or a metal bougie inserted into the blind anal canal, the infant is held upside down for three to four minutes and radiographed in the inverted position (fig. 1266). The gas in the rectum will rise to the top and indicate the distance between the site of the metal indicator and the blind end of the rectum. If the distance is over 1 inch (2.5 cm.), the abnormality is 'high'. This method, though useful, is sometimes vitiated by a plug of meconium in the rectum causing an apparent gap far in excess of that actually present. It may be necessary to wait till the baby is twenty-four hours old before rectal gas appears.



FIG. 1266.—X-ray of neonate while held upside down to show gas in rectum. Anal dimple marked by piece of shot. (Graham Arth, Bristol.)

Where a 'high' lesion is suspected an effort must be made to obtain a specimen of urine—the presence of *proteus* or *pyocyaneus* usually signifies that a fistula is present. An excretory radiograph is recommended by some, even though there is a definite radiation risk. There may be gas in the bladder. A diagnostic perineal exploration is usually unwise—it may prejudice the chances of further surgery.

Treatment.—In the *low* abnormalities this is usually simple and has been outlined when each condition was described above. The *high* abnormalities present a very difficult problem and each case must be considered on its merits. On the whole, newborn babies stand surgery very well, provided compatible blood is available and a clear airway is maintained post-operatively and inhalation of vomit prevented by naso-gastric suction. The presence of

other congenital abnormalities is also a most important factor to consider. The possibilities are:

1. Laparotomy, division of recto-urethral fistula, and transverse colostomy. A rectal 'pull-through' operation can be done later.
2. Laparotomy, division of fistula, and 'pull-through' operation in one stage.
3. Division of the fistula and rectal 'pull-down' operation through the (a) perineal or (b) sacral route (this method is now rarely used).
4. Colostomy only (for the cloacal variety).

For the 'pull-through' operation the lower bowel is mobilised, and a new passage is created through the pelvic floor by passing a pair of curved forceps through it, keeping close to the urethra, to the site of the future anus. This is dilated by Hegar's dilators so that the bowel can be pulled down and its mucosa stitched to the skin of the newly formed anus. (For details the reader is referred to the standard textbooks of operative surgery.)

In general, daily dilatation will be required for at least three months and it may be necessary for years.

In a high percentage of cases, imperforate anus is associated with other congenital abnormalities, especially of the urinary organs, and nearly half the deaths in cases of imperforate anus are due to other malformations.

Sacro-coccygeal teratoma, although rare, is among the most common of the large tumours seen during the first three months of life. The frequency of the pre-coccygeal region for the development of a teratoma is explained by the fact that this area is the site of the 'primitive knot', a group of totipotent cells that retain their totipotentiality longer than any others save the sex anlage. Females are more often affected than males.

The tumour, which arises between the sacrum and the rectum, is firmly attached to the coccyx, and occasionally to the last piece of the sacrum. At the time of birth some of these tumours are huge, and in 20 per cent. of cases the infant is still-born. In most instances the tumour is large (fig. 1267), but in a few it is small enough to pass unnoticed until it enlarges or a complication ensues. It is this variety that is prone to become malignant, usually at about ten months of age.

Treatment.—Removal soon after birth; delay is liable to result in fatal ulceration, infection, rectal or urinary obstruction, or a malignant change.

Operation.—Excision is undertaken through a longitudinal elliptical incision, the coccygeal attachment being left until the last. The coccyx must always be excised; occasionally the last piece of the sacrum must be removed also. There may be a fistula between the tumour and the rectum but as a rule this is small, and can be closed safely without performing a colostomy. The dead space in the pelvis is drained, the skin is united, and a pressure dressing applied.

When the operation is undertaken soon after birth, the prognosis is good.

Post-anal Dermoid.—The space in front of the lower part of the sacrum and coccyx is occupied by a soft, cystic swelling—a post-anal dermoid cyst—which is regarded as a simple form of teratoma. Hidden in the hollow of the sacrum, it is unlikely to be discovered unless a sinus communicating with the exterior is present, or develops as a result of inflammation. Such a cyst usually remains symptomless until adult life, when it is prone to become infected. Exceptionally, by its very size, it gives rise to difficulty in defæcation. The cyst is easily palpable on rectal examination.

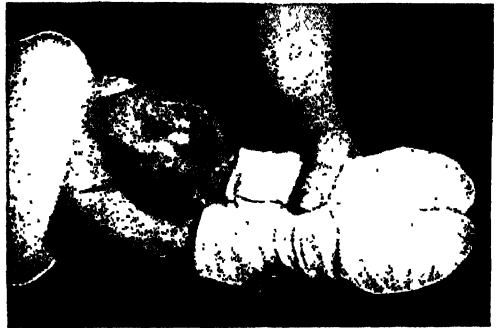


FIG. 1267.—Congenital sacro-coccygeal teratoma. The tumour was excised successfully.

Differential Diagnosis.—Especially in a child, an anterior sacral meningocele must be excluded. The latter enlarges when the child cries, and is frequently associated with paralysis of the lower limbs, and incontinence. When a discharging sinus is present, a post-anal dermoid will probably be mistaken for a pilonidal sinus unless pressure over the sacro-coccygeal region with a finger in the rectum causes a flow of sebaceous material, and/or injection of lipiodol and radiography reveals a bottle-necked cyst in front of the coccyx (fig. 1268).



FIG. 1268. — Post-anal dermoid cyst with sinus. Injected with lipiodol. Sinus and cyst excised.

Treatment is complete excision of the cyst and sinus if present. In the case of large cyst it is necessary to remove the coccyx in order to gain access.

Post-anal Dimple (*syn.* Fovea Coccygea).—A dimple, sometimes amounting to a short, blind pit, in the skin beneath the tip of the coccyx is noticed from time to time in the course of a clinical examination; Klass observed it in 9 per cent. of a large number of recruits. A dimple in this situation has the same significance as a dimple at other sites; it represents nothing more than a local fixation of skin by collagen fibres

to underlying structures. Such a dimple is a possible starting-point of a pilonidal sinus.

PILONIDAL SINUS

Ætiology.—The army of supporters of the congenital theory of the origin of pilonidal sinus has become reduced to a corporal's guard.

That, in rare instances, a sinus in the ano-coccygeal area is congenital must be allowed, but in these cases of proven congenital origin the sinus is not necessarily pilonidal. It could be (a) a sinus connected with a post-anal dermoid, referred to above, or (b) a sinus resulting from a persistent caudal remnant of the original neural canal. The latter occurs in the sacral rather than the coccygeal region, and is definitely connected with the spinal theca. On this account, meningitis from an extradural abscess may occur in a child.

The reasons which support the acquired theory of origin of pilonidal sinus can be summarised thus:

1. Interdigital pilonidal sinus is an occupational disease of men's hair-dressers, the hair within the interdigital cleft or clefts being the customers'. Also pilonidal sinuses of the axilla and umbilicus have been reported.
2. The age incidence of the appearance of pilonidal sinus (82 per cent. occur between the ages of twenty and twenty-nine years) is at variance with the age of onset of congenital lesions.
3. Hair follicles have never been demonstrated in the walls of the sinus.
4. The hairs projecting from the sinus are dead hairs, with their pointed ends directed towards the blind end of the sinus.

The mode of origin of a pilonidal sinus is now believed to be as follows:

On sitting, the buttocks take the weight of the body, and move independently, or together. Hairs broken off by friction against clothing, and shed short hairs, whether they originate from the nape of the neck, back, or buttocks, tend to collect in the cleft of the nates and/or a post-anal dimple. Furthermore, it is suggested that the use of toilet paper may contribute to hair entangled in fecal matter being swept into the cleft; pilonidal sinus is extremely rare in those races that employ ablution after defæcation. By reason of the shearing action of the buttocks, which is increased by sitting on a hard

¹ Appertaining to a nest of hair. (Latin—*pilus* = hair, *nidus* = nest.)

eat, and especially by vibration of a vehicle, loose hair travels down the intergluteal furrow, to penetrate the skin or the open mouth of a sudoriferous gland, such glands being more active in early manhood. It is not yet clear whether the initial entry of hairs through the skin is a primary event, or follows the softening of the skin due to pustular or other forms of dermatitis. Once a sinus has formed, intermittent negative pressure of the area may suck other loose hairs into the pit. So common was pilonidal sinus among jeep¹ riders in the 1939-45 war that it became known as 'jeep bottom'.

Pathology.—The sinus extends into the subcutaneous planes as a bulbous diverticulum. Branching side channels are not infrequent. A stratified squamous epithelial lining, of varying degrees of integrity, is found in about half the cases. Hair shafts are found either (a) lying loose in the sinus, (b) embedded in granulation tissue, or (c) deep in mature scar tissue in three-quarters of the cases. Foreign body giant cells are common.

Clinical Features.—There is a chronic or recurring sinus in the middle line about the level of the first piece of the coccyx (fig. 1269). Typically a tuft of hairs projects from its mouth. The discharge from the sinus or sinuses is often blood-stained, contains foul sebum, and sometimes hairs.

As has been indicated already, symptoms usually commence during the third decade; patients presenting later in life nearly always give a history dating back to this period.



FIG. 1269.—Pilonidal sinus. (R. S. Lawrie, F.R.C.S., London.)

Males with this condition outnumber females by four to one, the females being on an average three years younger than the males; this corresponds to the earlier maturation of the female. The condition rarely occurs in blondes; many of the patients are exceptionally hairy and are usually obese. In spite of the preponderance in dark-haired persons, whose hair is stiffer than the silky blonde (Oldham), the condition is practically confined to white races. The complaint is of a discharge, pain, or a tender swelling at the bottom of the spine. Even at the height

of an attack of inflammation the constitutional symptoms are slight. Often there is a history of repeated abscesses in the region that have discharged spontaneously or have been incised. The primary sinus may have one, or as many as six openings, all of which are strictly in the middle line between the level of the sacro-coccygeal joint and the tip of the coccyx. Unlike a fistula-in-ano, the sinus passes upwards and forwards towards the sacrum. It does not reach bone, but ends blindly near the bone. When an abscess forms (fig. 1270), it may discharge through a primary sinus; more frequently it points and bursts, or is incised to one side of the middle line, thus forming a secondary sinus.



FIG. 1270.—Infected pilonidal sinus with secondary abscess to the left of the middle line. (W. B. Gabriel, F.R.C.S., London.)

¹ Jeep—U.S. Army reconnaissance motor vehicle.

Conservative Treatment.—Patients reporting for the first time with mild symptoms can sometimes be cured by conservative measures, which consist of frequent washing of the parts with a detergent and water, and applying equal parts of witch hazel (*Liq. Ext. Hamamelis*) and alcohol. Long sitting, e.g. driving a car, avoided if possible. These measures, tried on a large scale in the U.S. Army, proved tolerably successful—more successful than similar attempts in civil life, because the sufferer could be relegated to duties that were unlikely to aggravate the condition.

Treatment of an Acute Exacerbation.—If rest, sitz baths, local antiseptic dressings, and the administration of a broad spectrum antibiotic fail to bring about resolution, the abscess should be opened through a comparatively small incision. Provided all hairs and granulation tissue are removed from the abscess cavity, there is some prospect of curing the lesion. In all other circumstances an elective operation must be planned.

Operation should be performed only when the inflammation has been controlled by the measures indicated already.

The patient is placed on the operating table, for preference in the 'jack-knife' position.

Methylene blue is injected into the sinus to colour all the tracts, the nozzle of the syringe being pressed against the opening to obtain some pressure. A director is then inserted, and the cavity is laid open along its length. If diverticula are present, they also are dissected out. All hair and debris must be removed. In this way all sinuses are exteriorised. Bleeding-points are not tied, but only clamped. At the end of the operation the hæmostats are removed, and a piece of ribbon gauze moistened with 1:1000 adrenaline is used to pack the wound. A pressure dressing is applied. After six hours the pressure dressing is removed, and gauze moistened with saline solution is placed over the ribbon gauze. The following day the whole dressing is removed, and moist dressings are continued as an out-patient. Twice weekly the wound is inspected for bridging, or for the presence of any sinus that has been overlooked. In eight to ten days the wound should be filled with healthy granulations, by which time a tulle gras covering is all that is necessary. The average time for complete epithelialisation is about a month, but possibly this can be speeded by skin grafting. The recurrence rate is less than 2 per cent.



Recurrent Pilonidal Sinus.—Three possibilities account for this disappointment. (1) A diverticulum of the main channel has been overlooked at the primary operation. (2) New hairs enter the skin or the scar. (3) When the natal fold is deformed by scarring following block dissection, the least trauma causes tearing of the scar, and the resulting crevice becomes contaminated with coliform and cutaneous bacteria.

INJURIES

The rectum or anal canal may be injured in a number of ways, all uncommon.

1. By falling in a sitting posture on to a spiked or blunt-pointed object. The up-turned leg of a chair, handle of a broom, floor-mop, pitchfork, or a broken shooting-stick (fig. 1271) have all resulted in rectal impalement.



FIG. 1271.—Rectal impalement by a broken shooting-stick.

2. By the foetal head during childbirth.

3. During the administration of an enema by a syringe fitted with a bone, glass, or vulcanite nozzle.

4. During sigmoidoscopy, usually when examining a patient suffering from ulcerative procto-colitis or amoebic dysentery.

5. 'Split Perineum'. A lacerated wound of the perineum, involving the anal canal, is an occasional pillion-riding accident.

6. Compressed-air rupture (p. 897).

Diagnosis.—When there is a history of rectal impalement, the first interrogation should be, 'Has the patient passed urine since the accident?' The anus having been inspected, the abdomen should be palpated. If rigidity or tenderness is present, early laparotomy is imperative. Prior to the operation an urethral catheter is passed. If the urine is blood-stained and/or the quantity recovered is unexpectedly small, it is wise to suspect ruptured bladder or urethra (pp. 1123 and 1186).

Treatment.—After the patient has been anaesthetised, the rectum is examined with a finger and a speculum, especial attention being directed to the anterior wall. A left lower laparotomy is then performed. If an intraperitoneal rupture of the rectum is found, the perforation is closed with sutures. Should blood be present beneath the pelvic peritoneum, it is necessary to mobilise the recto-sigmoid, which allows the rectum to be drawn upwards, thus permitting the perforation below the pelvic diaphragm to be closed securely. A perforation in the bladder also can be sutured *via* this avenue. After closing the laparotomy wound, a left iliac colostomy is performed through a separate grid-iron incision. In cases where the bladder has been injured a self-retaining urethral catheter is placed in position.

These vital steps having been accomplished, if there is a lacerated wound of the perianal tissues or the anal canal, debridement of the wound must be carried out. The wound is then lightly packed with gauze and allowed to heal slowly.

FOREIGN BODIES IN THE RECTUM

The variety of foreign bodies which have found their way into the rectum is hardly less remarkable than the ingenuity displayed in their removal (fig. 1272). A turnip has been delivered *per anum* by the use of obstetric forceps. A stick firmly impacted has been withdrawn by inserting a gimlet into its lower end. A tumbler, mouth looking downwards, has been extracted by filling the interior with a wet plaster of Paris bandage, leaving the end of the bandage protruding, and allowing the plaster to set.

If insurmountable difficulty is experienced in grasping any foreign body in the rectum, a left lower laparotomy is necessary, which allows that object to be pushed from above into the assistant's fingers in the rectum. If there is considerable laceration of the mucosa a temporary colostomy is advisable.

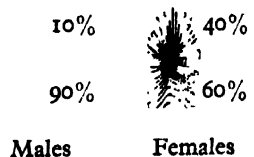


FIG. 1272.—Pepper-pot in the rectum. On removal it was found to be inscribed 'A present from Margate'! (Dr. L. S. Carstairs, London.)

ANAL FISSURE (syn. FISSURE-IN-ANO)

Definition.—An elongated ulcer in the long axis of the anal canal.

Location.—The site of election for an anal fissure is the mid-line posteriorly. The next most frequent situation is the mid-line anteriorly. The relative distribution of fissure in these two sites is —————→



Ætiology.—The cause of anal fissure, and particularly the reason why the midline posteriorly is so frequently affected, is not completely understood. A probable explanation is as follows: the posterior wall of the rectum curves forwards from the hollow of the sacrum to

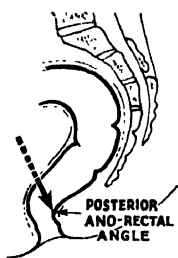


FIG. 1273.—
Probable mechanism of tearing of the anal mucosa in the case of a posterior fissure.

join the anal canal, which then turns sharply backwards. During defæcation the pressure of a hard fæcal mass is mainly on the posterior ano-rectal angle (fig. 1273) in which event the overlying epithelium is greatly stretched, and, being relatively unsupported by muscle, is placed in a vulnerable position where a scybalous mass is being expelled. Possibly some cases are due to tearing down of an anal valve of Ball. As shown by the above percentages, an anterior anal fissure is much more common in women, particularly in those who have borne children. This can be explained by the lack of support of the anal mucous membrane by a damaged pelvic floor.

One cause of anal fissure is certain—an incorrectly performed operation for hæmorrhoids in which too much skin is removed. This results in anal stenosis and tearing of the stricture when a hard motion is passed.

Pathology.—An anal fissure is either acute or chronic, and is situated either at a low or a high level. A high-level lesion is placed entirely within the anal canal, and extends through the dentate line. The vast majority of anal fissures are of the low-level variety.

Acute anal fissure is a deep tear through the skin of the anal margin extending into the anal canal. There is little inflammatory induration or œdema of its edges.

Chronic anal fissure is characterised by inflamed indurated margins, and a base consisting of either scar tissue or the lower border of the internal sphincter muscle. The ulcer is canoe-shaped, and at the inferior extremity frequently there is a tag of skin, usually œdematous. This tag is known picturesquely as a sentinel pile—‘sentinel’ because it guards the fissure. There is always spasm of the involuntary musculature of the internal sphincter. In long-standing cases this muscle becomes organically contracted by infiltration of fibrous tissue. Infection is common and may be severe, ending in abscess formation. A cutaneous fistula may follow.

Clinical Features.—The condition is more common in women, and generally occurs during the meridian of life. It is uncommon in the aged, because of muscular atony; on the other hand anal fissure is not rare in children, is sometimes encountered during infancy, and may cause acquired mega-colon (p. 893).

Pain is the symptom—sharp agonising pain starting during defæcation, often overwhelming in intensity and lasting an hour or more. As a rule it ceases suddenly, and the sufferer is comfortable until the next action of the bowel. Periods of remission occur for days or weeks. The patient tends to become constipated rather than go through the agony of defæcation.

Stools are frequently streaked with blood.

Discharge.—A slight discharge of serum accompanies fully established cases. A purulent discharge follows if a subcutaneous abscess bursts into the anal canal or externally.

On Examination.—Frequently, in cases of some standing, a sentinel skin tag can be seen. This, together with a typical history and a tightly closed, puckered anus (fig. 1274), is almost pathognomonic of the condition. By gently parting the margins of the anus, the lower end of the fissure can sometimes be displayed.

Because of the intense pain it causes, digital examination of the anal canal should not be attempted at this stage unless (a) the fissure cannot be seen (high-level fissure) or (b) it seems imperative to exclude some intrarectal condition. In these circumstances the local application of a surface anæsthetic such as 5 per cent. xylocaine on a pledget of cotton-wool, left in place for about five minutes, will enable the necessary examination to be made. In early cases the edges of the fissure are impalpable; in fully established cases a characteristic crater which feels like a vertical buttonhole can be palpated. On account of the intolerable pain it produces, proctoscopy should not be attempted.

Differential Diagnosis :

Carcinoma of the anus in its very early stages sometimes simulates a fissure. If real doubt exists, the lesion must be excised under general anæsthesia and submitted to histological examination.

Multiple fissures in the peri-anal skin are seen as a complication of pruritus ani (p. 996) and procto-colitis (p. 1001).

Anal chancre is becoming more common and presents as a moist, painless indurated ulcer of recent origin (fig. 1275). The serous discharge contains spirochaetes. A glass pipette is used to aspirate a few drops which are placed on a slide for examination by dark-ground illumination. Lubricating gel from the finger-stall may prevent adequate aspiration of serum from a chancre.



FIG. 1275.—Anal chancre.

Tuberculous ulcer (p. 38) has an undermined edge.

Proctalgia Fugax (p. 988) causes severe episodic pain.

Treatment:

The pain of an anal fissure is so great that usually the patient demands relief, and consequently many patients with an acute fissure present early. The object of all treatment for this condition is to obtain *complete relaxation* of the internal sphincter. Provided the complications are dealt with, the fissure will slowly heal as soon as all spasm has disappeared.

Conservative Treatment:

In cases where the fissure is acute and superficial and where the inflammation is minimal, simple conservative measures will usually give relief. Xylocaine 5 per cent. in a water-soluble lubricant is introduced with a fine nozzle into the anal canal. After waiting a few minutes for the surface anæsthetic to act, relaxation may be sufficient to permit the passage of a well-lubricated finger into the canal. Following this a small anal dilator (fig. 1276) may be passed and, if the anæsthesia is adequate, it may be possible to



FIG. 1274.—Sentinel pile associated with fissure. Puckering of the anus is also shown.

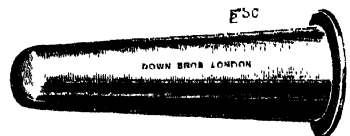


FIG. 1276.—St. Mark's Hospital anal dilator.

introduce the largest dilator. Anal dilators are commonly made in three sizes and it may not be possible to use the largest dilator until several days have passed. The patient is supplied with xylocaine lubricant and instructed to pass a dilator twice a day for a month, by which time the fissure is usually healed. Laxatives are prescribed to ensure that the motions are soft.

Operative Measures

The simplest and possibly the best procedure is wide dilatation of the sphincter. Under general anaesthesia the index and middle finger of each hand are inserted simultaneously into the anus and pulled apart to give maximal dilatation. The patient can go home the same day but should be warned that there may be some faecal incontinence lasting possibly for a week or ten days.

Should these measures prove ineffective, or if the fissure is chronic with fibrosis, a skin tag, or a mucous polypus, then operative measures are advisable. General anaesthesia is best, though some surgeons use a local anaesthetic in the form of xylocaine or procaine introduced into the ischio-rectal fossa on each side, in order to anaesthetise the nerves passing towards the rectum. With the patient in the lithotomy position a well greased Sims' speculum is introduced. This will permit a very good view of the fissure which becomes stretched to show the fibres of the internal sphincter running transversely in its floor. The essential part of the operation is to divide all of these transverse fibres so that the floor of the fissure is completely smooth. If a sentinel pile is present, this is excised, removing a triangular piece of skin with the apex at the anus and the base outwards. The ends of the divided muscle retract and a smooth wound is left. The after-treatment consists of attention to bowels, a daily bath, and the passage of an anal dilator until the wounds have healed, which usually takes about three weeks. Despite the presence of the wound there is little or no pain and the final results are excellent.

HYPERTROPHIED ANAL PAPILLA

Anal papillae occur at the dentate line, and are remnants of the ectodermal membrane that separated the hind-gut from the proctodæum. As these papillae are present in fully 60 per cent. of patients examined proctologically, they should be regarded as normal structures. Anal papillae can become elongated, as they frequently do in the presence of an anal fissure. Occasionally an elongated anal papilla may be the cause of pruritus. An elongated anal papilla associated with pain and/or bleeding at defaecation, is sometimes encountered in infancy. At any time of life, hæmorrhage into a hypertrophied anal papilla can cause sudden rectal pain. Prompt examination sometimes reveals blood-clot being extruded from a ruptured papilla.

A prolapsed papilla may become nipped by contraction of the sphincter mechanism after defaecation. It is possible that repeated traction on the pedicle of an anal papilla results in an anal fissure.

Treatment.—Using a slotted proctoscope, elongated papillae without hæmorrhoids should be crushed and excised after injecting the base with local anaesthetic. When elongated papillae complicate internal hæmorrhoids, this is an indication for operative treatment of the hæmorrhoids, as well as excision of the elongated papillae.

PROCTALGIA FUGAX

This disease is characterised by attacks of severe pain arising in the rectum, recurring at irregular intervals and apparently unrelated to organic disease. The pain is described as cramp-like, often occurs when the patient is in bed at night, lasts only a few minutes, and disappears spontaneously. It may follow straining at stool, sudden explosive bowel action or ejaculation. It seems to occur more commonly in patients suffering from anxiety or undue stress. The pain may be unbearable—it is possibly due to segmental cramp in the pubo-coccygeus muscle. It is unpleasant, incurable, but fortunately harmless and gradually subsides.

HÆMORRHOIDS¹ (*syn.* PILES²)

Hæmorrhoids are varicose veins occurring in the ano-rectum, and originating in the plexuses formed by radicles of the superior, middle, and inferior rectal veins.

Such hæmorrhoids may be **external** or **internal**—external or internal to the anal orifice. The external variety are covered by skin, while the internal variety are clothed by mucous membrane. When the two varieties are associated, they are known as **intero-external** hæmorrhoids.

Hæmorrhoids may be 'symptomatic' of some other condition, and this important fact must be remembered when they are found. Symptomatic hæmorrhoids may appear (a) in *carcinoma of rectum*. This, by compressing or causing thrombosis of the superior rectal vein, gives rise to hæmorrhoids (fig. 1277) sufficiently often to warrant examination of the rectum and the recto sigmoid junction for a neoplasm in every case of hæmorrhoids. (b) During *pregnancy*. Pregnancy piles are due to compression of the superior rectal veins by the pregnant uterus and the effect of progesterone on the smooth muscle in the walls of the veins. (c) From *straining on micturition* consequent upon a stricture of the urethra. (d) In *venous hypertension*. Contrary to the usual belief, in one hundred and twenty-eight consecutive cases of portal hypertension, McPherson did not encounter a single example of hæmorrhoids that could be attributed to portal cirrhosis, although bleeding œsophageal varices often complicate portal hypertension (p. 808).

The great majority of hæmorrhoids are not symptomatic, and the description that follows concerns hæmorrhoids that are *not* a manifestation of some underlying cause.

Internal hæmorrhoids, which include intero-external hæmorrhoids, are exceedingly common. Essentially the condition is a varicosity of the internal rectal plexus, but because of the communication between the internal and external rectal plexuses, if the former becomes varicose, the latter is liable to become involved also.

Ætiology: Hereditary.—The condition is so frequently seen in members of the same family that there must be a predisposing factor, such as a congenital weakness of the vein walls or an abnormally large arterial supply to the rectal plexus.

Varicose veins of the legs and hæmorrhoids often occur concurrently.

Morphological.—In quadrupeds, gravity aids, or at any rate does not retard, return of venous blood from the rectum. Consequently venous valves are not required. In man, the weight of the column of blood unassisted by valves produces a high venous pressure in the lower rectum, unparalleled in the body. Except in a few fat, old dogs, hæmorrhoids are exceedingly rare in animals.

Anatomical.—(1) The collecting radicles of the superior rectal vein lie unsupported in the very loose submucous connective tissue of the rectum. (2) These veins pass through muscular tissue and are liable to be constricted by its contraction



FIG. 1277.—Carcinoma of the rectum associated with hæmorrhoids. A not infrequent diagnostic pitfall.

¹ 'The common people call them piles, the aristocracy call them hæmorrhoids, the French call them figs—what does it matter so long as you can cure them?'—John Arderne, 1370.

² Greek—*haima* = blood, *rhoos* = flowing.

³ Latin—*pila* = a ball.

John Arderne, 1307–1390. Practised surgery in Newark, and later in London.
Archibald Ian Stewart McPherson, Contemporary. Surgeon, Royal Infirmary, Edinburgh.

during defæcation. (3) The superior rectal veins, being tributaries of the portal vein, have no valves.

Exciting Causes.—Straining accompanying constipation or that induced by over-purgation is considered to be a potent cause of hæmorrhoids. Less often the diarrhoea of enteritis, colitis, or the dysenteries aggravates latent hæmorrhoids.

Pathology.—Internal hæmorrhoids in association with the terminal divisions of the superior rectal artery are arranged in three groups at 3, 7, and 11 o'clock with the patient in the lithotomy position (fig. 1278). In between

these three principal hæmorrhoids there may be smaller, subsidiary hæmorrhoids. Each principal hæmorrhoid can be divided into three parts:

The pedicle is situated in the rectum just above the ano-rectal ring. As seen through a proctoscope, it is covered with pale pink mucosa through which large tributaries of the superior rectal vein can be seen. Occasionally a pulsating artery can be felt in this situation.

The internal hæmorrhoid, which commences at the ano-rectal ring and ends at the dentate line. It is bright red or purple, and covered by mucous membrane.

An external associated hæmorrhoid lies between the dentate line and the anal margin. It is covered by skin, through which blue veins can be seen, unless fibrosis has occurred. This associated hæmorrhoid is present only in well-established cases.

Entering the pedicle of each internal hæmorrhoid is a terminal branch of the superior rectal artery. Very occasionally there is a hæmangiomatous condition of this artery—an 'arterial pile'.

Clinical Features.—*Bleeding*, as the name hæmorrhoid implies, is the principal and earliest symptom. At first the bleeding is slight; it is bright red and occurs during defæcation (a 'splash in the pan'), and it may continue thus for months or years. Hæmorrhoids that prolapse sufficiently to be nipped by the anal sphincter for a moment at stool are called *first-degree hæmorrhoids*. Their only symptom is bleeding, which is the result of the distension and subsequent rupture of the veins by sphincteric compression.

Prolapse is a much later symptom. In the beginning the protrusion is slight and occurs only at stool, and reduction is spontaneous. As time goes on the hæmorrhoids do not reduce themselves, but have to be replaced digitally by the patient. Hæmorrhoids that prolapse on defæcation but need to be replaced manually and then stay reduced are called *second-degree hæmorrhoids*. Still later, prolapse occurs during the day, apart from defæcation, when the patient is tired or exerts himself. Hæmorrhoids that are permanently prolapsed are called *third-degree hæmorrhoids*. By now, the hæmorrhoids have become a source of great discomfort and cause a feeling of heaviness in the rectum.

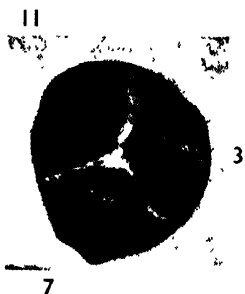


FIG. 1278.—Typical third-degree hæmorrhoids. Note their 3, 7, and 11 o'clock positions. (E. T. C. Milligan, F.R.C.S., London.)

Discharge.—A mucoid discharge is a frequent accompaniment of prolapsed hæmorrhoids. It is composed of mucus from the engorged mucous membrane, sometimes augmented by leakage of ingested liquid paraffin. *Pruritus* will almost certainly follow this discharge.

Pain is absent unless complications supervene.

On inspection there may be no evidence of internal hæmorrhoids. In more advanced cases redundant folds or tags of skin can be seen in the position of one or more of the three primary hæmorrhoids. When the patient strains, internal hæmorrhoids may come into view transiently, or if they are of the third degree they prolapse and remain prolapsed.

Palpation.—Internal hæmorrhoids can seldom be felt unless they are thrombosed. Possibly very large internal hæmorrhoids are palpable, but who can say if some thrombosis has not occurred in these apparent exceptions to the rule?

Proctoscopy.—A proctoscope is passed to its fullest extent and the obturator is removed. The instrument is then slowly withdrawn. Just below the ano-rectal ring internal hæmorrhoids, if present, will bulge into the lumen of the proctoscope (fig. 1279).

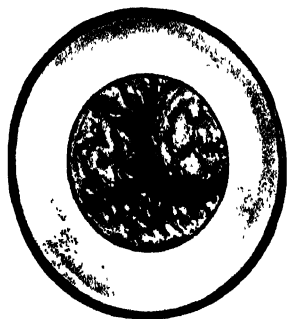


FIG. 1279.—Internal hæmorrhoids seen through a proctoscope.



FIG. 1280.—Strangulated internal hæmorrhoids.

Complications :

Profuse hæmorrhage is not rare. Most often it occurs in the early stages of the second degree. The bleeding occurs mainly externally, but it may continue internally after the bleeding hæmorrhoid has retracted or has been returned. In these circumstances the rectum is found to contain blood.

Strangulation.—One or more of the internal hæmorrhoids prolapse and become gripped by the external sphincter. Further congestion follows because the venous return is impeded. Second-degree hæmorrhoids are most often complicated in this way. Strangulation (fig. 1280) is accompanied by considerable pain, and is often spoken of by the patient as an 'acute attack of piles'.¹ Unless the internal hæmorrhoids can be reduced within an hour or two, strangulation is followed by thrombosis.

Thrombosis.—The affected hæmorrhoid or hæmorrhoids become dark purple or black (fig. 1281) and feel solid. Considerable œdema of the anal margin accompanies thrombosis. Once thrombosis has occurred the pain of strangulation largely passes off, but tenderness persists.

Ulceration.—Superficial ulceration of the exposed mucous membrane often accompanies strangulation with thrombosis.

Gangrene occurs when strangulation is sufficiently tight to constrict the arterial supply of the hæmorrhoid. The resulting sloughing is usually superficial and localised. Occasionally a whole hæmorrhoid sloughs off, leaving an ulcer



FIG. 1281.—Strangulated internal hæmorrhoids with thrombosis. (K. G. Buckler, F.R.C.S., Bristol.)

¹ An 'acute attack of piles' also embraces a thrombotic pile (p. 995) and an inflamed anal skin tag.

which heals gradually. Very occasionally massive gangrene extends to the mucous membrane within the anal canal and rectum.

Fibrosis.—After thrombosis, internal hæmorrhoids sometimes become converted into fibrous tissue. The fibrosed hæmorrhoid is at first sessile, but by repeated traction during prolapse at defæcation, it becomes pedunculated and constitutes a fibrous polypus that is readily distinguished by its white colour from an adenoma which is bright red. Fibrosis following transient strangulation commonly occurs in the subcutaneous part of a primary hæmorrhoid. Fibrosis in an external hæmorrhoid favours prolapse of an associated internal hæmorrhoid.

Suppuration is uncommon. It occurs as a result of infection of a thrombosed hæmorrhoid. Throbbing pain is followed by perianal swelling, and a perianal or submucous abscess results.

Pylephlebitis (*syn.* Portal Pyæmia).—Theoretically, infected hæmorrhoids should be a potent cause of portal pyæmia and liver abscesses (p. 797). Although cases do occur from time to time, this complication is surprisingly infrequent.

TREATMENT OF INTERNAL HÆMORRHOIDS

Palliative treatment is recommended when the hæmorrhoids are a symptom of some other condition or disease except, of course, when a carcinoma is present. The bowels are regulated by Isogel, and if necessary a small dose of Senokot at night. Lubafax or a cream of equal parts of zinc oxide and castor oil, inserted into the rectum from a collapsible tube fitted with a nozzle, at night and before defæcation, is of service.

In cases of inflamed and permanently prolapsed hæmorrhoids with a patulous anus, repeated dressings of glycerine acid tannic B.P. are more useful than the legion of remedies usually recommended (Eisenhammer). Surgery offers the only hope of permanent cure.

Active Treatment.—This consists of injection or formal operation, each with specific indications. Treatment should not be withheld because the patient is elderly or infirm.

Injection Treatment.—*Indications.*—This is ideal for first-degree internal hæmorrhoids which bleed. Early second degree hæmorrhoids are often cured by this method but a proportion relapse.

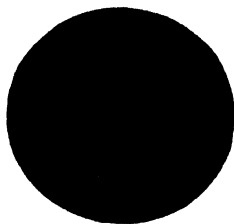


FIG. 1282.—Correct site for injecting a hæmorrhoid. (After W. B. Gabriel, F.R.C.S., London.)

from 3 to 5 ml. of 5 per cent. phenol in almond oil is injected.

The injection should produce elevation and pallor of the mucosa. The solution spreads in the submucosa upwards to the pedicle, and downwards to the internal hæmorrhoid and to secondary hæmorrhoids if present, but it is prevented by the inter-muscular septum from reaching the external hæmorrhoid. There is

Technique.—The patient should have an empty rectum, but no special preparation is necessary. A proctoscope is introduced, and the hæmorrhoids are displayed. The proctoscope is introduced farther in until the hæmorrhoid has almost disappeared from the lumen and only its upper end is visible. The injection is made at this point above the main mass of each hæmorrhoid (fig. 1282) into the submucosa at or just above the ano-rectal ring. Using Gabriel's syringe (fig. 1283) with the bevel of the needle directed towards the rectal wall,

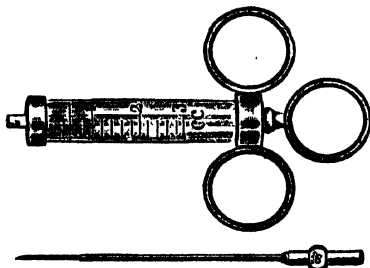


FIG. 1283.—Gabriel's syringe for injecting hæmorrhoids.

Stephen Eisenhammer, *Contemporary*. Proctologist, Johannesburg, South Africa.

In 1871, Mitchell of Clinton, Illinois, first used carbolic acid for injecting hæmorrhoids. Itinerant irregular practitioners exploited the method.

William Bashall Gabriel, *Contemporary*. Consulting Surgeon, St. Mark's and Royal Northern Hospitals, London.

slight, transient bleeding from the point of puncture. The injection is painless, and there is no special after-treatment. If there is only one hæmorrhoid present, it may be cured by one injection; if all three hæmorrhoids are equally enlarged, each is injected at the same session. Often three sessions at six-weekly intervals are required. Care should be taken not to re-inject submucosa already sclerosed otherwise sloughing may occur.

Operation.—Indications.—The following cases are unsuitable for injection treatment. (1) Third-degree hæmorrhoids; (2) failure of conservative treatment of second-degree hæmorrhoids; (3) fibrosed hæmorrhoids; (4) entero-external hæmorrhoids when the external hæmorrhoid is well defined; (5) when arterial pulsation can be felt in the pedicle. These are indications for hæmorrhoidectomy.

TREATMENT OF COMPLICATIONS

In cases of strangulation, thrombosis, and gangrene, it was formerly believed that surgery would cause pyelephlebitis. If an adequate antibiotic cover is given from the start, this is not found to be so. Bed rest with frequent hot sitz baths, warm saline compresses with firm pressure usually cause the pile mass to shrink considerably in three or four days when ligature and excision of the piles can be carried out. Some surgeons consider that the operation at this stage increases the risk of post-operative stenosis and delay surgery for a month or so. They then review the situation and only carry out hæmorrhoidectomy if necessary.

In cases where the patient has been admitted because of severe hæmorrhage the cause usually lies in a bleeding diathesis or the use of anti-coagulants (p. 131). If such are excluded, a local compress containing adrenaline solution, with an injection of morphine and blood transfusion if necessary, will usually control the hæmorrhage. After blood replacement is adequate, ligation and excision of the piles may be required.

HÆMORRHOIDECTOMY

Forty-eight hours' pre-operative treatment is necessary. An aperient, a little more than the patient usually takes, or, if none is habitually taken, $\frac{1}{2}$ drachm (2 ml.) of cascara evacuant, is given on the evening forty-eight hours prior to the operation. On the following morning a small dose of effervescent saline is given before breakfast. On the evening before the operation a soap-and-water enema is administered, and the anal region is shaved. On the morning of the operation the rectum is washed out with water by means of a funnel and tube attached to a catheter.

Ligation and Excision.—With the patient in the lithotomy position the sphincter is widely stretched, a manoeuvre which greatly reduces post-operative pain. The internal hæmorrhoids are then prolapsed by traction on the skin tags, or on the skin of the anal margin. Each hæmorrhoid is dealt with as follows: It is picked up with dissecting forceps and traction is exerted. Traction displays a longitudinal fold (the pedicle) above the hæmorrhoid. Each pedicle is grasped in a fine-pointed hæmostat, as also is each external hæmorrhoid or skin tag connected with each internal hæmorrhoid. These pairs of hæmostats, when held out by the assistants, form a triangle. The operator takes the left lateral pair of hæmostats in the palm of his hand and places the extended forefinger in the anal canal to support the internal hæmorrhoid. In this way traction is applied to the skin of the anal margin. With scissors, a V-shaped cut is made (fig. 1284 (A)), each limb of which is placed on either side of the skin-holding hæmostat. This cut traverses the skin and the corrugator cutis ani. Exerting further traction a little blunt dissection exposes the lower border of the internal sphincter. A transfixion ligature of No. 3 chromic catgut is applied to the

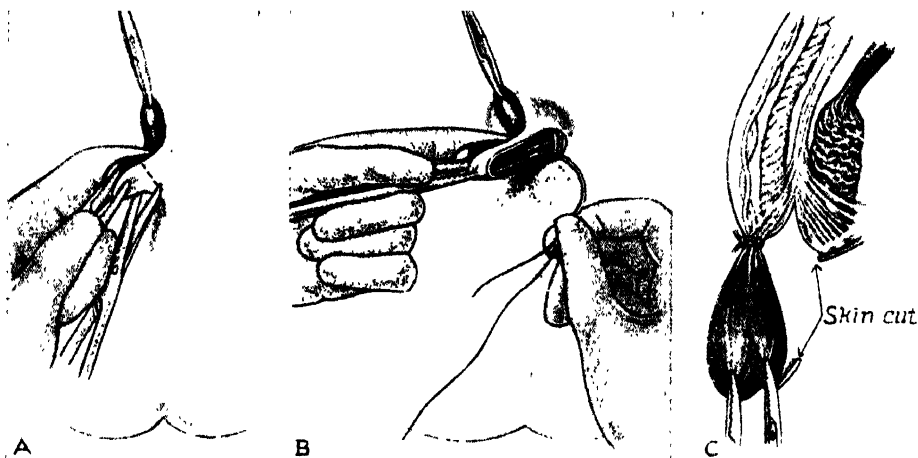


FIG. 1284.—Ligation and excision of hæmorrhoids: (A) The skin cut to the left lateral hæmorrhoid; (B) Transfixion of the pedicle; (C) Ligation.

pedicle at this level (fig. 1284 (B)). Each hæmorrhoid having been dealt with in this manner (fig. 1284 (C)), they are excised $\frac{1}{2}$ inch (1.25 cm.) distal to the ligature, the ends of which are cut about $\frac{1}{2}$ inch from the knot. The stumps of the ligated hæmorrhoids are returned to the rectum by tucking a piece of gauze into the anal canal with closed scissors.

The margins of the skin wounds are trimmed so as not to leave overhanging edges, viz. —————→¹.

Bleeding subcutaneous arteries having been secured, the corners of three pieces of petroleum-jelly gauze are tucked into the anus so as to cover the areas denuded of skin. A pad of gauze and wool, and a firmly applied T-bandage complete the operation.

Post-operative Treatment.—Twice daily baths are started from the first day and liquid paraffin in the evenings. The dressings float off in the bath; if not, they can be removed. An enema is rarely necessary as the bowels usually start on the fourth or fifth day. The baths and dry dressings (a sterile sanitary towel is best for this purpose) held in position by a T-bandage keep the area clean. On the seventh day a finger is passed into the rectum to assess the degree of spasm. If there is risk of stricture formation, a St. Mark's Hospital dilator (fig. 1276) is passed daily. The patient can leave hospital on the tenth to fourteenth day when the risk of secondary hæmorrhage has passed. In the later stages a dry dressing is employed during the day and a zinc and castor oil dressing at night. The paraffin should be stopped after the tenth day and the patient encouraged to pass solid motions in order to dilate the anal canal. The wounds heal in three to six weeks.

Post-operative complications may be early or late.

Early. *Pain* may demand repeated pethidine. Xylocaine jelly introduced through a fine nozzle into the rectum, as necessary, is of considerable value.

Retention of urine is not unusual after hæmorrhoidectomy in male patients, and frequently it is precipitated by the presence of a rectal tube, or pack, or both. Before resorting to catheterisation, the patient should be assisted to a hot bath into which he may be able to void urine. If this does not succeed, it is wise to give carbachol 1 ml. (0.25 mg.) by intramuscular injection.

Reactionary hæmorrhage is much more common than secondary hæmorrhage. The hæmorrhage may be mainly or entirely concealed, but will become evident on examining the rectum.

Treatment.—A suitable dose of morphine is given intravenously. If the bleeding persists, the patient must be taken to the operating theatre and the bleeding-point

¹ If it looks like a clover, the trouble is over,
If it looks like a dahlia, it's surely a failure.

cured by diathermy or under-running with a ligature on a needle. Should a definite bleeding-point not be found, suspected areas are under-run in this way.

Late. Secondary hæmorrhage is uncommon ; when it occurs, it does so about the seventh or eighth day after operation. It is usually controlled by morphia but the hæmorrhage is severe, an anæsthetic should be given and a catgut stitch inserted to occlude the bleeding vessel.

Anal stricture.—This must be prevented at all costs. A rectal examination at the tenth day will indicate if stricturing is to be expected. It may then be necessary to give a general anæsthetic and dilate the anus to take four fingers. After that daily use of the dilator should give a satisfactory result.

Anal fissure (p. 985) and submucous abscess (p. 1005) may also occur.

EXTERNAL HÆMORRHOIDS

Unlike internal hæmorrhoids, external hæmorrhoids comprise a conglomerate group of distinct clinical entities.

1. Acute external plexus hæmatoma is commonly termed a 'thrombotic pile.' It is a small hæmatoma occurring in the perianal subcutaneous connective tissue, usually superficial to the corrugator cutis ani muscle. The condition is due to the bursting of an anal venule consequent upon straining at stool, coughing, or lifting a heavy weight.

The condition appears suddenly and is very painful, and on examination a tense, tender swelling which resembles a semi-ripe blackcurrant is seen (fig. 1285). The hæmatoma is usually situated in a lateral region of the anal margin. Untreated it may resolve, suppurate, fibrose, and give rise to a cutaneous tag or burst and extrude the clot or continue bleeding.

In the majority of cases resolution or fibrosis occurs. Indeed, this condition has been called 'a five-day, painful, self-curing lesion' (Milligan).

Provided it is seen within thirty-six hours of the onset, a perianal hæmatoma is best treated as an emergency. Under local anæsthesia the hæmorrhoid is bisected and the two halves are excised together with $\frac{1}{2}$ inch (1.25 cm.) of adjacent skin. This leaves a pear-shaped wound which is allowed to granulate. The relief of pain is immediate and a permanent cure is certain. On the rare occasions in which a perianal hæmatoma is situated anteriorly or posteriorly, it should be treated conservatively because of the liability of a skin wound in these regions to become an anal fissure.



FIG. 1285.—'Thrombotic' pile.

2. Associated with internal hæmorrhoids = intero-external hæmorrhoids. These have been discussed (p. 989).

3. Dilatation of the veins of the anal verge becomes evident only if the patient strains, when a bluish cushion-like ring appears. This

variety of external hæmorrhoid is almost a perquisite of those who lead a sedentary life. The only treatment required is an adjustment in habits of the patient.



FIG. 1286.—Condylomata ani.

The diagnosis is seldom established until microscopic examination has been undertaken.

4. A 'sentinel' pile is associated with an anal fissure, and has been discussed on p. 986.

SOME CONDITIONS THAT SIMULATE EXTERNAL HÆMORRHOIDS

Anal warts are usually multiple and present the same characteristics as warts elsewhere. Occasionally they accompany gonococcal proctitis. Simple warts respond well to treatment by applications at weekly intervals of 25 per cent. podophyllin in liquid paraffin. After three or four applications, any residual warts should be excised.

Condylomas (fig. 1286) have a smoother surface and are more pedunculated than the foregoing. Also they are usually moist, with a glairy discharge.

Hypertrophic tuberculide of the anus is rare. These are multiple yellowish-brown papillomatous excrescences.

PRURITUS ANI

This is intractable itching around the anus. Usually the skin is white and hyperkeratotic and may become cracked and moist. The causes are numerous and varied :

1. *Lack of cleanliness, excessive sweating, and wearing rough or woollen under-clothing* are common causes.

2. *An anal or perianal discharge* which renders the anus moist. The causative lesions include an anal fissure, fistula-in-ano, prolapsed internal or external hæmorrhoids, and excessive ingestion of liquid paraffin.

3. *A vaginal discharge*, especially due to the trichomonas vaginalis.

4. *Parasitic Causes*.—Threadworms (*Enterobius vermicularis*) should be excluded, especially in young subjects.¹ Scabies and pediculosis pubis may infest the anal region. When the anal skin shows some form of dermatitis which has a well-defined border, mycotic diseases of the skin due to yeasts and fungi should be suspected. Microscopic and cultural examinations of muco-pus taken from the region are necessary to establish the diagnosis.

5. *Allergy* is sometimes the cause, in which case there is likely to be a history of other allergic manifestations, such as urticaria, asthma, or hay-fever. Antibiotic therapy may be the precipitating factor.

6. *A Raised pH of the Fæces*.—It would appear that the incidence of perianal dermatitis varies with the pH of the fæces ; the more acid the stool and the perianal skin, the less frequent is the occurrence of perianal dermatitis.

7. *A Psychoneurosis*.—It is alleged that in a few instances neurotic individuals become so immersed in their complaint that a pain-pleasure complex develops, the

¹ Children suffering from thread worms should wear gloves at night, lest they scratch the perianal region and are reinfested with ova by nail biting—'Parasites lost, Parasites regained'

leasure being the scratching. Possibly this is true, but such a syndrome should not be assumed without firm grounds for coming to this conclusion.

Treatment.—The cause is treated. Other methods include :

Hygienic Measures.—Wet cotton-wool should be substituted for toilet paper. Soap should be avoided, and replaced by a detergent. These measures alone, combined with wearing cotton cellular underwear and applications of calamine lotion, are all that is necessary to cure some cases.

The following measures are directed mainly to those frequent instances where no obvious reason for the itching can be demonstrated. The best sedative appears to be the alkaloid reserpine, 0.25 mg. daily. This has a tranquillising effect on many anxious patients, but is without direct effect on the pruritus itself.

Hydrocortisone.—In cases with dermatitis, and only in cases with dermatitis, prednisolone, applied topically in a cream of 1 per cent. is often beneficial; sometimes after discontinuation of the therapy the pruritus is liable to return, in which event 5 per cent. Xylocaine ointment can be substituted for a time.

Strapping the buttocks apart (fig. 1287) is a most useful procedure, especially when the pruritus is acute, and in chronic cases when the opposing surfaces are moist. The strapping is worn so long as the patient finds it beneficial.

Operative Treatment.—This may be necessary for a concomitant lesion of the ano-rectum which is thought to initiate or contribute to the pruritus. When other measures fail to bring permanent relief the advisability of performing an operation to endeavour to rid the patient of the pruritus should be considered. The operation about to be described is not recommended in the presence of moist dermatitis.

The clover-leaf operation consists of removal of a large piece of pruritic skin from each quadrant of the perianus, viz. —————→

Pre-operatively a daily enema is given, and sulphasuxidine is administered orally for five days. Shaving is undertaken after the patient has been anaesthetised. Turell has found this operation curative in one hundred and ten instances of otherwise refractory pruritus and not associated with cutaneous changes. Hughes covers the denuded areas with a skin graft, in the same manner as he recommends for fistula-in-ano.



PROLAPSE OF THE RECTUM Partial Complete

Partial Prolapse.—The mucous membrane and submucosa of the rectum protrude outside the anus for not more than between $\frac{1}{2}$ and $1\frac{1}{2}$ inches (1.25 and 3.75 cm.). When the prolapsed mucosa is palpated between the finger and thumb, it is evident that it is composed of no more than a double layer of mucous membrane (cf. complete prolapse). The condition occurs most often at the extremes of life—in children between one and three years of age, and in elderly people.

In Infants.—The direct downward course of the rectum, due to the as yet



FIG. 1288.—The absence of the normal sacral curve predisposes to rectal prolapse in an infant (cf. fig. 1273).

undeveloped sacral curve (fig. 1288) predisposes to this condition. The presence of a rectal adenomatous polyp must be excluded.

In children, partial prolapse often commences after an attack of diarrhoea as a result of severe whooping cough, or from loss of weight and consequent diminution in the amount of fat in the ischio-rectal fossæ.

In adults the condition is usually associated with third-degree hæmorrhoids. In the female a torn perineum predisposes to prolapse, and in the male straining from urethral obstruction. In old age, both partial and complete prolapse are due to atony of the sphincter mechanism.

Partial prolapse may follow an operation for fistula-in-ano where a large portion of muscle has been divided. Here the prolapse is usually localised to the damaged quadrant and is seldom progressive.

Prolapsed mucous membrane is pink (fig. 1290); prolapsed internal hæmorrhoids are plum coloured, and more pedunculated.

Treatment :

In Infants and Young Children : (1) *Digital Reposition*.—The mother must be taught to replace the protrusion. The distal two-thirds of the index finger is wrapped in Kleenex tissue. The finger is inserted into the protrusion, and the mass is eased into place. Gently the finger is withdrawn, leaving the Kleenex tissue to disintegrate. In cases of malnutrition, dietetic adjustments are necessary.

2. *Submucous Injections*.—If digital reposition fails after six weeks' trial, injections of 5 per cent. phenol in almond oil are carried out under general anaesthesia.

Technique.—The submucosa at the apex of the prolapse is injected circularly, so as to form a raised ring, up to 10 ml. of the solution being injected. A similar injection is made at the base of the prolapse. Alternatively, if the prolapse cannot be brought down, the injections are given through a proctoscope.

As a result of the aseptic inflammation following these injections, the mucous membrane becomes tethered to the muscle coat.

3. *Thiersch's Operation*.—When the prolapse persists in spite of these measures, Thiersch's operation (p. 999) is almost certain to succeed. In infants, insertion of the little finger into the anus before the wire is tied is recommended.

In Adults : (1) *Submucous injections* of phenol in almond oil occasionally are successful in cases of early partial prolapse.

(2) *Excision of the Prolapsed Mucosa*.—When the prolapse is unilateral the redundant mucosa can be excised after inserting and tying Goodsall's ligature (fig. 1289) which, after the needles have been cut off, permits the base of the prolapsed mucous membrane to be ligated in three portions lying in juxtaposition. When necessary, the operation is combined with hæmorrhoidectomy, and if the pedicle of one or more of the hæmorrhoids is broad, Goodsall's ligature is applied.

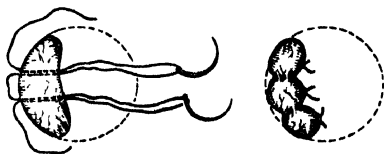


FIG. 1289.—Goodsall's ligature.

Complete prolapse (*syn.* procidentia) is less common than the partial variety. The protrusion consists of all layers of the rectal wall. It is more

than $1\frac{1}{2}$ inches (3.75 cm.) and commonly as much as 4 to 6 inches (10 to 15 cm.) in length. On palpation between the finger and the thumb the prolapse feels much thicker than a partial prolapse, and obviously consists of a double thickness of the entire wall of the rectum. Any prolapse over 2 inches (5 cm.) in length contains anteriorly between its layers a pouch of peritoneum, viz. —→ on this account a complete prolapse is a sliding hernia occurring through the pelvic diaphragm.

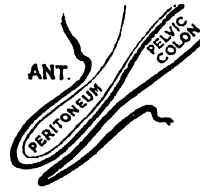


FIG. 1290.—Complete rectal prolapse.
(G. D. Adhia, F.R.C.S., Bombay.)

When large, the peritoneal pouch contains small intestine which returns to the general peritoneal cavity with a characteristic gurgle when the prolapse is reduced. The prolapsed mucous membrane (fig. 1290) is often arranged in a series of circular folds. Complete prolapse is uncommon in children. In adults it can occur at any age. Women are six times more often affected than men. Many of the patients suffering from this condition are obese.

In women, prolapse of the rectum is commonly associated with prolapse of the uterus.

Differential Diagnosis.—In the case of a child with abdominal pain, prolapse of the rectum must be distinguished from **ileo-cæcal intussusception** protruding from the anus. Figs. 1291 and 1292 make the differential diagnosis clear. In **recto-sigmoid intussusception** in the adult there is a deep groove (2 inches (5 cm.) or more) between the emerging protruding mass and the margin of the anus.



FIG. 1291.—Partial prolapse of the rectum.



FIG. 1292.—Intussusception protruding from the anus.

Treatment.—The Thiersch operation can be recommended in elderly patients, in those suffering from injury or disease of the spinal cord, and in the feeble-minded, in whom the condition is

relatively common, as well as in very early life (p. 997).

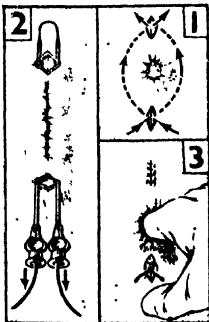


FIG. 1293.—The Thiersch operation (Dodd's modification).

The Thiersch Operation.—A short incision is made in the midline anteriorly and posteriorly about $\frac{1}{2}$ inch (1.25 cm.) from the anal verge. Large-bore hollow needles are inserted one at a time through the posterior wound, in such a way as to encircle the anus $\frac{1}{2}$ inch from the orifice (fig. 1293 (1)), until their points emerge from the anterior wound (fig. 1293 (2)). A malleable silver wire, gauge 19 or 20, is introduced through the points of the needles. The needles are withdrawn. The assistant introduces his index finger into the anal canal (fig. 1293 (3)), and the surgeon tightens the wire around the finger by twisting the ends of the wire. The finger is withdrawn and the ends of the wire are clipped short, and bent back. The wounds are then closed. A glycerol suppository or a low-pressure enema is given as required post-operatively. Confinement to bed is unnecessary.

By and large, the results of Thiersch's operation are remarkably successful but in about 50 per cent. of cases the wire breaks; it is, however, easily removed and the operation can be carried out several times, at intervals, on the same patient. Its main disadvantage is that spurious incontinence of fæces caused by fæcal impaction (p. 952) is wont to occur in all age groups. Constipation must, therefore, be guarded against vigilantly.

Operative Suspension of the Prolapsed Bowel.—Of many operations designed to cure complete prolapse of the rectum, the following procedures are relatively simple. They are recommended in patients with complete prolapse who are otherwise in good health.

Lahaut's Operation.—The abdomen is opened through a left lower paramedian incision. A circular incision is made around the rectum through the peritoneum covering the pelvic floor, the prolapsed rectum is liberated and drawn into the peritoneal cavity. A cuff of pelvic peritoneum is attached to the rectum as low as possible. The second step of the operation consists of extraperitonealisation of the



FIG. 1294.—Extraperitonealisation of redundant rectosigmoid. (After J. Lahaut.)

redundant portion of the rectum and the distal portion of the sigmoid colon. This is accomplished by dissecting the medial and lateral leaves of the anterior parietal peritoneum from the abdominal wall, ligating and dividing the inferior epigastric vessels during the procedure. The peritoneal coat now being free from the anterior abdominal wall, its cut edges are sutured behind the rectum and the sigmoid (fig. 1294). The anterior rectus sheath and the skin are then closed in front of the redundant bowel. Adhesions soon develop between the exteriorised gut, the peritoneum, and the abdominal muscles. The long-term results of this operation are extremely good.

Wells' Operation.—In this operation the rectum is fixed firmly to the sacrum by inserting a sheet of polyvinyl alcohol sponge between them. The rectum is separated from the sacrum in the usual way. The sponge is fixed by a series of sutures to the periosteum over the midline of the sacrum and is then wrapped loosely about the rectum covering all except the anterior wall. The free margins of the polyvinyl sponge are sutured to the lateral margins of the anterior wall of the rectum. The peritoneal floor is resutured so that the sponge lies excluded from the peritoneal cavity. Polyvinyl sponge does not give rise to a foreign body reaction but it does produce very marked fibrous tissue formation. Many proctologists regard this as the method of choice.

ANAL INCONTINENCE

Congenital.—(a) In cases of imperforate anus, partial or complete lack of the sphincter mechanism is the exception rather than the rule; (b) patulous anus associated with mental deficiency.

Traumatic.—By far the most frequent cause is a complication of a complete perineal tear during parturition. Other injuries resulting in a torn anal sphincter are extremely rare.

Post-operative.—(a) After the ano-rectal ring has been severed during an operation for a high fistula-in-ano; (b) following conservative perineal resection of the rectum for carcinoma, an operation that cannot be recommended.

Associated with advanced complete prolapse due to atony of the sphincter.

Interruption of the reflex arc responsible for sphincter control in diseases of the nervous system, notably neuro-syphilis (e.g. tabes dorsalis).

Carcinoma of the Anus involving the Sphincter.**Treatment :**

Partial incontinence often can be remedied by the Thiersch operation (p. 999) This also is the operation of choice in cases of complete incontinence associated with mental deficiency.

Partial and often complete incontinence due to parturition is particularly amenable to operative treatment. Posterior perineorrhaphy with repair of the torn edges of the sphincter often cures the condition.

PROCTITIS

Inflammation is sometimes limited to the rectal mucosa ; in others it is associated with a similar condition in the colon (procto-colitis). The inflammation can be acute or chronic. The symptoms are tenesmus, the passage of blood and mucus and, in severe cases, of pus also. Although the patient has a frequent intense desire to defæcate, the amount of fæces passed at a time is small. Acute proctitis is usually accompanied by malaise and pyrexia. On rectal examination the mucosa feels swollen and is often exceedingly tender. Proctoscopy is seldom sufficient and sigmoidoscopy is the more valuable method of examination. Skilled pathological assistance is required to establish or exclude the diagnosis of specific infection by bacteriological examination and culture of the stools, examination of scrapings or swabs from ulcers, and serological tests. When early carcinoma cannot be excluded, biopsy is necessary.

Non-specific proctitis is an inflammatory condition affecting the mucosa, and to a lesser extent the submucosa, confined to the terminal rectum and anal canal. It is the most common variety.

Ætiology is unknown. The concept that the condition is a mild and limited form of ulcerative colitis (although actual ulceration is not present) is the most acceptable hypothesis.

Clinical Features.—The patient is usually middle-aged, and complains of slight loss of blood in the motions. Often the complaint is one of diarrhœa, but on closer questioning it transpires that usually one relatively normal action of the bowels occurs each day, although it is accompanied by some blood. During the day the patient attempts to defæcate, with the passage of flatus and a little blood-stained fæcal matter ; it is this that is interpreted as diarrhœa. On rectal examination the mucosa feels warm and smooth. Often there is some blood on the examining finger. Proctoscopic and sigmoidoscopic examination shows inflamed mucous membrane of the rectum, but no ulceration. The inflammation extends for only 5 or 6 inches (12·5 or 15 cm.) from the anus, the mucosa above this level being quite normal.

Treatment.—Although, fortunately, the condition is self-limiting, much relief may be obtained from the use of Salazopyrin, Acetarsol suppositories or Prednisolone retention enemas. Milk should be rigidly excluded from the diet and the motions kept soft with Isogel.

Ulcerative Procto-colitis.—Proctitis is present in a high percentage of cases of ulcerative colitis, and the degree of severity of the rectal involvement may influence the type of operative procedure (p. 907).

Proctitis Due to Specific Infections :

Bacillary Dysentery.—The appearance is that of an acute purulent proctitis with multiple small shallow ulcers. The examination of a swab taken from the ulcerated mucous membrane is more certainly diagnostic than is a microscopical examination of the stools. Proctological examination is painful ; agglutination tests may render it unnecessary.

Amœbic dysentery.—The infection is more liable to be chronic, and exacerbations after a long period of freedom from symptoms often occur. Proctoscopy and sigmoidoscopy are not painful. The appearance of an amœbic ulcer is described on p. 909. Scrapings from the ulcer should be immersed in warm normal saline solution and sent to the laboratory for immediate microscopical examination.

Amœbic granuloma presents as a soft mass, usually in the recto-sigmoid region. This lesion is frequently mistaken for a carcinoma. Sigmoidoscopy shows an ulcerated surface, but the mass is less friable than a carcinoma. A scraping should be taken, preferably with a small sharp spoon on a long handle, and the material sent for immediate microscopical examination, as detailed above. If doubt exists, a provocative dose of emetine may cause cysts of the amœbæ to appear in the stools. A biopsy is also required.

Amœbic granuloma of the rectum is from time to time encountered in a patient who has never visited a country in which the disease is endemic. Persons living in old people's institutions are liable to harbour this deceptive lesion.

Tuberculous proctitis is nearly always associated with active pulmonary tuberculosis, and is often complicated by a tuberculous fistula-in-ano or tuberculous ulceration of the anus. Submucous rectal abscesses burst and leave ulcers with an undermined edge (fig. 1295). A hypertrophic type of tuberculous proctitis occurs in association with tuberculous peritonitis or tuberculous salpingitis. This type of tuberculous proctitis requires biopsy for confirmation of the diagnosis.

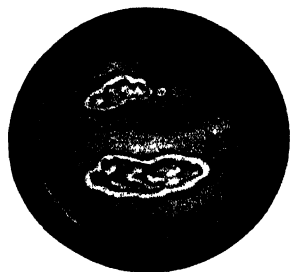


FIG. 1295.—Tuberculous ulceration of the rectum. Sigmoidoscopic appearance.

Gonococcal proctitis occurs in both sexes as the result of rectal coitus, and in the female from direct spread from the vulva. In the acute stage the mucous membrane is hyperæmic and thick pus can be expressed as the proctoscope is withdrawn. In the early stages the diagnosis can be readily established by bacteriological examination, but later, when the infection is mixed, it is more difficult to recognise. Specific treatment is so effective that local treatment is unnecessary.

Lymphogranuloma Inguinale.—The modes of infection are similar to those of gonococcal proctitis, but in the female infection spreading from the cervix uteri via lymphatics to the pararectal lymph nodes is common.

The proctological findings are similar to those of gonococcal proctitis. The diagnosis of lymphogranuloma inguinale should be strongly suspected when the inguinal lymph nodes are greatly enlarged, although the enlargement may be subsiding by the time proctitis commences. Frei's intradermal test is positive in 95 per cent. of cases, and the complement-fixation test is even more accurate (p. 28).

Primary Syphilis.—A primary chancre may occur inside the anus (p. 987).

'Strawberry' lesion of the recto-sigmoid is due to an infection by *Spirochaeta vincenti* and *Bacillus fusiformis*. The leading symptom is diarrhoea, often scantily blood-stained. Occasionally the diagnosis can be made by the demonstration of the specific organisms in the stools. More often sigmoidoscopy is required. The characteristic lesion is thickened, somewhat raised mucosa with superficial ulceration in the region of the recto-sigmoid. The inflamed mucous membrane oozes blood at numerous pin-points, giving the appearance of an over-ripe strawberry. A swab should be taken from the lesion and examined for Vincent's and fusiform organisms. Swabs from the gums and the throat are also advisable.

Treatment.—Acetarsol suppositories together with vitamin C is almost specific.

Rectal bilharziasis is caused by the *Schistosoma mansoni*, which is endemic in many tropical and subtropical countries, and particularly in the delta of the Nile.

Stage 1.—A cutaneous lesion develops at the site of entrance of the cercariæ.¹

Stage 2 is characterised by pyrexia, urticaria, and a high eosinophilia.

Both these stages are frequently overlooked.

Stage 3 is due to deposition of the ova in the rectum (much more rarely in the bladder) and is manifested by bilharzial dysentery. On examination in the late

¹ Cercariæ = a parasite of freshwater snails.

ages papillomas are frequently present. The papillomas, which are sessile or pedunculated (fig. 1296), contain the ova of the trematode, the life-cycle of which resembles that of *Schistosoma hæmatobium* (fig. 1297).

Untreated, the rectum becomes festooned, and prolapse of the diseased mucous membrane is usual. Multiple fistula-in-ano are prone to develop.

General Treatment of Bilharziasis Mansonii.—(a) *Tartar emetic* (potassium antimony tartrate) is given intravenously on alternate days, $\frac{1}{2}$ grain (30 mg.) at the first injection, increasing by $\frac{1}{2}$ grain until a dose of 2 grains (130 mg.) has been reached, the total dose being 21 grains (1,360 mg.). For this treatment the patient must be in fairly good condition, i.e. a hæmoglobin not below 60 per cent. Alternatively :



FIG. 1297.—Ovum of *Schistosoma hæmatobium* (terminal spike¹). (Dr. M. Kinawi, Cairo.)



FIG. 1296.—Bilharzial papilloma. (The late Dr H. P. Keatinge, Cairo.)

(b) *Fouadin*, *Stibophen*, or *Repodral* (all of which are preparations of antimony) is given intramuscularly. A robust adult receives a daily injection of 5 ml. for ten days. In long-standing cases the dose is reduced by half, and given on alternate days. These preparations deteriorate and should not be used after six months from the date of manufacture.

(c) *Miracil D* or *Nilodin* is given by mouth, one 200 mg. tablet t.d.s. for twenty days. These are drugs of great promise, and are safer than antimony (Halawani).

Local Treatment.—When the papillomas persist in spite of general treatment, they must be treated in the same manner as other papillomas (p. 1012).

Proctitis due to herbal enemats is a well-known clinical entity to those practising in tropical Africa. Following an enema consisting of a concoction of ginger, pepper, and tree-bark administered by a witch doctor, a most virulent proctitis sets in. Pelvic peritonitis frequently supervenes. Not infrequently a complete gelatinous cast of the mucous membrane of the rectum is extruded. Very large doses of morphine, together with streptomycin, often prevent a fatal issue if commenced early (Bowesman). Temporary colostomy is often advisable.

Treatment of severe proctitis necessitates confinement to bed. It will be appreciated that in most instances specific treatment can be given once the cause has been elucidated. The stools should be kept soft with Isogel. Local instillations of 5 ounces (150 ml.) of olive oil are soothing. Suppositories of succinyl-sulphathiazole are often beneficial. The specific treatment for the dysenteries, tuberculosis, gonorrhœa, lymphogranuloma inguinale, and syphilis are described in the appropriate sections of this book.

ANO-RECTAL ABSCESSSES

In 60 per cent. of cases the pus from the abscess yields a pure culture of *Esch. coli*; in 23 per cent. a pure culture of *Staphylococcus aureus* is obtained. In diminishing frequency, pure cultures of *Bacteroides*, a streptococcus, or *B. proteus* are found. In many cases the infection is mixed. In a high percentage of cases—some estimate it as high as 90 per cent.—the abscess commences as an infection of an anal gland (figs. 1298 and 1299). Other causes are penetration of the rectal wall, e.g. by a fish bone, a blood-borne infection, or an extension of a cutaneous boil.

A large percentage of ano-rectal abscesses result in a fistula-in-ano. This incidence can be lowered by early and adequate drainage. For this reason,

The ovum of *S. Mansonii* has a lateral spike.

Ahmed Halawani, *Contemporary*. Director of the Institute of Tropical Medicine, Cairo.
Charles Bowesman, *Contemporary*. Formerly Surgical Specialist, lately Colonial Medical Service, Kumasi, Ghana.
Theodor Escherich, 1857–1911. Professor of Pædiatrics, Vienna.

ano-rectal abscess becomes a highly important subject. Moreover, as antibiotics cannot reach the contents of an abscess in adequate concentration, no reliance can be placed on antibiotic therapy alone.

Differential Diagnosis—The only conditions with which an ano-rectal abscess is likely to be confused are an abscess connected with a pilonidal sinus, Bartholin's gland, or Cowper's gland (p. 1182).

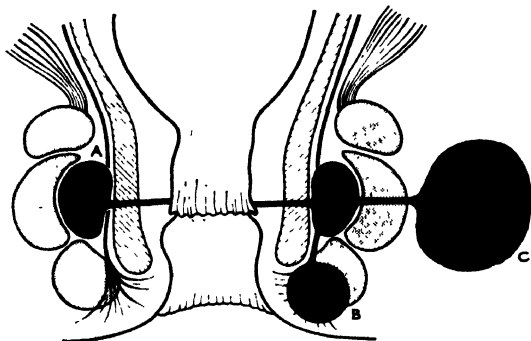


FIG. 1298.—To illustrate the spread of infection from the primary anal gland abscess (A) to the perianal region (B) and the ischio-rectal fossa (C). (After Alan Parks, F.R.C.S., London.)

Classification of Ano-rectal Abscesses.—A clear understanding of suppuration in this area is dependent on a concise knowledge of the anatomy (figs. 1298 and 1299). There are four main varieties — perianal, ischio-rectal, submucous, and pelvi-rectal.

1. Perianal (60 per cent.).

—This usually occurs as the result of suppuration in an anal gland, which spreads

superficially to lie in the region of the subcutaneous portion of the external sphincter (fig. 1299A). It may also occur as a result of a thrombosed external pile. If the hæmatoma is not evacuated, it may become infected and a perianal abscess results. This is the most common abscess of the region. Persons of all ages are affected, and the condition is not uncommon, even in infancy and childhood. The constitutional symptoms and the pain are less pronounced than in the ischio-rectal abscess because the pus can expand the walls of this part of the intermuscular space comparatively easily. Early diagnosis is made by inspecting the anal margin when an acutely tender rounded cystic lump about the size of a cherry is seen and felt at the anal verge below the dentate line (fig. 1300).

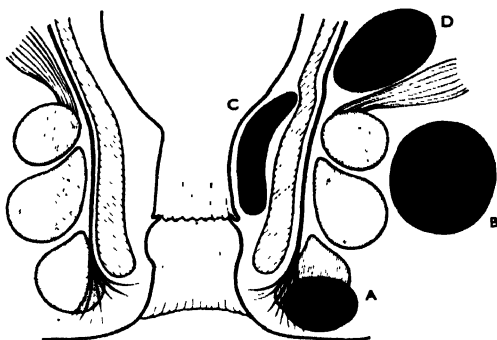


FIG. 1299.—The four types of ano-rectal abscess —(A) Perianal. (B) Ischio-rectal. (C) Submucous. (D) Pelvi-rectal. (After W. F. W. Southwood, F.R.C.S., Bath.)

Treatment.—No time should be lost in evacuating the pus.

Operation.—Thorough drainage is achieved by making a cruciate incision over the abscess and excising the skin edges—this completely removes the 'roof' of the abscess. Healing commonly occurs within a few days.

2. Ischio-rectal abscess (30 per cent.).—Commonly, this is due to an extension laterally through the external sphincter of a low intermuscular anal

abscess (fig. 1299B). Rarely, the infection is either lymphatic or blood-borne. The fat, which fills the ischio-rectal fossa (fig. 1301), is particularly vulnerable because it is poorly vascularised; consequently it is not long before the whole space becomes involved. The ischio-rectal fossa communicates with that of the opposite side via the post-sphincteric space, and if an ischio-rectal abscess is not evacuated early, involvement of the contralateral fossa is not uncommon.

An ischio-rectal abscess gives rise to a tender, brawny induration palpable on the corresponding side of the anal canal and the floor of the fossa. Constitutional symptoms are severe, the temperature often rising to 102°F . (38.9°C). Men are affected more often than women.

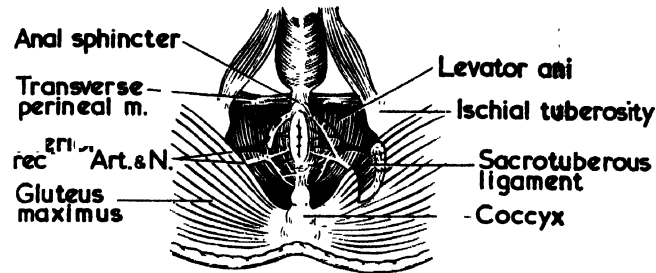


FIG. 1301.—The ischio-rectal fossa. (After V.C. Shah and Y.V. Shah.)

Operation.—*Stage 1.*—A \dagger -shaped incision (fig. 1302 inset) is made into the abscess. An adequate portion of skin that includes most, if not all, of the roof of the abscess is excised (fig. 1302). The cavity is explored and, if septa exist, they should be broken down gently with a finger and the necrotic tissue lining the walls of the abscess is removed by the finger wrapped in gauze. Nothing further is done at this stage.

Stage 2.—As soon as the acute infection has subsided, the wound should be re-examined, preferably under general anaesthesia. A careful search is made for a fistulous opening communicating with the anal canal (fig. 1303). If such is found, the treatment should be as for fistula (p. 1008). If no fistula is found, the cavity should be lightly packed with gauze wrung out in any weak antiseptic favoured by the operator. A T-bandage is applied. When the cavity has become covered with granulation tissue skin grafting expedites final epithelialisation.

3. **Submucous abscess** (5 per cent.) occurs above the dentate line (fig. 1299C). When it occurs after the injection of hæmorrhoids it always resolves. It can be opened with sinus forceps when adequately displayed by a proctoscope.

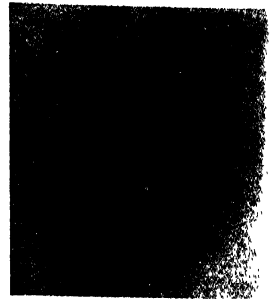


FIG. 1300.—Perianal abscess. (W. B. Gabriel, F.R.C.S., London.)

Treatment.—Operation should be undertaken early—as soon as it is certain that an abscess is present in this area—remembering that antibiotic therapy often masks the general signs.



FIG. 1302.—Excision of the skin forming the floor of the ischio-rectal fossa is an essential step in the efficient treatment of an ischio-rectal abscess.

4. **Pelvi-rectal abscess** is situated between the upper surface of the levator ani and the pelvic peritoneum. It is nothing more or less than a pelvic abscess and as such is usually secondary to appendicitis, salpingitis, diverticulitis, or parametritis. An important point to remember is that rarely it may be due to over-enthusiastic attempts to drain an ischio-rectal abscess or to display the site of a fistula resulting therefrom, when a probe is pushed through the levator ani from below (p. 1009).

5. **Fissure Abscess.**—This is the name given to a subcutaneous abscess lying in immediate association with an anal fissure (p. 986). Drainage is achieved at the same time as the fissure is treated by sphincterotomy.

FISTULA-IN-ANO

A fistula-in-ano is a track, lined by granulation tissue which opens deeply in the anal canal or rectum and superficially on the skin around the anus. It usually results from an ano-rectal abscess which burst spontaneously or was opened inadequately (fig. 1258). The fistula continues to discharge and, because of constant reinfection from the anal canal or rectum, seldom, if ever, closes permanently without surgical aid. An ano-rectal abscess may produce a sinus the orifice of which has the appearance of a fistula, but it does not communicate with the anal canal or the rectum. By definition this is *not* a fistula, but a sinus.

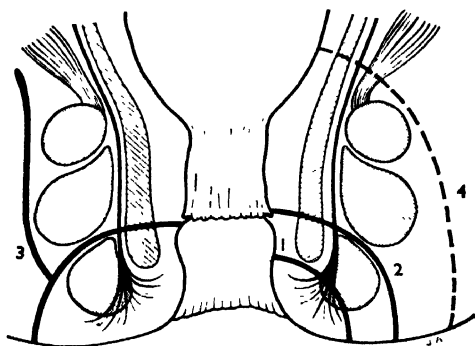


FIG. 1303.—Types of ano-rectal fistulae. *Low Level.*—(1) Submucous, (2) inter-muscular, (3) ano-rectal. (4) *High Level.*—Total fistula. (After W. F. W. Southwood, F.R.C.S., Bath.)

usually results from an ano-rectal abscess which burst spontaneously or was opened inadequately (fig. 1258). The fistula continues to discharge and, because of constant reinfection from the anal canal or rectum, seldom, if ever, closes permanently without surgical aid. An ano-rectal abscess may produce a sinus the orifice of which has the appearance of a fistula, but it does not communicate with the anal canal or the rectum. By definition this is *not* a fistula, but a sinus.

Types of Anal Fistulae.—These are divided into two groups, according to whether their internal opening is below or above the ano-rectal ring.

Low Level.—There are three varieties (fig. 1303). The ano-rectal fistula is an intermuscular fistula with a branching track leading towards the apex of the ischio-rectal fossa.

High Level.—These are rare and are commonly iatrogenic,¹ being caused by over-enthusiastic insertion of a probe up the track and through the levator ani into the rectum. (p. 1009)

LOW-LEVEL FISTULÆ

Clinical Features.—Commonly, the principal symptom is a persistent sero-purulent discharge that irritates the skin in the neighbourhood and causes discomfort. Often the history dates back for years. So long as the opening is large enough for the pus to escape, pain is not a symptom, but if the orifice is occluded pain increases until the discharge recurs. Frequently there is a solitary external opening, usually situated within $1\frac{1}{2}$ inches (3.75 cm.) of the anus, presenting as a small elevation with granulation tissue pouting from the

¹ iatro = Physician (Gk.). iatrogenic = Arising as a result of treatment.

For treating successfully Louis XIV's fistula-in-ano, Charles Félix, barber-surgeon to the Court, received a gift of a farm, 300,000 livres, and a title. 300,000 livres today would be worth about £20,000.

mouth of the opening. Sometimes superficial healing occurs, pus accumulates and an abscess reforms and discharges through the same opening, or a new opening. Thus there may be two or more external openings, usually grouped together on the right or left of the middle line, but occasionally, when both ischio-rectal fossæ are involved, an opening is seen on each side, in which case there is often intercommunication between them (fig. 1304). As a rule there is much induration of the skin and subcutaneous tissues around the fistula.

Goodsall's Rule.—Fistulæ with an external opening in relation to the anterior half of the anus tend to be of the direct type (fig. 1305). Those with an external

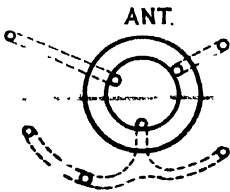


FIG. 1305.—Illustrating Goodsall's rule.

opening or openings in relation to the posterior half of the anus, which are much more common, usually have curving tracks, and may be of the horseshoe-variety.

Rectal Examination.—Not infrequently an internal opening can be felt as a nodule on the wall of the anal canal. Irrespective of the number of external openings, there is almost invariably but one internal opening.

Proctoscopy sometimes will reveal an internal opening of the fistula. A hypertrophied papilla is suggestive that the internal orifice lies within the crypt related to the papilla (fig. 1307).

Probing.—In the past it was the universal practice to probe a fistula in the ward or the out-patient department. Such manœuvres accomplish nothing, are painful, and are liable to reawaken dormant infection. Furthermore, if probing is performed without the utmost gentleness, or if the patient, experiencing pain, makes a sudden jerk, a false passage may result which complicates the condition still further. Probing should be postponed until the patient is under an anæsthetic in the operating theatre.

The injection of lipiodol, or other opaque medium, along the sinus, prior to radiography, has little to recommend it. The radiographs thus obtained are seldom illuminating, and the procedure is likely to cause a recrudescence of inflammation.

Radiography of the thorax should be undertaken and the possibility of pulmonary tuberculosis considered, despite the fact that today it will be found in only a very small proportion of patients with fistula-in-ano.

Special Clinical Types of Fistulæ-in-ano :

1. **Fistula connected with an Anal Fissure.**—Unlike the usual fistula-in-ano, pain (due to the fissure) is a leading symptom. The fistula is very near the anal orifice, usually posterior, and the external opening is often hidden by the sentinel pile (fig. 1274).

2. **Fistula with an internal opening above the ano-rectal ring** is due, almost invariably, to penetration by a foreign body or simple intraluminal incision of a high intermuscular abscess erroneously thought to be submucous.

3. **Tuberculous.**—If induration around a fistula is lacking, if the opening is ragged and flush with the surface, if the surrounding skin is discoloured and the discharge is watery, it strongly suggests that the fistula is due to a tuberculous infection. In more than 30 per cent. of patients suffering from pulmonary tuberculosis, virulent tubercle bacilli are present in the rectum. About 2 to 3 per cent. of fistulæ-



FIG. 1304.—Horseshoe fistula-in-ano. Both ischio-rectal fossæ involved. Usually there is only one internal orifice.

in-ano are tuberculous, but in sanatoria and settlements for tuberculous patients the incidence is higher. Histo-pathological examination supplies the only criterion of importance as to whether the tissue removed is tuberculous. The fistula will usually respond to anti-tuberculous drugs alone (p. 23).

4. **Fistulæ with many external openings** may arise from tuberculous proctitis, ulcerative procto-colitis, Crohn's disease of colon or ileum, bilharziasis, and lymphogranuloma inguinale with a fibrous rectal stricture. Colloid carcinoma may complicate fistulæ-in-ano.

5. **Colloid Carcinoma arising within Perianal Fistulæ.**—Colloid carcinoma of the rectum is notoriously liable to be complicated by perianal fistulæ. In some instances the fistulous condition, with its discharge of colloid material, overshadows the primary carcinoma, and not a few unfortunate patients have had their condition diagnosed for a time as an inflammatory fistula-in-ano. If a primary tumour is present in the rectum, usually it can be detected and its nature established by biopsy. Dukes has established conclusively that colloid carcinomatous fistulæ can develop without a primary neoplasm in the rectum (fig. 1306). He regards such cases as examples of colloid carcinoma developing in a reduplicated portion of the intestinal tract.

Hydradenitis Suppurativa.—This is a chronic infection of the apocrine glands around the anal margin giving rise to numerous sinuses discharging thin sero-purulent material. Clinically it looks like a relatively superficial inflammatory lesion with considerable fibrous tissue reaction. Treatment is excision of the area with skin grafting.

Treatment.—That the fistulous track must be laid open from its termination to its source was a rule promulgated by John Arderne more than 500 years ago. Today, pre-operative treatment by antibiotic and cleansing enemata is a necessity.

The operation can best be described in stages :

Step 1.—When the patient has been anaesthetised, he is placed in the lithotomy position or in the prone jack-knife position, according to the preference of the operator.

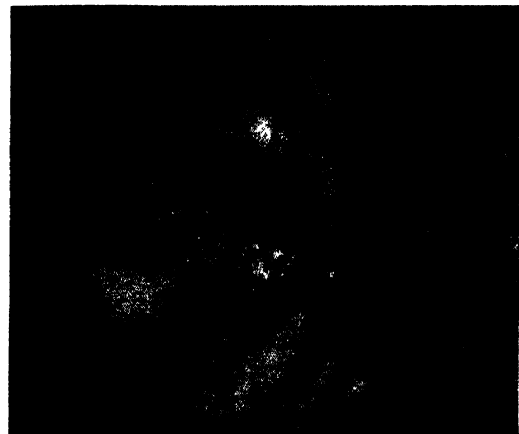


FIG. 1306.—Perianal fistulæ due to colloid carcinoma. No primary in the rectum. (C. E. Dukes F.R.C.S., London, and C. Galvin, F.R.C.S.)

Using bidigital palpation under anaesthesia, it is often possible to obtain more information concerning a fistula than can be learned from probing; it is surprisingly easy to insert a probe through the wall of the track. Unfortunately, many inexperienced operators find it more reassuring to create a false passage than to risk criticism for not being able to demonstrate the internal opening. Careful bidigital palpation of the peri-anal tissues will often reveal a cord-like induration, representing the track, which will lead the intra-anal finger towards the proximal opening. Rather than insert a probe through the distal orifice at this stage, it is better to endeavour to find the internal opening via a proctoscope. If the internal opening still cannot be seen, the insertion of a probe retrogradely into an anal crypt, especially one with a nearby hypertrophied papilla, often reveals the internal portion of the track (fig. 1307). The injection of methylene blue or other dye into the external mouth of the fistula before commencing the cutting part of the operation is not recommended, for it is unnecessary, and the result is sometimes confusing.

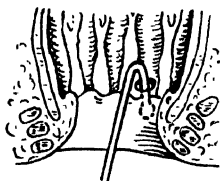


FIG. 1307.—Retrograde probing of an anal crypt sometimes reveals the internal orifice of the fistula.

Step 2.—A probe-pointed director (fig. 1308) is inserted into the distal orifice, and is advanced delicately until it reaches a point where it does not pass readily. The track is opened along the director, and bleeding is controlled.

FIG. 1308.—A director with a probe-pointed malleable extremity is a useful instrument.



Step 3.—If it is not at once evident in which direction the track passes, granulations are wiped away with gauze (it is seldom necessary to use a curette). Often this will leave a granulation-filled spot at one site only. Gentle probing at this spot frequently will give the clue to the continuation of the fistula. The director is reinserted, and again followed with the knife for a short distance. This procedure is repeated until the entire track, and any side channels, are laid open. As far as possible, all muscle is divided at right angles to its fibres. In the rare event of the track passing above the ano-rectal ring, cutting should cease at the level of the dentate line, and from thenceforth the operation is conducted as suggested below. In most instances probing and laying open the track can be repeated until the entire track is laid open. Pursuing this course, if there is no internal opening the track will become bereft of granulations on wiping it. As a rule the internal opening can be demonstrated either by direct inspection through a proctoscope, or by a bent probe inserted into an anal crypt. In the latter circumstance, the internal portion of the track is excised in continuity.

Step 4.—The edges of the track are trimmed, 1 to 3 mm. of tissue being removed—a step that makes post-operative packing unnecessary after the first twenty-four to thirty-six hours. Hughes advocates primary split skin grafting of the wound resulting from fistulotomy. The grafts are taken from the inner aspect of the thigh and applied to the anal wound, being stitched to the skin edges and to each other in the depths of the wound. Tulle gras is then superimposed, and a firm pack of cotton-wool applied. The first dressing is done on the fifth post-operative day.

When skin grafting is not employed, digital dilatation of the anus, or the passage of a St. Mark's Hospital dilator, every other day prevents pocketing or bridging of the granulating wound.

HIGH-LEVEL FISTULÆ

The treatment of these cases is difficult. If the track is laid open as for low-level fistulæ, incontinence will follow. There are three types (Parks):

(1) *Total Fistula*—secondary to local disease (type 4, fig. 1303).—It occurs as a result of Crohn's disease, ulcerative colitis, carcinoma, foreign body perforating the rectal ampulla from above, or trauma. This fistula is quite unrelated to the ordinary type and the treatment is that of the cause. A traumatic fistula usually needs a colostomy. None of these fistulæ require to be laid open, which would in any case cause incontinence.

(2) *Total Fistula with high perforating secondary track*.—This is an iatrogenic tragedy. The condition starts as an intermuscular fistula (type 2, fig. 1303) with a high secondary track in the ischio-rectal fossa up to the levator ani (type 3, fig. 1303). Here lies the danger. During exploration of the secondary track, unless great care is taken, the probe can be pushed through the levator ani into the rectal ampulla, thus converting a low fistula into a high-level type. Treatment should first of all be directed to the intermuscular fistula and healing of the upper track may follow. If it fails to do so, or if the opening into the rectum is of any size, a colostomy must be done before complete healing will take place. A seton is now rarely used for this condition as it is unsatisfactory.

(3) *High Intersphincteric Fistula*.—The track starts as a primary anal gland abscess (type A, fig. 1298), and it runs between the internal and external sphincter along the plane of the longitudinal muscle fibres. It has an opening into the rectum above the ano-rectal ring and below at the site of a perianal abscess (type B, fig. 1298). Providing it is recognised it is easy to treat. The internal sphincter is divided and the whole track is laid open without fear of incontinence.

NON-MALIGNANT STRICTURE OF THE RECTUM AND ANAL CANAL

1. Congenital :

(a) A stricture at the level of the anal valves, due to incomplete obliteration of the proctodeal membrane, sometimes does not give rise to symptoms until early childhood.

(b) Patients who have had an operation for imperforate anus in infancy may require periodic ano-rectal dilatation.

2. Spasmodic :

(a) An anal fissure causes spasm of the internal sphincter, which in time becomes fibrotic.

(b) Rarely, a spasmodic stricture accompanies Secondary Megacolon (p. 896).

3. Organic :

(a) *Post-operative stricture* sometimes follows hæmorrhoidectomy performed incorrectly, and also occurs after sleeve resection of a portion of the rectum for a neoplasm.

(b) *Irradiation stricture* is an aftermath of irradiation proctitis.

(c) *Senile Anal Stenosis*.—A condition of chronic internal sphincter contraction is sometimes seen in the aged. Increasing constipation is present with pronounced straining at stool. Fæcal impaction is liable to occur. The muscle is rigid and feels like a tight umbrella-ring. There is no evidence of a fissure-in-ano. The treatment is internal sphincterotomy with skin grafting of the bare area thus exposed.

(d) *Lymphogranuloma Inguinale* (p. 1206).—This is by far the most frequent cause of a *tubular* inflammatory stricture of the rectum, and 80 per cent. of the sufferers are women. Frei's reaction is usually positive. This variety of rectal stricture is particularly common in Negro races, and may be accompanied by elephantiasis of the labia majora.

(e) *Ulcerative Colitis*.—Stricture of the rectum also complicates ulcerative proctocolitis; in this instance the stricture is *annular*, and often more than one are present.

(f) *Endometriosis* of the recto-vaginal septum may present as a stricture. There is usually a definite history of epimenorrhœa with the recent appearance of severe pain during the first two days of the menstrual flow (p. 1013).

(g) *Neoplastic*.—When free bleeding occurs after dilatation of a supposed inflammatory stricture, carcinoma should be suspected (Grey Turner), and a portion of the stricture should be removed for biopsy. Sometimes, in these cases, repeated biopsies show inflammatory tissue only. If, however, the symptoms show a marked progression, malignancy should be strongly suspected.

Clinical Features.—Increasing difficulty in defæcation is the leading symptom. The patient finds that increasingly large doses of aperients are required, and if the stools are formed, they are ‘pipe-stem’ in shape. In cases of inflammatory stricture, tenesmus, bleeding, and the passage of muco-pus are superadded. Sometimes the patient comes under observation only when subacute or acute intestinal obstruction has supervened.

Rectal Examination.—The finger encounters a sharply defined shelf-like interruption of the lumen at a varying distance from the anus. If the calibre is large enough to admit the finger, it should be noted whether the stricture is annular (fig. 1309) or tubular. Sometimes this point can be determined only after dilatation.

Treatment :

Prophylactic.—The passage of an anal dilator during convalescence after hæmorrhoidectomy greatly reduces the incidence of post-operative stricture. Efficient treatment of lymphogranuloma inguinale in its early stages should lessen the frequency of rectal stricture from that cause.

Dilatation by Bougies.—For anal and many rectal strictures dilatation by bougies at regular intervals, combined at first with lavage, is occasionally curative. When the stricture is in the lower two-thirds of the rectum, Hegar’s dilators are suitable for this purpose. *Stricture of the upper third of the rectum should never be treated by dilatation* because of the risk of extra- or intra-peritoneal perforation of the bowel.

Incision and Primary Free Skin Graft.—For the post-operative and senile strictures this operation gives by far the best results.

The stricture is exposed and divided posteriorly so as to remove about 1 cm. of the fibrosed ring. A ‘back cut’ is made and a triangular piece of skin removed with its apex above in such a way as to ensure complete and adequate enlargement of the strictured region. A split skin graft is then taken from the inner side of the thigh, laid firmly into the defect, and sutured into position with fine catgut sutures. Greasy dressings are applied to keep it firmly in position and renewed on about the fourth day when the bowels are allowed to act. The percentage ‘take’ is high and the results surprisingly good.

Colostomy must be undertaken when a stricture is situated in the upper third of the rectum, when it is causing intestinal obstruction, and in advanced cases of stricture complicated by fistulæ-in-ano. In selected cases this can be followed by restorative resection of the stricture-bearing area. If this step is anticipated, the colostomy is placed in the transverse colon.

Excision of the rectum is required in some resistant cases due to lymphogranuloma inguinale, especially when the associated proctitis persists in spite of general treatment.



FIG. 1309.—Simple annular stricture of the rectum.

BENIGN TUMOURS OF THE RECTUM

There are two important varieties—adenoma and villous papilloma. Other types occur but are uncommon and usually insignificant. It is not

unusual to find features of the adenoma and villous papilloma in the same tumour; it is then called a papillary adenoma.

1. **Adenoma** is the commonest benign tumour of the rectum. Histologically it is composed of tubular glands similar to the glands of Lieberkühn, situated on a fibromuscular stroma.

In children it occurs as a bright-red, slightly lobulated, pedunculated tumour in the lower rectum, and is commonly termed a 'rectal polypus'. At the time of presentation the child is usually between one and six years of age, which suggests that the neoplasm commences to grow soon after birth. Nearly always the patient is brought for advice because of the passage of bright-red blood or blood-stained mucus per rectum. If the pedicle is long enough, the adenoma appears at the anus during defæcation, causing tenesmus and pain. On digital examination of the rectum a mobile, rounded lump is felt, the stalk of which can often be hooked beneath the finger, permitting the bulbous end of the tumour to be withdrawn from the anus. The treatment



FIG. 1310.—Rectal polypi seen through a sigmoidoscope.

consists of excision of the polyp and its stalk by diathermy (p. 1013).

In adults an adenoma may give rise to similar symptoms, and it can occur at any age. Often adenomas are multiple, and are found on routine proctoscopy and sigmoidoscopy in cases of rectal hæmorrhage attributed to hæmorrhoids. Such adenomas are usually sessile but may become pedunculated (fig. 1310). An adenoma of the pelvic colon not infrequently causes intussusception, and only on sigmoidoscopy is its precise origin discovered.

Differential Diagnosis.—Multiple adenomas of the rectum may be a part of *multiple colonic polyposis*, either familial or acquired (p. 917), and sigmoidoscopy, together with contrast radiography of the colon, is necessary to diagnose this condition. Children who are destined to be affected do not usually develop adenomas until between the ages of twelve and sixteen.

Patients with multiple polyposis frequently have associated osteomas and multiple sebaceous cysts. Multiple adenomatous polypi must also be distinguished from inflammatory *pseudo-polyposis* occurring as a complication of ulcerative colitis, and from those of hypertrophic tuberculosis. With the exception of those occurring in children, adenomas are prone to become *carcinomatous*. The sign of malignancy, if the tumour can be felt, is induration of its base. Probably its malignant nature will be detected microscopically after local excision. Recurrence after complete removal of an adenoma is proof that a malignant change in its base has occurred. Adult patients suffering from an adenoma of the rectum must be examined sigmoidoscopically at intervals after the tumour has been excised, in order to detect possible recurrence.

Treatment.—A solitary pedunculated adenoma situated in the lower two-thirds of the rectum is removed easily by drawing it down, ligating its base by transfixion, and dividing the pedicle with a diathermy knife. Pedunculated adenomas too high to be delivered through the anus can be removed by a diathermy snare through a sigmoidoscope. Sessile adenomas can be destroyed by a stiff insulated electrode applied through a sigmoidoscope.

2. Villous papilloma occurs in middle-aged or elderly patients. It has fine finger-like projections, viz. —————→



Histologically it consists of columnar epithelium on a fine connective tissue stroma. It is more velvety in appearance, and when within reach of the finger it feels smoother than an adenoma. Untreated, the tumour grows to a large size and sometimes encircles the rectum completely. While papillomas often eventually become carcinomatous, they may remain innocent for years. The most typical symptom is the passage of considerable amounts of clear mucus with bleeding occurring at intervals. When the tumour is large the discharge may amount to 2 or 3 litres of mucus daily. Such a profuse loss of fluid, containing a large concentration of potassium, may cause the patient to show signs of severe fluid and electrolyte depletion. All villous tumours are potential carcinomas. These growths, although less common than adenomas, have a slightly greater malignant potentiality. In 10 per cent. of specimens of carcinoma of the rectum removed by excision, the growth originated in a villous papilloma (Dukes).

Differential Diagnosis.—In patients who have lived in Egypt or any country where bilharzial infestation is rife, bilharzial papilloma must be excluded.

Treatment.—Diathermy coagulation is satisfactory in the case of a small papilloma, but the patient must be examined at regular intervals, for recurrence is common, as in the case of the bladder. For large papillomas, especially the sessile variety, excision of the rectum is the only curative treatment. Some cases (not, as a rule, those invading the anal canal) are suitable for conservative resection of the rectum.

Fibroma (*syn.* fibrous polypus) is not uncommon. *It is not a neoplasm*, but is due to fibrosis of a thrombosed hæmorrhoid.

Benign lymphoma, which occurs as a circumscribed movable nodule, firm but not hard, and greyish-white to pink in colour, is essentially submucosal. This neoplasm, which occurs at all ages and in both sexes, has no definite capsule. Notwithstanding, complete local excision is curative.

Endometrioma is not exceedingly rare, and as a rule it is diagnosed as a carcinoma. This neoplasm produces either a constricting lesion of the recto-sigmoid, or a tumour invading the rectum from the recto-vaginal septum. The latter variety gives rise to a very tender submucous elevation of the rectal wall. Endometrioma occurs usually between twenty and forty years of age; less often at the menopause. Dysmenorrhœa with rectal bleeding are the main symptoms. On sigmoidoscopy endometriosis involving the recto-sigmoid junction usually presents as a stricture with the mucous membrane intact. Should the correct pre-operative diagnosis be established by biopsy, bilateral oophorectomy is not infrequently followed by regression of the tumour, rendering resection either unnecessary, or justifying purely local excision.

Hæmangioma of the rectum, which is an uncommon tumour, is a cause of serious and, if the neoplasm is large, sometimes fatal hæmorrhage. When localised in the lower part of the rectum or anal canal, a hæmangioma can be excised after applying Goodsall's ligature (p. 998). When the neoplasm is diffuse, or lying in the upper part of the rectum, the symptoms simulate ulcerative colitis, and often the diagnosis is missed for a long period. At other times the neoplasm is mistaken for a vascular carcinoma, an error which, fortunately, is not often a cause for serious regret, because the correct treatment of an extensive hæmangioma is excision of that portion of the ano-rectum bearing the neoplasm. Lesser procedures are followed nearly always by recurrence and renewed loss of blood.

CARCINOMA OF THE RECTUM

This is the fourth most common variety of malignant tumour found in women, and its frequency in men is surpassed only by carcinoma of the bronchus and stomach.

Origin.—The carcinoma commences as a nodule of atypical columnar epithelium, the rapidly proliferating cells of which extend on the surface by exuberant growth at its edges. Similar changes occur on the deep (sub-mucosal) surface of the nodule. The less malignant varieties continue to extend towards the lumen; the more malignant varieties soon become necrotic in their centre, and give place to an ulcer with indurated, everted edges. In about 30 per cent. of cases, operation specimens show that in some part of the bowel that has been removed, in addition to the carcinoma, there are one or more adenomas or papillomas, proof indeed that adenoma and papilloma of the rectum are pre-carcinomatous conditions. In approximately 3 per cent. of cases there is more than one carcinoma present.

Pathological Histology.—Three types are recognised:

- (1) Adenocarcinoma (the most common variety);
- (2) Colloid carcinoma;
- (3) Anaplastic carcinoma.

Local spread occurs circumferentially rather than in a longitudinal direction, which rarely extends more than 1 to 2 cm. up and down the rectum. Usually a period of six months is required for involvement of one-quarter of the circumference, and eighteen months to two years for complete encirclement, the annular variety being common at the recto-sigmoid junction. After the muscular coat has been penetrated the growth spreads into the underlying fat, but is still limited by the fascia propria (peri-rectal fascia). Eventually the fascia propria is penetrated but this occurrence is rare before eighteen months from the commencement of the disease. If penetration occurs anteriorly, the prostate, seminal vesicles, or the bladder become involved in the male; in the female the vagina or the uterus are invaded. In either sex, if the penetration is lateral, a ureter may become implicated, while posterior penetration involves the sacrum and the sacral plexus.

Lymphatic Spread.—Enlargement of lymph nodes from bacterial infection is more frequent than enlargement from metastasis, and microscopical examination is required to detect carcinomatous involvement of the nodes. Lymphatic spread from a carcinoma of the rectum above the peritoneal reflexion occurs almost exclusively in an *upward* direction; below that level to within 1 to 2 cm. of the anal orifice the lymphatic spread is still *upwards*, but the first halting place is in the para-rectal lymph nodes of Gerota. The exception to this rule is when the neoplasm lies within the field of the middle rectal artery, i.e. between 4 and 8 cm. from the anus, in which case primary *lateral* spread along the lymphatics that accompany the middle rectal vein is not infrequent. *Downward* spread is exceptional, drainage along the subcutaneous lymphatics to the groins being confined, for practical purposes, to the lymph nodes draining the perianal rosette and the epithelium lining the distal 1 to 2 cm. of the anal canal.

Metastasis at a higher level than the main trunk of the superior rectal artery occurs only late in the disease. A radical operation should ensure that the high-lying lymph nodes are removed by ligating the inferior mesenteric artery and vein at the highest possible level.

Venous Spread.—As a rule, spread via the venous system occurs late, except in that portion of the anal canal where the anoderm is firmly adherent to deeper structures (fig. 1255). Anaplastic and rapidly growing tumours in younger patients are much more liable to spread in this way than tumours of relatively low malignancy. The principal sites for blood-borne metastases are: liver (34 per cent.), lungs (22 per cent.), adrenals (11 per cent.). The remaining 33 per cent. is divided among the many other locations where secondary carcinomatous deposits are wont to lodge.

Peritoneal dissemination may follow penetration of the peritoneal coat by a high-lying rectal carcinoma.

Stages of Progression.—As a rule carcinoma of the rectum does not metastasise early. Dukes classifies carcinoma of the rectum into three stages (fig. 1311).

(A) The growth is limited to the rectal wall (15 per cent.).

(B) The growth is extended to the extra-rectal tissues, but no metastasis to the regional lymph nodes (35 per cent.).

(C) There are secondary deposits in the regional lymph nodes (50 per cent.). These are subdivided into C¹ where the para-rectal lymph nodes alone are involved, and C² where the nodes accompanying the supplying blood-vessels are implicated.

This does not take into account cases that have metastasised beyond the regional lymph nodes or by way of the venous system.

Histological Grading.—In the great majority of cases carcinoma of the rectum is a columnar-celled adenocarcinoma. The more nearly the tumour cells approach normal shape and arrangement, the less malignant is the tumour. Conversely, the greater the percentage of cells of an embryonic or undifferentiated type, the more malignant is the tumour. Broders' histological classification (p. 52) into four grades has been simplified by Dukes into three grades:

Low grade = well-differentiated tumours	11 per cent.	Prognosis good.
Average grade	64 per cent.	„ fair.
High grade = anaplastic tumours	25 per cent.	„ poor.

Colloid carcinoma is present in 12 per cent. of cases. There are two forms—primary and secondary; much the more frequent is secondary mucoid degeneration of an adenocarcinoma. Histologically the glandular arrangement is preserved and mucus fills the acini. This type is of average malignancy. In a small number of cases the tumour is a primary mucoid carcinoma. The mucus lies within the cells, displacing the nucleus to the periphery, like the seal of a signet ring. Primary mucoid carcinoma gives rise to a rapidly growing bulky growth which metastasises very early and the prognosis of which is very bad. See also Colloid Carcinoma arising in a fistula-in-ano (p. 1008).

Clinical Features.—Carcinoma of the rectum is not uncommon early in life, and when the disease commences in youth, in spite of radical treatment

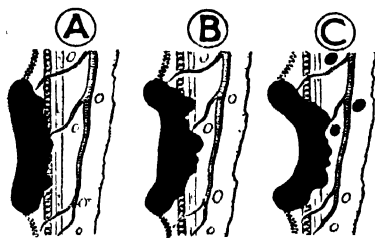


FIG. 1311.—The three cardinal stages of progression of the neoplasm. (After Cuthbert Dukes.)

death usually results within a year. Usually the early symptoms are so slight that the patient does not seek advice for six months or more.

Bleeding is the earliest and most constant symptom. There is nothing characteristic about the time at which it occurs, neither is the colour nor the amount of blood distinctive: often the bleeding is slight in amount, and occurs at the end of defæcation, or is noticed because it has stained underclothing. Indeed, more often than not, the bleeding in every respect simulates that of internal hæmorrhoids¹, and it is lamentable that, in spite of oft-repeated exhortations, the patient's doctor sometimes fails to examine the rectum and prescribes a salve while the growth advances to inoperability (footnote, p. 958).

Sense of Incomplete Defæcation.—The patient has his bowels open but feels that there is more fæces to be passed. This is a very important early symptom² and is almost invariably present in tumours of the lower half of the rectum. The patient may endeavour to empty the rectum several times a day (spurious diarrhœa), often with the passage of flatus and a little blood-stained mucus ('bloody slime').

Alteration in bowel habit is the next most frequent symptom, and the commonest deviation from normality is increasing constipation. The patient finds it necessary to start taking an aperient, or to supplement his usual dose, and as a result a tendency towards diarrhœa ensues. A patient who has to get up before the accustomed hour in order to defæcate, and one who passes blood and mucus in addition to fæces³, is usually found to be suffering from carcinoma of the rectum. Usually it is the patient with an annular carcinoma at the pelvi-rectal junction who suffers with increasing constipation, and the one with a cauliflower growth in the ampulla of the rectum with early morning diarrhœa (Bruce).

Pain is a late symptom, but pain of a colicky character accompanies advanced growths of the recto-sigmoid and is due to some degree of intestinal obstruction. When a deep carcinomatous ulcer of the rectum erodes the prostate or bladder, there is severe pain. Pain in the back, or sciatica, occurs when the growth invades the sacral plexus. Weight loss is suggestive of hepatic metastases.

Abdominal examination is negative in early cases. Occasionally when an advanced annular growth is situated at the recto-sigmoid junction, signs of obstruction to the large intestine are likely to be present. By the time the patient seeks advice metastases in the liver may be palpable (fig. 1312). When the peritoneum has become studded with secondary deposits, ascites results.

Rectal Examination.—In approximately 90 per cent. of cases the neoplasm can be felt digitally: in early cases as a plateau or as a nodule with an indurated base. When the centre ulcerates, a shallow depression will be found, the edges of which are raised and everted; this, combined with induration of the base of the ulcer, is a frequent and unmistakable finding. On bimanual

¹ Hæmorrhoids and carcinoma sometimes co-exist (fig. 1277).

² *Tenesmus* is painful straining to empty the bowels without resultant evacuation.

³ 'Early morning bloody diarrhœa.'

examination it may be possible to feel the lower extremity of a carcinoma situated in the recto-sigmoid junction. After the finger has been withdrawn, if it has been in direct contact with a carcinoma, it is smeared with blood, or mucopurulent material tinged with blood. When a carcinomatous ulcer is situated in the lower third of the rectum, involved lymph nodes can sometimes be felt as one or more hard oval swellings in the extra-rectal tissues posteriorly or posterolaterally above the tumour. In females a vaginal examination should be performed, and when the neoplasm is situated on the anterior wall of the rectum, with one finger in the vagina and another in the rectum, very accurate palpation can be carried out.

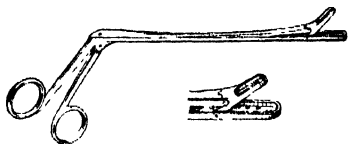


FIG. 1313.—Yeomans' biopsy forceps.

Proctoscopy in the complete knee-elbow position (fig. 1262) will nearly always show a carcinoma, if present. If

there is any doubt, a sigmoidoscopy must be done.

Biopsy.—Employing biopsy forceps (fig. 1313) by way of a proctoscope, a portion of the edge of the tumour is removed. If possible another specimen from the more central part of the growth is obtained also. Expert histological examination will not only enable the diagnosis of carcinoma to be confirmed, but the tumour can be graded as to its relative malignancy.



FIG. 1312.—Massive irregular enlargement of the liver. Rectal examination revealed a carcinomatous ulcer on the right side of the ano-rectal junction. Hard lymph node in right Scarpa's triangle. The patient also has a left inguinal hernia and a right saphenous varix.

Barium enema is not required except (a) in rare instances when sigmoidoscopy is unsatisfactory because of spasm, (b) in cases of suspected carcinoma of the pelvi-rectal junction when sigmoidoscopy fails to reveal the growth because of spasm of the bowel below it, and (c) when multiple adenomatous polyposis of the colon must be excluded.

Differential Diagnosis.—When a seemingly benign **papilloma** or **adenoma** shows evidence of induration or friability, it is certain that malignancy has occurred, even in spite of biopsy findings to the contrary. On the other hand, biopsy is invaluable in distinguishing carcinoma from an **inflammatory stricture** or an **amebic granuloma**, which simulates a carcinoma very closely. The possibility of a neoplasm being an **endometrioma** should always be entertained in patients with dysmenorrhoea. Finally, the importance of bearing in mind the possibility of a **carcinoid tumour** in atypical cases has already been emphasised. In the last four instances biopsy will frequently establish the correct diagnosis.

TREATMENT

Some form of excision of the rectum is mandatory, if at all possible, because of the extreme suffering entailed if the neoplasm remains.

Apart from co-existent disease or senile enfeeblement, the only prohibitions to excision of the rectum are widespread distant metastases and extensive peritoneal deposits. Many instances have been reported where a presumed solitary metastasis

in the liver has been resected, either at the time of excision of the rectum, or subsequently. Even when metastases in the liver are irremovable, resection of the rectum is often justifiable, and the patient may survive in comfort for a year or two.

A combined (abdominal and perineal) excision offers the best prospect of eradicating the disease. The indications and techniques of other procedures will be discussed later (p. 1020).

The combined operation can be carried out as an abdomino-perineal procedure (Miles), by which is meant that the abdominal part of the operation is undertaken first, or a perineo-abdominal (Gabriel), where the perineal stage is performed before the abdominal stage, or as a synchronised procedure, where two operating teams work simultaneously.

Five days pre-operative sterilisation of the alimentary tract by sulphasuccidine is required. The colon is cleansed by enemas or a colon wash as necessary. Blood and electrolyte deficiencies are corrected. Before commencing the operation, an indwelling catheter is inserted into the bladder.

Combined Synchronised Excision of the Rectum.—This is the operation which is now commonly performed. It combines the advantages of both the abdomino-perineal and the perineo-abdominal operations. With the patient in Trendelenburg-lithotomy position, the legs being supported in special crutches designed by Lloyd-Davies

access is afforded to the abdomen and the perineum at the same time. Two surgeons operate simultaneously, one performing the perineal dissection and the other the abdominal portion of the operation. This considerably reduces the time expended in performing the operation, and obviates turning the patient. The resectability rate is increased by this operation.



The *Abdominal Surgeon* makes a left paramedian incision, extending it well above the umbilicus. The liver and the peritoneum are examined for metastases and the degree of fixity of the growth established. The small intestine is packed away from the pelvis. A self-retaining retractor is placed in the wound and the pelvic colon freed by dividing the congenital adhesions on the left side. The peritoneum and the pelvic floor are divided with a knife by an incision which runs from the colon at the proposed site of division over the mesocolon and across the base of the bladder or near the cervix on the peritoneal floor and then upwards on the right side of the mesocolon. The peritoneum is now raised, using the points of the scissors to expose the ureters and testicular or ovarian artery. The mesocolon is now divided at the site of the proposed division on the colon and the trunk of the inferior mesenteric artery (fig. 1314) ligated and divided distal to the first branch. (Some surgeons emphasise 'flush ligation' of the artery at its origin from the aorta.) The recto-sigmoid mesentery is further divided and separated from the sacrum by blunt dissection with the fingers. In this way the sacrum is cleared almost down to the coccyx.

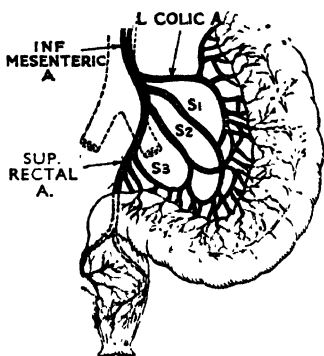


FIG. 1314.—The inferior mesenteric artery. S_1 S_2 S_3 = sigmoid branches.

The peritoneal incision anterior to the rectum is now deepened and the seminal vesicles or the vaginal wall is identified so that the fascia behind them is cleared by a dissection leading down to the prostate or perineal body. The middle rectal vessels usually lying anterior to the lateral ligaments on each side are now seized with clamps, divided and ligated. The site of division of the pelvic colon is cleared of fat and the colon divided between clamps with diathermy.

By this time the perineal surgeon working from below has mobilised the anus and the lower rectum so that the whole of the bowel below together with a clamp can be passed through the perineal wound by the abdominal surgeon. Hæmostasis over the sacrum may be difficult but it is achieved by diathermy and a hot saline pack left in position for a few minutes. The pelvic peritoneum is now united by continuous cat-gut stitches from the bladder right back over the promontory of the sacrum (fig. 1315).

The site in the R.I.F. for the colostomy is then selected. This should be equidistant from the umbilicus and the left anterior superior iliac spine and is usually sited at the linea semilunaris about 1 inch above the spino-umbilical line. A circular piece of skin and fascia, the size of a penny, is incised and this hole deepened to excise similar layers of fascia and peritoneum. The protected end of the colon with the clamp is now passed through this incision and the colostomy performed by suturing the colon to the peritoneum and the mucosa directly to the skin. If the paracolic gutter is narrow it is encircled with a running suture. This is tied when the colon has been passed through the colostomy incision—this will close the lateral space. The abdomen is closed and the layers of the incision protected from the colostomy. An adherent plastic colostomy bag is then fitted in position and the dressings are placed on the abdominal wound.



FIG. 1315.—The cut edges of the pelvic peritoneum are being united by the abdominal surgeon over the space (filled by packing) where the perineal surgeon is working. *Inset:* The rectum and pelvic colon drawn through the left iliac incision to form a terminal colostomy.

Perineal Surgeon.—When the abdominal surgeon has made certain that the condition is operable, the perineal surgeon closes the anus with purse-string sutures of stout silk. An elliptical incision between the tip of the coccyx and the central perineal point is made around the anus and deepened. The left forefinger is insinuated into the levator ani which is divided lateral to the finger first on one side and then on the other. The dissection is deepened posteriorly by incising Waldyer's fascia which is a thick condensation of pelvic fascia lying between the rectum and the sacrum. Contact is made with the abdominal surgeon. The apex of skin anterior to the anus is grasped in a hæmostat, which serves as a retractor, and by scissors and gauze dissection the wound is deepened, when the catheter within the membranous urethra will be felt. Both in the male and the female a plane of cleavage will be found between the rectum and the prostate or the rectum and the vagina, respectively. This plane having been carefully determined, the strong median raphe of the perineum is divided, after which the rectum can be stripped from the prostate or the vagina. The posterior wall of the vagina is frequently taken with the rectum. When the abdominal surgeon has cleared the rectum laterally, the whole of the anus and rectum can be drawn downwards and removed. Hæmostasis must be secured and the perineal wound closed anteriorly and posteriorly in layers around a large drainage tube. Large dressings of gauze and wool are applied over the area and a triangular bandage is used to keep the dressing in place.

After Treatment.—The patient is returned to bed, blood transfusion being continued as necessary. The catheter is connected to a closed catheter drainage system (fig. 1475) and left in for five days. It may have to be re-inserted if voluntary micturition is not re-established; carbachol may be necessary to establish this.

Reactionary hæmorrhage from the perineal wound may demand return to the theatre to open and pack the wound with gauze. The colour of the colostomy must be watched to make sure the blood supply is adequate. Small bowel obstruction may occur by herniation in the lateral space of the colostomy or through the pelvic peritoneal closure line. Discharge of urine from the perineal wound demands immediate investigation for bladder and ureteric damage.

Less Extensive Operations :

Abdominal Radical Restorative Resection (anterior resection).—In cases of carcinoma of the rectum situated above the peritoneal reflection, lymphatic spread is virtually confined to the upward paths, and wide resection of the bowel with its lymphatic field, followed by end-to-end anastomosis and preservation of the sphincter mechanism is both justifiable and highly desirable.

This apparently ideal treatment was, and still is, unpopular in many centres because it has been found that local recurrence occurred in a formidable percentage of cases. It has now been substantiated that the so-called recurrences are due, for the most part, to local implantation of free malignant cells into the distal segment that is to be preserved, the survival and rooting of these cells being favoured by disinfection of the intestine by sulphasuccidine and antibiotics. Naunton Morgan has shown that when a suitable clamp is placed at least 2 inches (5 cm.) below the neoplasm, and the rectum is irrigated with buffered Dakin's solution (Chlorinated soda-0.5 per cent available chlorine), and the lumen of the proximal end and the edges of both segments are swabbed with the same solution before anastomosis, the incidence of local recurrence is reduced considerably.

Restorative resection should be performed only when the tumour is high in the rectum, i.e. at least 4 inches (10 cm.) above the anal orifice, and even then it should not be attempted if the build of the patient, and/or the anatomy of the colon and its vascular supply, makes restoration of the continuity of the alimentary canal difficult, nor in a young subject in whom these neoplasms grow rapidly and recur.

The perineal operation (Lockhart-Mummery) does not remove the lymphatic field in relation to the inferior mesenteric artery, and it is reserved mainly as a palliative for poor-risk patients when the neoplasm is situated in the lower third of the rectum.

Hartmann's Operation.—This is an excellent procedure in an old and feeble patient who would not stand an abdomino-perineal procedure. Through an abdominal incision the rectum is excised down to within an inch of the anus, a colostomy performed and the peritoneum oversewn to cover the pelvic defect in the usual way. The cavity below can be drained through the anus, which is divided posteriorly. In the old patient, where the neoplasm is usually slow growing and spread is late, this is a most useful operation.

Palliative colostomy is indicated only in cases giving rise to intestinal obstruction, or where there is gross infection of the neoplasm. It is often possible to resect the growth later.

More Extensive Operations.—When the carcinoma of the rectum has spread to contiguous organs, the radical operation can often be extended to remove these structures. Thus in the male, where the spread is usually to the bladder, a total cystectomy and resection of the rectum can be effected. In the female the uterus acts as a barrier preventing spread from the rectum to the bladder. Accordingly, a total hysterectomy should be undertaken in addition to excision of the rectum. Should the bladder be involved, then pelvic evisceration must include that structure. Pelvic evisceration for carcinoma of the rectum is justifiable only when the surgeon is reasonably confident that the growth can be removed *in toto*. When the growth is of high-grade malignancy, the likelihood of local recurrence is so great that pelvic evisceration is contraindicated.

Pelvic Evisceration (Brunschwig's operation).—The aim is to remove all the pelvic organs, together with the internal iliac and the obturator groups of lymph

John Percy Lockhart-Mummery, 1875-1957. Surgeon, St. Mark's Hospital, London.

Robert Hartmann, 1831-1893. Professor of Anatomy, Berlin.

Alexander Brunschwig, Contemporary. Surgeon, Memorial Hospital for the Treatment of Cancer, New York.

nodes (fig. 1316). The lithotomy-Trendelenburg position facilitates the procedure, and ligation of both internal iliac arteries diminishes the blood loss. Rarely is it possible to preserve sufficient peritoneum to form a pelvic peritoneal floor, and the small intestine fills the empty pelvis. Especial care must therefore be taken to suture accurately the perineal skin, and to avoid pressure necrosis of the perineal incision by nursing the patient on alternate sides. Some form of urinary diversion is necessary (p. 1156).

Carcinoid Tumour.—Although it must be categorised as a malignant tumour, carcinoid tumour of the rectum, as far as its lethal properties are concerned, can be looked upon as a gradation between a benign tumour and a carcinoma. Formerly considered very rare, such a large number of carcinoid tumours of the rectum have been reported during recent years that a latter-day aphorism is 'keep carcinoid in mind when an atypical neoplasm of the rectum is encountered'. Like benign lymphoma, carcinoid tumour originates in the submucosa, the mucous membrane over it being intact. Consequently it seldom produces evidence of its presence in the early stages, when it appears as a small plaque-like elevation. The incidence of clinical malignancy, i.e. the occurrence of metastases, is 10 per cent. This is much less than that for carcinoid tumour of the small intestine (p. 916) but it is greater than that of carcinoid tumour of the vermiform appendix (p. 974). Multiple primary carcinoid tumours of the rectum are not infrequent. In common with carcinoid tumours elsewhere, the neoplasm is slow of progression, and usually metastasises late.

Treatment.—Resection of the rectum is advisable if the growth is more than 1 inch (2.5 cm.) in diameter, if recurrence follows local excision, or if the growth is fixed to the peri-rectal tissues. Even when metastases are present in the liver, resection may prolong life.

MALIGNANT TUMOURS OF THE ANUS

Carcinoma of the anus differs from carcinoma of the rectum in histological structure, behaviour, and the requisites for curative treatment. This is mainly because of its abundant lymph drainage, both superficial and deep (fig. 1260).

Squamous-celled Carcinoma.—

Because of its superficial situation the presence of the lesion is recognised by the patient, who often presents early. The exception is when radiation carcinoma develops in the anal and perianal skin of a patient unwisely treated with lightly filtered X-rays for pruritus ani. The chronic radiation dermatitis becomes so familiar to the patient that too often he does not perceive the superimposition of carcinoma. Simple papillomas (anal warts) sometimes take on a carcinomatous change (fig. 1317).

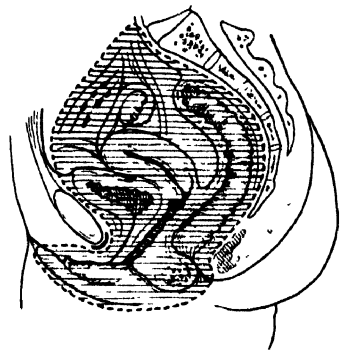


FIG. 1316.—Radical pelvic evisceration, indicating the extent of the dissection and the viscera removed.



FIG. 1317.—Neglected papillomas of the anus which have become malignant. The patient was a woman of forty-three years of age and symptoms had been present for eight years.

Basal-celled Carcinoma.—Of twenty-eight cases occurring at the Mayo Clinic, seven were basaloid small-celled carcinomas that occurred near the dentate line, and showed a high degree of malignancy. With these exceptions, basal-celled carcinomas are found predominantly at the anal margin and are of relatively low malignancy.

Melanoma.—Melanoma of the anus presents as a bluish-black soft mass that frequently has been confused with a thrombotic pile, and therefore unfortunately incised. Such trauma, followed by the trauma of defæcation, incites the tumour to rapid metastasis. Left undisturbed, it ulcerates, and the colour of the tumour changes from blue to black. The inguinal lymph nodes are soon involved. Unless a melanoma is excised at an early stage, it disseminates by the blood-stream.

Treatment :

Radiotherapy.—Either low-voltage contact X-rays or interstitial gold radon seeds, 2 millicuries each, placed 1.5 cm. apart at the base of a small tumour, should be reserved for the following types of cases (Pack):

1. Early papillary or basal-celled carcinoma of low-grade malignancy.
2. Advanced lesions that have recurred after operation.
3. In an inoperable case, with the hope of converting an inoperable carcinoma into one that is resectable.

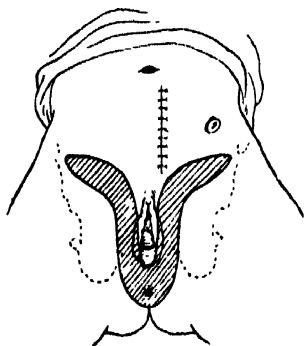


FIG. 1318.—Radical operation for carcinoma of the anus, showing the area of skin removed. In the male the lower half of the scrotum is included. (After G. T. Pack and J. C. Baldwin.)

Operation.—With the above exceptions, all cases should be treated by the radical operation described below.

Radical operation for malignant disease of the anus must be even more radical than that employed for the average case of carcinoma of the rectum. A curative operation, therefore, comprises removal of the area of skin shown in fig. 1318, together with a block dissection of the inguinal lymph nodes and a radical abdomino-perineal excision of the rectum. This operation is very formidable and causes considerable morbidity. It is usual therefore to do an abdomino-perineal excision, removing the growth and perianal area widely. If and when the inguinal lymph nodes become involved, a radical dissection of the groin is carried out. This formidable operation, which is founded on sound premises based on pathology, gives more favourable results than has been generally believed. It should be noted that dissection of the lymph nodes of the groin or groins, is carried out only when involvement of the nodes can be recognised clinically, or when a doubtful lymph node, submitted to frozen section at the time of the operation, proves to be involved. As has been emphasised already, patients with carcinoma of the anus often present early, and in many patients the inguinal lymph nodes are not involved clinically. Should they become so later, dissection of the groin is carried out.

CHAPTER 42

THE UMBILICUS AND THE ABDOMINAL WALL
(EXCLUDING HERNIA)

THE UMBILICUS

The **umbilical cord**, formed in the second month of foetal life from the body stalk, is composed of Wharton's jelly, covered by ectoderm, and contains:

1. **The allantois**, a diverticulum from the yolk sac, is a narrow tube passing from the cloaca through the umbilicus and umbilical cord to end blindly at the placenta. The part within the body forms the urachus and the bladder, except the trigone. The remainder disappears.

2. **The vitello-intestinal duct** is a tubular outgrowth of yolk sac from which the gut is derived (fig. 1319). In 98 per cent. of cases by the time the embryo is six weeks old the vitello-intestinal duct disappears (p. 1025).

3. **The Umbilical Vein**.—Originally two, early in foetal life the right one atrophies and disappears. The vein carries oxygenated blood from the placenta to the foetal liver where, having given off branches, it bifurcates (*a*) to join the left branch of the portal vein, and (*b*) to pass on as the ductus venosus to empty its contents into the inferior vena cava.

4. **The umbilical arteries** pass from the internal iliac arteries to, and through, the umbilicus, and, in a spiral fashion, traverse the umbilical cord to join the placental plexus.

The fibrous ring of the umbilicus is situated in the blended aponeurosis of the linea alba at, or below, the centre of that line. In the early days of infancy, while the umbilicus becomes a consolidated scar, the structures just referred to shrink into impervious cords (except that section of the allantois which forms part of the bladder). These cords diverge from the abdominal aspect of the umbilicus into the extraperitoneal fatty tissue, and produce peritoneal folds. Upwards, in the free edge of the falciform ligament, runs the ligamentum teres (the obliterated umbilical vein) to the longitudinal fissure of the liver. Directly downwards to the apex of the bladder runs the urachus (obliterated allantois). Obliquely downwards and outwards pass the obliterated hypogastric (umbilical) arteries to join the internal iliac arteries.

Lymphatics.—The lymphatic vessels drain into both groins and both axillæ.

A society lady wished to be re-vaccinated 'where the scar wouldn't show'. She selected an area just beneath the umbilicus. Four days later she was confined to bed with painful adenitis of both groins and both axillæ.

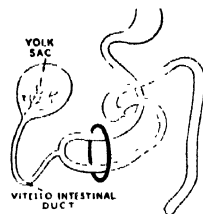
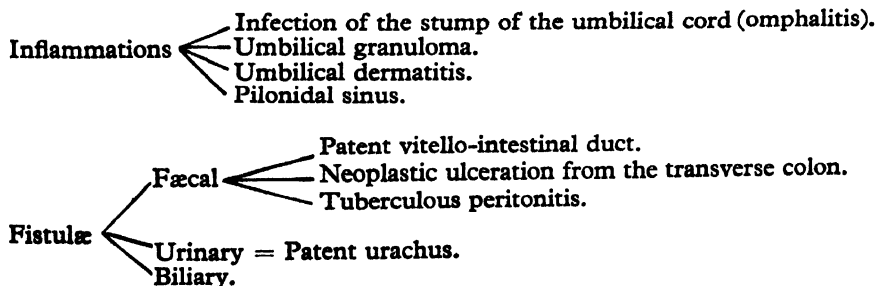
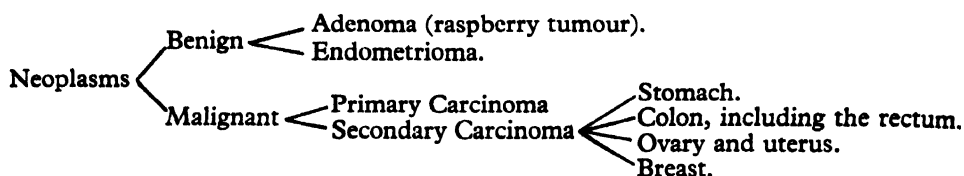


FIG. 1319.—Embryonic canal, showing the vitello-intestinal duct attached to the yolk sac.

DISEASES OF THE UMBILICUS





Hernia (p. 1051).
Umbilical calculus.

INFLAMMATIONS

Infection of the Umbilical Cord.—By the third or fourth day of life the stump of the umbilical cord is found to be carrying staphylococci in over 50 per cent. of babies born in maternity hospitals. Less commonly the stump of the cord harbours streptococci, and epidemics of puerperal sepsis in maternity hospitals have been traced to the umbilical cord of one infant in the nursery thus infected. *Esch. coli* and *Cl. Tetani* (causing neonatal tetanus) are other possible invaders. The chief prophylaxis is strict asepsis during severance of the cord, and the daily use of 1 per cent. chlorhexidine jelly.

Omphalitis.—The incidence of an infected umbilicus is much higher in communities that do not practise aseptic severance of the umbilical cord. When the stump of the umbilical cord becomes inflamed, antibiotic therapy usually localises the inflammation. By employing warm moist dressings the crusts separate, giving exit to pus. Exuberant granulation tissue requires a touch of silver nitrate. In more serious cases infection is liable to spread along the defunct hypogastric arteries or umbilical vein, when, in all probability, one or other of the following complications will supervene:

1. **Abscess of the Abdominal Wall.**—If gentle pressure is exerted below or above the navel, and a bead of pus exudes at the umbilicus, a deep abscess associated with one of the defunct umbilical vessels is present. This must be opened. A probe is passed into the sinus to determine its direction and this is followed by a grooved director on to which the skin and overlying tissues are incised in the middle line.

2. **Extensive ulceration of the abdominal wall**, due to a synergic infection, and is treated in the same way as post-operative subcutaneous gangrene (p. 1029).

3. **Septicæmia** can occur from organisms entering the blood-stream via the umbilical vein. Jaundice is often the first sign. An abscess in the abdominal wall above the umbilicus should be sought. In other respects the treatment of this grave complication follows the usual lines (p. 15).

4. **Jaundice in the Newborn.**—Infection reaching the liver via the umbilical vein may cause a stenosing intrahepatic cholangiolitis, appearing some three to six weeks after birth.

5. **Portal vein thrombosis** and subsequent portal hypertension (p. 808).

6. **Peritonitis** carries a particularly bad prognosis. If an abscess of the abdominal wall is present, it should be drained. Should peritoneal fluid accumulate it should be drained by the insertion of an intraperitoneal suprapubic drainage tube.

Umbilical Granuloma.—Chronic infection of the umbilical cicatrix which continues for weeks causes granulation tissue to pout at the umbilicus. There is no certain means of distinguishing this condition from an adenoma (p. 1026). Usually an umbilical granuloma can be destroyed by one application of a silver nitrate stick followed by dry dressings. An adenoma soon recurs in spite of these measures.

Dermatitis of and around the umbilicus is common at all times of life. Fungus and parasitic infections are more difficult to eradicate from the umbilicus than from the skin of the abdomen. Sometimes the dermatitis is

consequent upon a discharge from the umbilicus, as is the case when an umbilical fistula or a sinus is present. A deep, tender swelling in the midline below the umbilicus signifies an abscess present in the extraperitoneal fat, and is usually due to an *infected urachal remnant*. Exploration and proper drainage is necessary.

Pilonidal sinus (a sinus containing a sheaf of hairs) is sometimes encountered. It should be excised.

Umbilical calculus, often black in colour, is composed of desquamated epithelium which becomes inspissated and collects in a deep recess of the umbilicus. Eventually it gives rise to inflammation, and often a blood-stained discharge. The treatment is to dilate the orifice and extract the calculus, but, to prevent recurrence, it may be better to excise the umbilicus.

UMBILICAL FISTULA

The umbilicus being a central abdominal scar, it is understandable that a slow leak from any viscus is liable to track to the surface at this point (fig. 1320). Added to this, very occasionally, the vitello-intestinal duct or the urachus remains patent; consequently it has been remarked aptly that the umbilicus is a creek into which many fistulous streams may open.

For instance, an enlarged inflamed gall-bladder perforating at its fundus may discharge gall-stones through the umbilicus. Again, an unremitting flow of pus from a fistula at the umbilicus of a middle-aged woman led to the discovery of a length of gauze overlooked during hysterectomy five years previously.

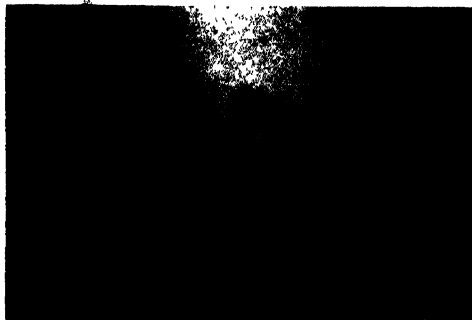


FIG. 1320.—This patient, aged sixty-two, complained only of a purulent discharge from the umbilicus. A lump could be felt in the position outlined. Perforated carcinoma of the transverse colon.

The vitello-intestinal duct occasionally persists, and gives rise to one of the following conditions :

1. It remains patent (figs. 1321 and 1322 (a)). The resulting umbilical fistula discharges mucus and, rarely, fæces.



FIG. 1321.—Patent vitello-intestinal duct opening into the umbilicus. (After A. L. Taylor.)

(b) A small portion only of the duct near the umbilicus remains unobliterated. This gives rise to a sinus that discharges mucus. The epithelial lining of the sinus often becomes everted to form an adenoma (p. 1026).

2. Sometimes both the umbilical and the intestinal ends of the duct close, but the mucous membrane of

the intervening portion remains, and an intra-abdominal cyst develops (fig. 1322 (c)).

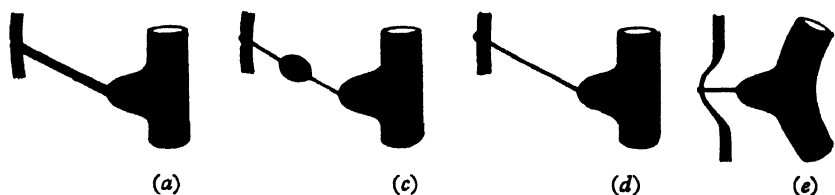


FIG. 1322.—Anomalies connected with the vitello-intestinal duct : (a) umbilical fistula ; (b) umbilical sinus (not illustrated) ; (c) intra-abdominal cyst ; (d) intraperitoneal band ; (e) Meckel's diverticulum adherent to the sac of a congenital umbilical hernia.

3. With its lumen obliterated or unobliterated, the vitello-intestinal duct provides an intraperitoneal band (fig. 1322 (d)) which is a potential danger, for intestinal obstruction is liable to occur. The obstruction results from a coil of small intestine passing under or over (fig. 1323), or becoming twisted around, the band.



FIG. 1323.—Vitello-intestinal duct causing intestinal obstruction.

4. Such a band may contract and pull a Meckel's diverticulum into a congenital umbilical hernia (fig. 1322(e)).

5. A vitello-intestinal cord connected to Meckel's diverticulum, but not attached to the umbilicus, becomes adherent to, or knotted around, another loop of small intestine, and so causes intestinal obstruction.

6. Sometimes a band extending from the umbilicus is attached to the mesentery near its junction with a distal part of the ileum. In this case the band is probably an obliterated vitelline artery, and is not necessarily associated with a Meckel's diverticulum.

Treatment.—A patent vitello-intestinal duct should be excised, together with a Meckel's diverticulum, if such be present, preferably when the child is about six months old. When a vitello-intestinal band gives rise to acute intestinal obstruction, after removing the obstruction by division of the band, it is expedient, when possible, to excise the band and to bury the cut ends.

A patent urachus seldom reveals itself until maturity, or even old age. This is because the contractions of the bladder commence at the apex of the organ and pass towards the base. A patent urachus, because it opens into the apex of the bladder, is closed temporarily during micturition, and so the potential urinary stream to the umbilicus is cut off. Therefore the fistula remains unobtrusive until a time when the organ is overfull, usually due to some form of obstruction.

Treatment.—Usually treatment is directed to removing the obstruction to the lower urinary tract. If, after this is remedied, the leak continues or a cyst develops in connection with the urachus, umbilectomy and excision of the urachus down to its insertion into the apex of the bladder, with closure of the latter organ, is indicated.

Abscess in a urachal remnant, causing persistent umbilical dermatitis and discharge, is described on p. 1025.

NEOPLASMS OF THE UMBILICUS

Umbilical adenoma or Raspberry tumour is commonly seen in infants (fig. 1324), but only occasionally later in life. It is due to a partially

(occasionally a completely) unobliterated vitello-intestinal duct. Mucosa prolapsing through the umbilicus gives rise to a raspberry-like tumour, which is moist with mucus and tends to bleed.

Treatment.—If the tumour is pedunculated, a ligature is tied around it, and in a few days the polypus drops off. Should the tumour reappear after this procedure, umbilectomy is indicated. Sometimes a patent vitello-intestinal duct, or more often a vitello-intestinal band, will be found associated with a Meckel's diverticulum. The Meckel's diverticulum and the attached cord or duct should be excised at the same time as the umbilicus. Histologically, the tumour at the umbilicus consists of columnar epithelium rich in goblet cells.

Endometrioma occurs in women between the ages of twenty and forty-five. On histological examination it is found to consist of endometrial glands occupying the same plane in the dermis as the sudoriferous glands, and opening on to the surface in the same way. The umbilicus becomes painful and bleeds at each menstruation, when the small fleshy tumour between the folds of the umbilicus becomes more apparent. Occasionally an umbilical endometrioma is accompanied by endometriomas in the uterus or ovary. When, as is usually the case, the tumour is solitary, umbilectomy will cure the condition.

Secondary carcinoma at the umbilicus¹ (fig. 1325) is not very uncommon, but is always a late manifestation of the disease. The primary neoplasm is often situated in the stomach, colon or ovary, but a metastasis from the breast, probably transmitted along the lymphatics of the round ligament of the liver, is sometimes located here.



FIG. 1325.—Secondary nodule at the umbilicus in a case of carcinoma of the stomach.

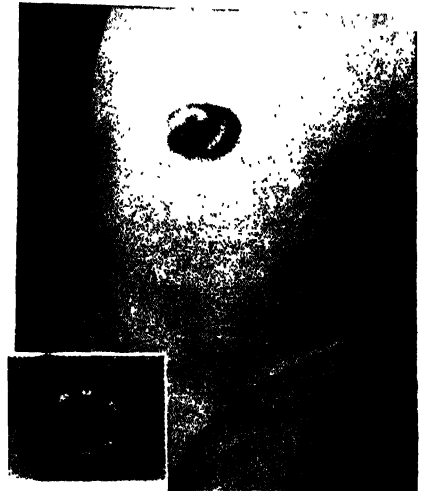


FIG. 1324.—Adenoma (raspberry tumour) of the umbilicus.

THE ABDOMINAL WALL

Tearing of the inferior epigastric artery occurs in three dissimilar types of individual, viz. elderly women, often thin and feeble; athletic, muscular men, usually below middle age; and pregnant women, mainly multiparae late in pregnancy. The site of the hæmatoma is usually at the level of the arcuate line, where the posterior sheath of the rectus abdominis is lacking.

¹ The neoplastic nodule so caused is known as Sister Joseph's nodule. Sister Joseph of the Mayo Clinic imparted this clinical observation to the late Dr. William Mayo.

Clinical Features.—The possibility of tearing of the epigastric vessels should always be considered when, following a bout of coughing, or a sudden blow to the abdominal wall, an exquisitely tender lump appears in relation to the rectus abdominis (fig. 1326). Occasionally, a hæmatoma occurs within the muscles lateral to the rectus sheath. Unless there is bruising of the overlying skin the diagnosis may be difficult.



FIG. 1326.—Tearing of the inferior epigastric artery, showing the tell-tale swelling and bruising (see text). (Dr. C. L. Colton, Bristol.)

Differential Diagnosis.—The conditions for which the hæmatoma is frequently mistaken are, in the female, a twisted ovarian cyst, and in both sexes, when the lump is on the right side, an appendix abscess. The sign most likely to be of value in differentiating a hæmatoma of the abdominal wall from these conditions, namely tensing the abdominal musculature, is often unsatisfactory because of the pain it causes. Again, the differential diagnosis between the hæmatoma and a strangulated Spigelian hernia (p. 1058) may be difficult. The absence of vomiting suggests a hæmatoma, and the presence of

resonance over the swelling favours a Spigelian hernia, while a plain radiograph of the abdomen sometimes gives positive evidence of the latter.

As a Complication of Pregnancy.—Rupture of the inferior epigastric artery occurs occasionally during pregnancy. Surprising to relate, the hæmorrhage into this closed space from this comparatively small artery has proved fatal.

Treatment.—With rest, a comparatively small hæmatoma may resolve, but sometimes renewed hæmorrhage causes the hæmatoma to rupture into the peritoneal cavity. Therefore it is safer to operate early, evacuate the clot, and ligate the artery.

BURST ABDOMEN (*syn.* ABDOMINAL DEHISCENCE)

In 1 to 2 per cent. of cases a laparotomy wound disrupts and viscera are everted. The peak incidence of the catastrophe is between the sixth and eighth days after operation. The predisposing causes are infection, persistent cough, abdominal distension, leakage of pancreatic enzymes, hypoproteinaemia, delayed healing in anæmia and malignancy, and the too early removal of deep sutures, which should remain in place for twelve to fourteen days.

It is interesting and instructive to note that upper abdominal incisions disrupt more frequently than lower abdominal incisions, and that the suture material employed appears to have no bearing on the incidence of the disaster. Surgeons who use stainless steel wire sutures have the same incidence as those employing catgut, cotton, silk, or nylon (Long).

In most cases dehiscence of the deeper layers occurs some days before the wound actually bursts asunder; the peritoneal stitches snap or become untied within the first three days of operation, though often it is the peritoneum at the site of the suture line which tears. Sometimes the damage is done while the patient is coming round from the anæsthetic; a most potent factor in this respect is the violent coughing reflex set up if an endotracheal tube is withdrawn while the patient is but lightly anæsthetised.

Prophylaxis.—Great care should be taken in suturing the anterior layer of the rectus sheath—by interrupted sutures if there is any predisposing cause of dehiscence. The insertion of deep tension sutures of nylon is an additional insurance. When infection of the wound has supervened, or in

other conditions in which it is thought that the stitches are liable to give way, the abdominal wall should be supported by 'corsets' of adhesive plaster. In debilitated and aged subjects wound healing is retarded, so vitamins, particularly vitamin C, and a high protein diet are indicated.

Clinical Features.—A serosanguineous (pink) discharge from the wound is a forerunner of disruption in fully 50 per cent. of cases. It is the most pathognomonic sign of impending wound disruption, and it signifies that intraperitoneal contents are lying extraperitoneally. The patient often volunteers the information that he "felt something give way". If skin sutures have been removed, omentum or coils of intestine may be forced through the wound and will be found lying on the skin. Pain and shock are usually absent, but there may be symptoms and signs of intestinal obstruction.

Treatment.—An emergency operation is required to replace the bowel, relieve any obstruction, and to resuture the wound. While awaiting operation, reassure the patient and cover the wound with a sterile towel. The stomach is emptied by a gastric tube and intravenous fluid therapy commenced.

Operation.—Each protruding coil of intestine is washed gently with saline solution, and returned to the abdominal cavity. Then protruding greater omentum is treated similarly and spread over the intestine. The abdominal wall having been cleansed, all layers are approximated by through-and-through sutures of braided nylon or strong silk. The abdominal wall is supported by strips of adhesive plaster encircling the anterior two-thirds of the circumference of the trunk. Antibiotic therapy is started.

Contrary to what might be thought, peritonitis rarely supervenes and, though the skin wound becomes infected, healing is satisfactory. A second dehiscence rarely occurs.

INFECTIONS

Cellulitis can occur in any of the planes of the abdominal wall.

Superficial cellulitis is usually discovered when an abdominal wound is inspected following pyrexia. The earliest sign is when the stitches become embedded in the oedematous skin. Later there is a blush extending for a variable distance from the incision or the stitch holes. On palpation with the gloved hand usually one area is found to be more indurated and tender than the remainder. A stitch should be removed from the immediate vicinity, and if pus or sero-pus escapes it is sent for bacteriological examination, meanwhile one of the tetracyclines is administered.

Deep cellulitis is characterised by brawny oedema towards one or both flanks, and not infrequently of the scrotum or vulva as well. Antibiotic therapy is the mainstay of treatment. When tenderness persists, an incision dividing the muscles carefully, layer by layer, until pus or purulent fluid is encountered, is often advisable.

Progressive post-operative bacterial synergistic gangrene is fortunately a rare complication after laparotomy, usually for a perforated viscus (notably perforated appendicitis). It has also occurred after gall-bladder operations and even after drainage of an empyema thoracis. The condition is due to the synergic action of microaerophilic non-hæmolytic streptococci and, usually, a staphylococcus. The skin

in the immediate vicinity of the wound exhibits signs of cellulitis. Within a few days a central purplish zone with an outer brilliant red zone can be distinguished, and the whole region is extremely tender. Soon the purplish area becomes gangrenous and looks like suede leather. Slowly the condition advances (fig. 1327). The gangrenous skin liquefies, exposing underlying granulation tissue. If the condition persists, the general condition deteriorates.



FIG. 1327.—Bacterial synergistic gangrene of the chest and abdominal wall.

Treatment.—Identification of the organisms and a report on their sensitivity to antibiotics is essential. The usual antibiotics may be of little avail. Bacitracin can be effective (10,000 to 20,000 units I.M., every six hours with local application of 500 units per ml., 4 times daily). Without vigorous and effective treatment the gangrene spreads to the flanks and the patient may die of toxæmia. Hyperbaric oxygen (p. 80), if available, can be a useful ancillary treatment.

NEOPLASMS OF THE ABDOMINAL MUSCULATURE

Desmoid tumour is a tumour arising in the musculo-aponeurotic structures of the abdominal wall, especially below the level of the umbilicus. It is a completely unencapsulated fibroma, and is so hard that it creaks when it is cut. Some cases recur repeatedly in spite of apparently adequate excision.

Ætiology.—Eighty per cent. of cases occur in women, many of whom have borne children, and the neoplasm occurs occasionally in scars of old hernial or other abdominal operation wounds. Consequently, trauma—e.g. the stretching of the muscle fibres during pregnancy or possibly a small hæmatoma of the abdominal wall—appears to be an ætiological factor.

Pathology.—The tumour is composed of fibrous tissue containing multinucleated plasmodial masses resembling foreign-body giant-cells. Usually of very slow growth, it tends to infiltrate muscle in the immediate neighbourhood. Eventually it undergoes a myxomatous change: it then increases in size more rapidly. Metastasis does not occur. Unlike fibromata elsewhere, no sarcomatous change occurs.

Treatment.—Unless the tumour is excised widely, with a surrounding margin of at least 2·5 cm. of healthy tissue, recurrence commonly takes place. After removal of a large tumour, repair of the defect in the abdominal wall by tantalum gauze or nylon mesh (p. 1056) is required. These tumours are moderately radio-sensitive.

Fibrosarcoma of the abdominal wall is rare. It is resistant to radiotherapy and only in some cases can a wide excision, with tantalum or nylon net repair, offer hope of a cure.

Adenocarcinoma of the colon (fig. 1320) or of other viscera may invade the abdominal wall. In such cases the resection of this extension, along with the primary growth, may require special repair of the resulting defect.

CHAPTER 43

HERNIA

A HERNIA is the protrusion of a viscus or part of a viscus through an abnormal opening. The external abdominal hernia is the commonest form (fig. 1328).

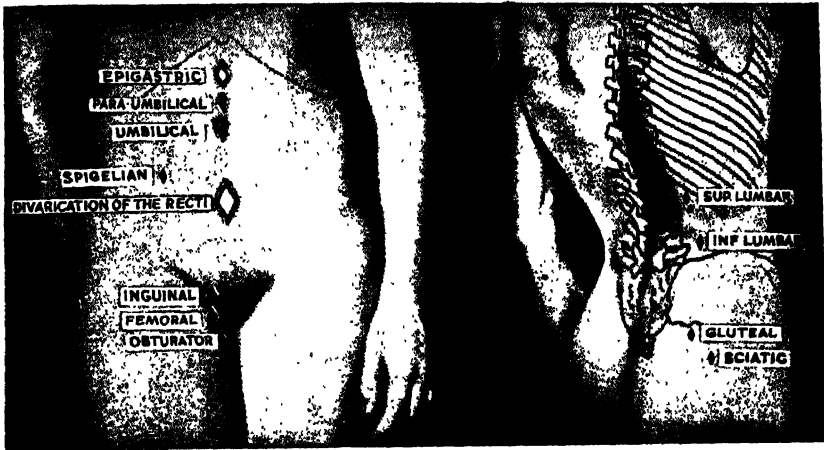


FIG. 1328.—External herniæ. Red = common. White -- not unusual. Black = rare.

At least 2 per cent. of male inhabitants of Great Britain have a hernia (Keith), the common types being inguinal, femoral, and umbilical. The most frequent of these is inguinal which occurs in 73 per cent. of cases; then comes femoral, in 17 per cent. of cases, followed by umbilical, which occurs in 8·5 per cent. This leaves 1·5 per cent. for the rarer forms. Post-operative incisional hernia has not been included.

Aetiology.—In most cases no cause can be elucidated. It is probable that indirect inguinal herniæ occur in a congenital preformed sac—the remains of the processus vaginalis (Hamilton Russell). Sometimes a powerful muscular effort or strain occasioned by lifting a heavy weight will cause such a hernia. Any condition which raises intra-abdominal pressure is liable to be followed by a hernia. Whooping cough is a predisposing cause in childhood, whilst a chronic cough favours the appearance of a hernia in an adult. Straining on micturition because of urethral obstruction and straining on defæcation¹ may precipitate a hernia.

Stretching of the abdominal musculature because of an increase in contents as in obesity and in pregnancy, can be another factor. Fat acts as a kind of 'pile-driver' for it separates muscle bundles and layers, weakens aponeuroses, and favours the appearance of paraumbilical, direct inguinal, and hiatus herniæ.

¹ If a patient over forty presents with recent hernia one should enquire particularly as to bowel habits, for rectal and colonic carcinoma can be brought to light in this way.

Sir Arthur Keith, 1866–1955. Curator of the Museum, Royal College of Surgeons of England.
Hamilton Russell, 1860–1933. Surgeon, Alfred Hospital, Melbourne.

Pathological Anatomy.—As a rule, a hernia consists of three parts—the sac, the contents of the sac, and the coverings of the sac.

The sac consists of a diverticulum of peritoneum which is divided into a mouth, neck, body, and fundus (fig. 1329). Usually the neck is well-defined, but in certain direct inguinal herniæ and in many incisional herniæ there is no actual neck. The body of the sac varies greatly in size and is not necessarily occupied. In cases occurring in infancy and childhood the sac is more delicate than the parietal peritoneum with which it is continuous. In old-standing cases, especially after years of pressure by a truss, the wall of the sac is comparatively thick and even (in places) of cartilaginous consistency.

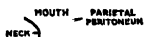


FIG. 1329.—The various parts of a hernial sac.

Contents.—The most common contents of a hernial sac are one or more of the following:

1. Omentum = omentocele (*syn.* epiplocele).
2. Intestine = enterocele. Usually small intestine, but, in some instances, large intestine or the vermiform appendix.
3. A portion of the circumference of the intestine = Richter's hernia.
4. A portion of the bladder, or a diverticulum of the bladder, is sometimes present in addition to other contents in a direct inguinal, a sliding inguinal, and in a femoral hernia.
5. Ovary with or without the corresponding Fallopian tube.
6. A Meckel's diverticulum = Littre's hernia.
7. Fluid. As a part of ascites, or as a residuum thereof. Blood-stained fluid accompanies strangulation.

Coverings are derived from the layers of the abdominal wall through which the sac passes. In long-standing cases they become atrophied from stretching and so amalgamated that they are indistinguishable one from another.

Classification.—A fundamental classification of herniæ, irrespective of their site, is as follows:

- | | |
|------------------------------|-------------------------|
| 1. Reducible. | |
| 2. Irreducible. | (Complication of 1.) |
| 3. Obstructed ¹ . | } (Complications of 2.) |
| 4. Strangulated. | |
| 5. Inflamed. | |

REDUCIBLE HERNIA

The hernia either reduces itself when the patient lies down, or can be reduced by the patient or by the surgeon. The physical signs of reduction vary somewhat with the nature of the contents of the sac. *Intestine* gurgles on reduction, and the first portion is more difficult to reduce than the last. *Omentum* is doughy, and the last portion is more difficult to reduce than the first.

¹ The term 'incarcerated' should be reserved for impaction of fæces within large intestine.

August Gottlieb Richter, 1742–1812. Surgeon, Göttingen, Germany.
 Gabriele Fallopio, 1523–1562. Professor of Anatomy, Surgery and Botany, Padua.
 Johann Friedrich Meckel (The Younger), 1781–1833. Professor of Anatomy and Surgery, Halle.
 Alexis Littre, 1668–1726. Surgeon and Unofficial Teacher of Anatomy, Paris. Littre described 'Meckel's' diverticulum in a hernial sac long before Meckel was born.

IRREDUCIBLE HERNIA

A hernia is said to be irreducible when its contents cannot be returned to the abdomen, and there is no evidence of other complications. Usually such a condition is brought about by adhesions between the sac and its contents or from overcrowding within the sac. Irreducibility without other symptoms is almost diagnostic of an omentocoele. Femoral and umbilical herniæ are most often thus complicated. An inguinal hernia is not often irreducible, but in long-standing cases it is sometimes only partially reducible. Any degree of irreducibility predisposes to strangulation.

OBSTRUCTED HERNIA

An obstructed hernia is an irreducible hernia containing intestine the lumen of which is obstructed from without or from within; but there is no interference to the blood-supply to the bowel. The symptoms are less severe and the onset more gradual than is the case in strangulation, but more often than not the obstruction culminates in strangulation. Usually no clear distinction can be made between obstruction and strangulation in herniæ, so the safe course is to assume that strangulation is imminent and treat the case accordingly.

Incarcerated Hernia.—The term ‘incarceration’ is often used loosely as an alternative to obstruction or strangulation. As emphasised already, the term ‘incarceration’ should be employed only when it is considered that the lumen of that portion of the *colon* occupying a hernial sac is blocked with fæces. In that event the scybalous contents of the bowel should be capable of being indented with the finger, like putty.

STRANGULATED HERNIA

A hernia becomes strangulated when the blood-supply of its contents is seriously impaired, rendering gangrene imminent. Gangrene may occur as early as five or six hours after the onset of the first symptoms of strangulation.

Although inguinal hernia is four times more common than femoral hernia, a femoral hernia is more likely to strangulate.

Pathology.—The intestine is obstructed, and in addition its blood supply is constricted. At first only the venous return is impeded. The wall of the intestine becomes congested and bright red, and serous fluid is poured out into the sac. As the congestion increases, the intestine becomes purple in colour. As a result of increased intestinal pressure the strangulated loop becomes distended, often to twice its normal diameter. As venous stasis increases, the arterial supply becomes more and more impaired. Ecchymoses appear under the serosa. Blood is effused into the lumen of the loop, and also through the serosa, causing the fluid in the sac to become blood-stained. The shining serosa becomes dull and covered by a fibrinous, sticky exudate. By this time the walls of the intestine have lost their tone; they feel flabby, and are very friable. The lowered vitality of the intestine favours migration of bacteria through the intestinal wall, and the fluid in the sac teems with bacteria. Gangrene appears at the rings of constriction

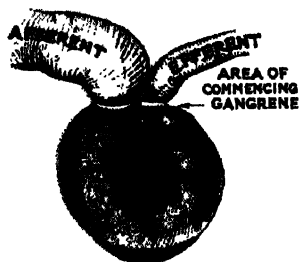


FIG. 1330.—Gangrene commences at the areas of constriction and then at the antimesenteric border.

(fig. 1330), which become deeply furrowed and grey in colour. Also from the anti-mesenteric border gangrene spreads upwards, and the colour changes, which vary from black to grey or green, are due to decomposition of blood in the subserosa. If the strangulation is unrelieved, perforation of the wall of the intestine occurs, either on the convexity of the loop or at the seat of constriction. The mesentery involved in the strangulation becomes congested and hæmorrhagic, and thrombosis of its vessels occurs. Finally it, too, becomes gangrenous. Peritonitis spreading from the sac to the

peritoneal cavity is the usual terminal event.

Clinical Features.—Pain comes on suddenly, and is at first situated over the hernia. Generalised abdominal pain soon supervenes; it is paroxysmal in character and is often located mainly at the umbilicus. Vomiting is forcible and usually oft-repeated. The patient often says that the hernia has recently become larger. On examination, the hernia is *tense*, extremely *tender*, and there is *no expansile impulse on coughing*.

The patient is seriously ill and treatment is vitally urgent. Unless the strangulation is relieved, the paroxysms of pain continue until peristaltic contractions cease with the onset of gangrene when paralytic ileus (often the result of peritonitis) develops. Spontaneous cessation of pain is therefore of grave significance.

Strangulated Richter's Hernia (*syn.* Strangulated Partial Enterocoele).—A portion of the circumference of the intestine is affected (fig. 1331). The condition, which usually complicates femoral hernia (p. 1047), and, rarely, an obturator hernia, is particularly dangerous as operation is frequently delayed because the clinical features mimic gastro-enteritis. The local signs of strangulation are often not obvious, the patient may not vomit, or vomits only once or twice. Intestinal colic occurs, but the bowels are often opened normally or there may be diarrhœa; absolute constipation (p. 929) is delayed until paralytic ileus supervenes. For these reasons gangrene of the knuckle of bowel and peritonitis often have occurred before operation is undertaken.

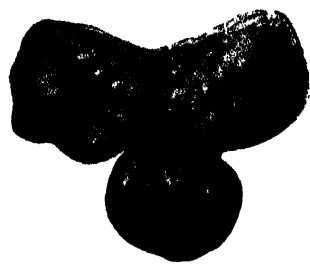


FIG. 1331.—Gangrenous Richter's hernia from a case of strangulated femoral hernia.

Strangulated Omentocoele.—The initial symptoms are in general similar to those of strangulated bowel, but the recurring attacks of generalised abdominal pain are not maintained. Vomiting and constipation may be absent. Unlike intestine, omentum can exist on a very meagre blood-supply. The onset of gangrene is therefore correspondingly delayed, and it occurs first in the centre of the fatty mass. Unrelieved, a bacterial invasion of the dying contents of the

sac will almost certainly occur. Infection is limited to the sac for days, and sometimes for weeks. In an inguinal hernia infection usually terminates as a scrotal abscess, but extension of peritonitis from the sac to the general peritoneal cavity is always a possibility.

INFLAMED HERNIA

Inflammation can occur from external causes, such as pressure from an ill-fitting truss, or from inflammation of contents within the sac, e.g. acute appendicitis or salpingitis. The hernia is tender but *not* tense, and the overlying skin becomes red and œdematous. If it is considered the condition is due to inflammation of an organ within the sac, herniotomy is necessary in order to deal with the cause.

INGUINAL HERNIA

SURGICAL ANATOMY

The **superficial inguinal ring** is a triangular aperture in the aponeurosis of the external oblique, and lies 1.25 cm. ($\frac{1}{2}$ inch) above the pubic tubercle. The ring is bounded by a superomedial and an inferolateral crus joined by criss-cross intercrural fibres. Normally the ring will not admit the tip of the little finger (fig. 1332D).

The **deep inguinal ring** lies 1.25 cm. above the inguinal (Poupart's) ligament, midway between the symphysis pubis and the anterior superior iliac spine. It is a U-shaped condensation of the transversalis fascia, incomplete above (fig. 1332A₃). The competency of the deep inguinal ring depends upon the integrity of this fascia.¹

The **Inguinal Canal**.—In infants the superficial and deep inguinal rings are almost superimposed, and the obliquity of this canal is slight. In adults the inguinal canal, which is about 3.75 cm. ($1\frac{1}{2}$ inches) long, is directed downwards and medially between the superficial and deep inguinal rings. In the male the inguinal canal transmits the spermatic cord, the ilio-inguinal nerve, and the genital branch of the genito-femoral nerve. In the female the round ligament replaces the spermatic cord.

Boundaries of the Inguinal Canal.—The best way to understand these is to study these diagrams (viewing the canal from the deep to the superficial layers):

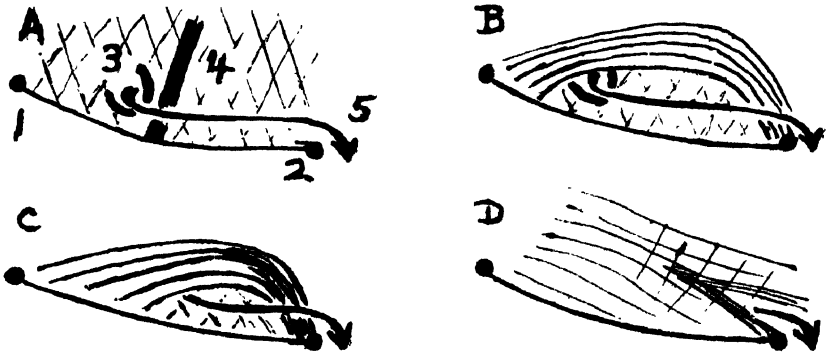


FIG. 1332.—Sketches of the boundaries of the right inguinal canal. A. The deepest layer. Hatched area = transversalis fascia. The inguinal ligament passes between (1) the anterior superior iliac spine and (2) the pubic tubercle. (3) = The U-shaped internal ring with the inferior epigastric artery (4) lying medially. (5) = The direction of the spermatic cord. B. The transversus muscle arches above the internal ring to join C. The internal oblique to form the conjoined tendon behind the cord. D. The external oblique aponeurosis, showing the crura of the external ring and the intercrural fibres.

Thus, the boundaries of the inguinal canal are as follows:

Posteriorly.—**Internal oblique** (here conjoined tendon); inferior epigastric artery; fascia transversalis (see A and B).

¹ The transversalis fascia is the 'fascial envelope' of the abdomen.

Anteriorly.—External aponeurosis; fibres of **internal oblique** (see C and D).

Superiorly.—**Internal oblique** (see C).

Inferiorly.—**Inguinal ligament** (see A, B, C and D).

An indirect inguinal hernia travels down the canal on the outer (lateral and anterior) side of the spermatic cord. A direct hernia comes out directly forwards through the posterior wall of the inguinal canal. While the neck of the indirect hernia is lateral to the inferior epigastric vessels, the direct hernia usually emerges medial to this except in the saddle-bag or pantaloony type, which has both a lateral and a medial component (p. 1043). An inguinal hernia can be differentiated from a femoral hernia by ascertaining the relation of the neck of the sac to the medial end of the inguinal ligament and the pubic tubercle, i.e. in the case of an inguinal hernia the neck is above and medial, while that of a femoral hernia is below and lateral (fig. 1355).

INDIRECT (*syn.* OBLIQUE) INGUINAL HERNIA

An indirect inguinal hernia is the most common of all forms of hernia. Many subscribe to the belief that, at whatever age it appears, it occurs into a preformed sac which is a partially or completely patent processus vaginalis. Normally, shortly before birth, the processus vaginalis becomes obliterated, at first at the deep inguinal ring, and a little later immediately above the upper pole of the epididymis; the tunnel of peritoneum between these two points becomes a fibrous cord.

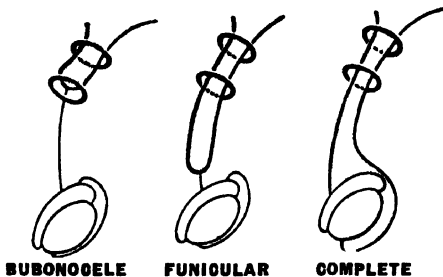


FIG. 1333.—Types of oblique inguinal hernia.

In the first decade of life inguinal hernia is more common on the right side in the male. This is no doubt associated with the later descent of the right testis (p. 1215). After the second decade left inguinal herniæ are as

frequent as right. The hernia is bilateral in nearly 30 per cent. of cases.

There are three types of oblique inguinal hernia (fig. 1333):

1. **Bubonocoele.**¹—The hernia is limited to the inguinal canal, the processus vaginalis having been obliterated at the superficial inguinal ring. This type of hernia is seen commonly in young adults; the history is usually a short one.

2. **Funicular.**²—The processus vaginalis is closed only at its lower end, just above the epididymis. When the sac is occupied, the contents of the sac can be felt separately from the testis, which lies below the hernia. The funicular variety is common in adults who give a history of inguinal hernia of some standing.

3. **Complete** (*syn.* Scrotal).—There is a persistence of the prenatal condition before the processus vaginalis becomes obliterated; nevertheless a complete inguinal hernia is rarely present at birth. Commonly encountered in infancy, it may not appear until adolescent or adult life. The testis appears to lie within the lower part of the hernia.

Clinical Features.—An oblique inguinal hernia can appear at any age. Males are twenty times more commonly affected than females.

In the early stages of the development of the hernia when the sac is still limited to the inguinal canal (bubonocoele), the diagnosis presents some difficulty. Often the patient complains of pain in the groin or pain referred

¹ Bubon. Gr. = the groin. Bubo = an enlarged lymph node in the groin or axilla.

² Funiculus. L. = a small cord, or funnel.

to the testicle when he is performing heavy work, or taking strenuous exercise. The patient is asked to cough and a small transient bulging may be seen and felt together with an expansile impulse. Often the bulge may be better seen by observing the inguinal region from the side or even looking down the abdominal wall while standing slightly behind the respective shoulder of the patient.

When an oblique inguinal hernia has become larger it produces a swelling that appears at first intermittently. In these circumstances the swelling often becomes apparent when the patient coughs, and it persists (fig. 1334) until it is reduced, usually by the act of lying down.¹ Local pain is unusual unless complications have occurred.

As time goes on the hernia comes down as soon as the patient assumes the upright position. In large herniæ (fig. 1335) there is a sensation of weight, and dragging on the mesentery may produce epigastric pain. If the contents of the sac are reducible, the inguinal canal will be found to be commodious.

In infants the swelling appears when the child cries. An inguinal hernia may be translucent in infancy and early childhood, but never in an adult.

Differential Diagnosis in the Male

(a) *A vaginal hydrocele.* (b) *An encysted hydrocele of the cord.* (c) *Spermatocele.* (d) *A femoral hernia* (p. 1047). (e) *An incompletely descended testis inguinal canal.* An inguinal hernia is often associated with this condition. (f) *A lipoma of the cord.* This is often a difficult, but unimportant, diagnosis. It is usually not settled until the parts are displayed by operation.

Differential Diagnosis in the Female

(a) *A hydrocele of the canal of Nuck* (fig. 1336) is the commonest differential diagnostic problem. (b) *A femoral hernia.*



FIG. 1334.—Oblique left inguinal hernia which became apparent when the patient coughed, and persisted until it was reduced.



FIG. 1335.—Bilateral oblique inguinal herniæ which have descended into the scrotum.



FIG. 1336.—Hydrocele of the canal of Nuck. The swelling is irreducible, but brilliantly translucent.

¹ *Notes on the clinical examination.*—The clinician is seated in front of the patient who stands with his legs apart. He is instructed to look at the ceiling and to cough at the ceiling. If the hernia will come down it usually does. The examiner looks for the impulse and feels for the impulse and then satisfies himself on the following points: (1) Is the hernia right, or left, or bilateral? (2) Is it an inguinal or a femoral hernia (see p. 1048)? (3) Is it a direct or an indirect inguinal hernia? (4) Is it reducible or irreducible? (patient has to lie down for this to be ascertained). (5) Is the inguinal hernia incomplete (bubonocoele) or complete (scrotal)? (6) What are the contents — bowel (enterocoele), or omentum (omentocoele) or epiplocele?

TREATMENT OF INDIRECT INGUINAL HERNIA

A Truss: A truss is used when operation is contraindicated because of cardiac, pulmonary (fig. 1337), or other systemic disease, or when operation is refused. The hernia must be reducible. A rat-tailed spring truss (fig. 1338) with a perineal band to prevent the truss slipping will, with due care and attention, control a small or moderate-sized inguinal hernia. A truss must be worn continuously during waking hours, kept clean, and in proper repair, and renewed when it shows signs of wear. It should be applied before the patient



FIG. 1337.—Accoutrements of a hernia patient, in which operation is contraindicated because of asthma. Left: rat-tailed truss. Right: asthma inhaler.

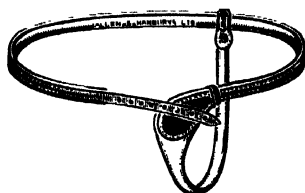


FIG. 1338.—Rat-tailed truss.

gets up and while the hernia is reduced. A properly-fitting truss controls the hernia when the patient stands with his legs wide apart, stoops, and coughs violently. However, as Farquharson stresses, often the pad of a truss is seen to lie to the side of the swelling, where the pressure it exerts is both useless and harmful. In these circumstances the truss is a menace, for it increases the risk of strangulation.

For a large irreducible hernia a bag truss is sometimes employed, but as a rule operation should be urged in cases of irreducibility, because of the danger of strangulation.

Infants and Trusses.—Special inflatable rubber trusses, formerly popular for the control of an infant's hernia are rarely used. Provided urgent admission is not required for sudden irreducibility, the parents are advised to wait until operation is performed when the infant is three months old.

Operative Treatment

Operation is undoubtedly the treatment of choice. Contraindications and the use of a truss are mentioned above. It is important to remember that patients who have a bad cough from chronic bronchitis should not necessarily be denied operation, for these are the very people who are in danger of getting a strangulated hernia. Operation can, if necessary, be undertaken in adults, using spinal, epidural or local anæsthesia.

The types of operation are usually classified as (1) *Herniotomy*, (2) *Herniorrhaphy* and *Hernioplasty*.

Inguinal Herniotomy.—This is the essential basic operation and it entails dissecting out and opening the hernial sac, reducing any contents and

then tying off the neck of the sac and removing the remainder. It is employed either by itself or as the first step in herniorrhaphy or hernioplasty. By itself it is sufficient for the treatment of hernia in infants, adolescents and young or fit adults who have good inguinal musculature. In fact, any attempts at repair (herniorrhaphy or hernioplasty) in such cases are meddlesome, and do more harm than good.

In infants it is not necessary to open the canal, as the internal and external rings are superimposed. Excellent results are obtained. The operation should be done in the morning and the child allowed home in the evening. Usually there is no need for the child to stay in hospital. 'The best nurse is the mother.'

Inguinal Herniorrhaphy (and Hernioplasty).—This operation consists of (1) excision of the hernial sac (herniotomy) plus (2) repair of the stretched internal inguinal ring (transversalis fascia) and (3) reconstruction of the posterior wall of the inguinal canal by suturing and darning, or by the use of fascial sutures, fascial flaps, tantalum wire or Dacron or Teflon net implants.

Operative Procedures

(1) Excision of the Hernial Sac (Herniotomy)

An incision¹ is made in the skin and subcutaneous tissues 1.25 cm. ($\frac{1}{2}$ inch) above and parallel to the medial two-thirds of the inguinal ligament. In large irreducible herniæ the incision is extended into the upper part of the scrotum. After dividing the superficial fascia, the external oblique aponeurosis and the superficial inguinal ring are identified. The external oblique aponeurosis is incised in the line of its fibres, so as to open the inguinal canal. The structures beneath the external oblique aponeurosis are separated from its deep surface before completing the incision into the superficial inguinal ring, which is divided. In this way the ilio-inguinal nerve is safeguarded. By blunt dissection the upper leaf of the external oblique aponeurosis is separated from the internal oblique. The lower leaf is likewise dissected until the inner aspect of the inguinal ligament is seen. The cremasteric muscle fibres are divided longitudinally to open up the subcremasteric space and display the spermatic cord, which is then hooked up on the forefinger.

Excision of the sac. The sac is easily distinguished as a pearly white structure lying on the outer side of the cord and, when the internal spermatic fascia has been incised longitudinally, it can be dissected out and then opened between hæmostats.

Variations in Dissection.—If the sac is small (e.g. bubonocoele) it can be freed *in toto*. If it is of the long funicular or scrotal type, or is extremely thickened and adherent (the result of wearing a truss), the fundus need not be sought. The sac is cut across in the inguinal canal. Care must be taken to avoid damage to the vas and the spermatic artery, including the blood supply to the epididymis.

An adherent sac can be separated from the cord by first injecting saline through its posterior wall. A similar tactic is used when dissecting the gossamer sac of infants and children (p. 1216).

Reduction of Contents.—Intestine or omentum is returned to the peritoneal cavity. Omentum is often adherent to neck or fundus of the sac; if to the neck, it is freed, and if to the fundus of a large sac, it may be transfixed, ligated and cut across at a suitable site. The distal part of omentum, like the distal part of a large scrotal sac, can be left *in situ* (the fundus should not, however, be tied off).

Isolation and Ligation of the Neck of the Sac.—Whatever type of sac is encountered, it is necessary to free its neck by blunt and gauze dissection until the parietal peritoneum can be seen on all sides. Only when the extraperitoneal fat is encountered and the

¹ Prior to the skin incision in large inguino-scrotal hernias, the usual antiseptic preparation of the skin should not be extended to the perineal aspect of the scrotum, for, by so doing, severe bacterial contamination of the operation site is likely.

inferior epigastric vessels are seen on the medial side has the dissection reached the required limit. If it has not been done already, the sac is opened. The finger is passed through the mouth of the sac in order to make sure that no bowel or omentum is adherent. The neck of the sac is transfixed and ligated as high as possible and the sac is excised 1.25 cm. ($\frac{1}{2}$ inch) below the ligature.

(2) Repair of the Stretched Internal Ring (Lytle) (fig. 1339).

This is an important stage in the operation and must never be omitted. The transversalis fascia is plicated with unabsorbable sutures, the most lateral of these displacing the cord laterally as much as possible and narrowing the diameter of the internal ring to the size of half a finger-tip.

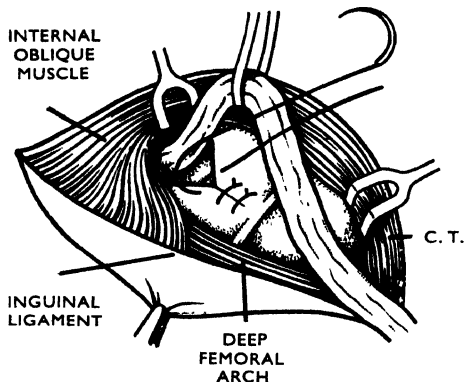


FIG. 1339.—Repair of the internal inguinal ring with lateral displacement of the cord. (After F. S. A. Doran, F.R.C.S., Bromsgrove.) C.T. Conjoined Tendon.

Lateral displacement of the stump of the sac. Frequently, the surgeon displaces the stump of the sac laterally beneath the arching fibres of the internal oblique. A large curved round-bodied needle, threaded with each of the ends of the ligature used for the transfixion and closure of the neck of the sac, is passed through the lateral fibres of the muscle from within outwards, and tied without tension.

Completion of Operation.—If possible the cremasteric muscle should be reconstituted; the external oblique is directly sutured or overlapped leaving a new external ring which will accommodate the tip of the little finger.

(3) Reconstruction of a weak Posterior Wall of the Inguinal Canal

What to do with a large indirect hernia with a neck of over 10 cm., or a direct inguinal hernia, is often a problem. All are agreed that the first step is the careful repair of the transversalis fascia with lateral displacement of the cord (above), and that the second step, reinforcement of the posterior wall, must be accomplished without tension.

The First Step.—Repair the stretched transversalis fascia as well as circumstances permit, not forgetting to take the repair as far laterally as possible, and narrowing the remnants of the internal ring. Even in a direct hernia the internal ring needs attention.

The Second Step.—Two main types of herniorrhaphy are possible:

(a) Reconstruction of the posterior wall with a flap of rectus sheath, hinged on the linea semilunaris and sewn to the inguinal ligament (Bloodgood) (fig. 1340).

Should further reinforcement be required, the external oblique aponeurosis is overlapped behind the spermatic cord, thus directing this structure into the subcutaneous fat at the site of the repaired internal ring (Halstead and Keynes modifications).

(b) Reinforcement of the posterior wall by a two-layer darn, using monofilament nylon (Moloney), steel wire, floss nylon (Maingot) (fig. 1341), or a strip of fascia lata (Gallie) (fig. 1342), or by using sheets of foreign material, e.g., Tantalum gauze (fig. 1343), Dacron net, etc.

Bassini's operation and the rectus sheath relaxing incision (fig. 1344).—Bassini's method of reinforcing the posterior wall of the inguinal canal by suturing the conjoint muscle and tendon to the inguinal ligament, behind the spermatic cord, has

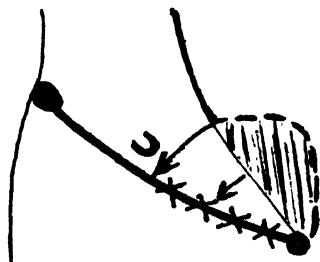


FIG. 1340.—Bloodgood's operation (see text).

William James Lytle, *Contemporary*. Consulting Surgeon, Royal Infirmary, Sheffield.
 Joseph Colt Bloodgood, 1867–1935. Surgeon, Johns Hopkins Hospital, Baltimore, U.S.A.
 William S. Halstead, 1852–1922. Surgeon, Johns Hopkins Hospital, Baltimore, U.S.A.
 Sir Geoffrey Langdon Keynes, *Contemporary*. Consulting Surgeon, St. Bartholomew's Hospital, London.
 George Edward Moloney, *Contemporary*. Surgeon, The Radcliffe Infirmary, Oxford.
 Rodney Maingot, *Contemporary*. Surgeon, Royal Free Hospital, London.
 William Edward Gallie, 1882–1959. Professor of Surgery, University of Toronto, Canada.
 Edoardo Bassini, 1844–1924. Professor of Surgery, University of Padua.

FIG. 1341.—The two-layer darn of the posterior wall of the inguinal canal. The darning is conducted from the neighbourhood of the pubic tubercle to the deep inguinal ring and back to the starting point. The darning is kept fairly loose, and it forms a lattice upon which fibrous tissue is laid down. The external oblique aponeurosis is reunited either in front or behind the cord. Meticulous asepsis is essential. The long-term results of the operation are good and the recurrence rate is low. It is a very satisfactory operation for the repair of direct inguinal hernia. Floss nylon is being used. (After Rodney Mamgot.)

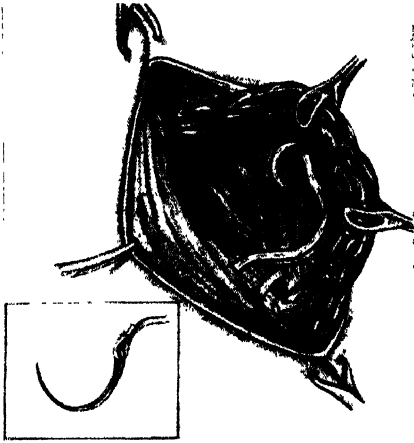


FIG. 1342.—Darn using fascia. In Gallie's graft, a strip of fascia lata 1.25 cm. ($\frac{1}{2}$ inch) wide is obtained from the outer side of the thigh. This can be obtained by a long incision, or by means of a fasciotome which cuts a strip from the fascia lata through a small skin incision. Inset: Method of attaching strip of fascia to the needle, which has especially large eye (Gallie's needle).

FIG. 1343.—Tantalum gauze or Dacron net reinforcement. The gauze is attached to the inguinal ligament and the coverings of the pubis below, and a slot is cut to accommodate the spermatic cord laterally. Usually the external oblique is united over the gauze but under the cord. In this figure the gauze has been used to reinforce a Bassini operation with a rectus sheath relaxation incision (see text). (After A. R. Koontz.)

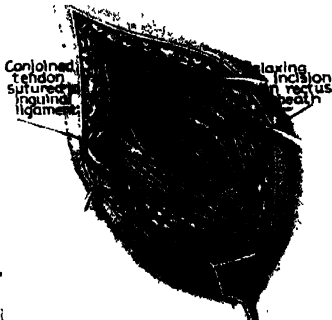
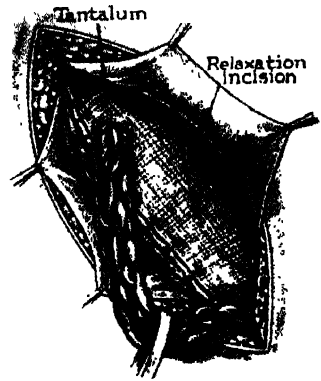


FIG. 1344.—Bassini's operation with relaxing incision. The conjoined muscle and tendon is approximated to the inguinal ligament. (After W. L. Estes Jr.)

been widely practised for many years. To obtain good results the conjoint muscle and tendon must be well developed and the space between it and the inguinal ligament must be narrow enough to permit approximation without tension. A relaxing incision in the rectus sheath (e.g. Tanner's slide) is necessary to achieve this essential requirement. Unabsorbable sutures, e.g. braided nylon, are employed. The external oblique aponeurosis is reunited in front of the cord.



FIG. 1345.—A direct hernia pushes through Hesselbach's triangle which is bounded medially by the outer margin of the rectus abdominis, laterally by the inferior epigastric artery, and below by the medial half of the inguinal ligament.

DIRECT INGUINAL HERNIA

Between 10 and 15 per cent. of inguinal herniæ are direct. Over half of the herniæ are bilateral.

A direct inguinal hernia is always acquired. The sac passes through a weakness or defect of the transversalis fascia in the posterior wall of the inguinal canal. In some cases the defect is small and closely related to the insertion of the conjoint tendon (occasionally congenitally deficient), while in others there is a generalised bulge through Hesselbach's triangle (fig. 1345). Often the patient is a man with poor abdominal musculature, as shown by the presence of Malgaigne's bulgings (fig. 1346). Women practically never develop a direct inguinal hernia (Brown). Pre-

disposing factors are a chronic cough, straining, and heavy work.

Direct herniæ rarely attain a large size and descend into the scrotum. In contradistinction to an oblique inguinal hernia, a direct inguinal hernia lies behind the spermatic cord. A finger inserted into the superficial inguinal ring passes directly backwards into the abdomen. On coughing the impulse is felt on the pulp of the finger whereas in an oblique hernia the impulse is felt on the finger-tip, viz. —————→

The inferior epigastric artery lies lateral to the aperture, but because of its small size and the nature of its coverings, it cannot be felt. "Those who pretend to feel it surrender themselves to a flattering delusion" (Macready).

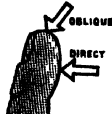


FIG. 1346.—Malgaigne's bulgings.

At operation the distinguishing features of a direct inguinal hernia are that the sac lies medially to the inferior epigastric artery, and the spermatic cord is not attached to the wall of the sac. The sac is often smaller than the hernial mass would indicate, the protruding mass mainly consisting of extraperitoneal fat. As the neck of the sac is wide, direct inguinal herniæ rarely strangulate.

Injury to the ilio-hypogastric or ilio-inguinal nerves during the gridiron operation for appendicitis appears to be associated with the development of a right inguinal hernia, usually, but not necessarily, of the direct variety.

Treatment.—For herniæ that are enlarging, and those in comparatively young adults, especially when the patient is engaged in a strenuous occupation, operation must be advised. A *small* direct inguinal hernia is not necessarily an indication for operation, as usually it is symptom free and the risk of strangulation is very small.

Operation for Direct Hernia.—The principles of repair of direct hernia are the same as those of an indirect hernia with the exception that the hernial sac need not be removed. The sac, after it has been dissected free from surrounding structures, is inverted into the abdomen and the transversalis fascia repaired in front of it. Some form of reconstruction of the posterior wall of the inguinal canal completes the operation. Fascial flaps, fascial or nylon dams, and tantalum gauze and Dacron net implantations are all popular (pp. 1040-42).

Funicular direct inguinal hernia (*syn.* *prevesical hernia*) is a narrow-necked hernia with prevesical fat and a portion of the bladder that occurs through a small oval defect in the medial part of the conjoined tendon just above the pubic tubercle. It occurs principally in elderly males, and occasionally it becomes strangulated. Unless there are definite contraindications, operation should always be advised.

Dual (*syn.* **Saddle-bag; Pantaloon Hernia.**)—Here there are two sacs which straddle the inferior epigastric artery (fig. 1345), one sac being medial and the other lateral to this vessel. The condition is not a rarity, and is a cause of recurrence, one of the sacs having been overlooked at the time of operation.



FIG. 1347.—Bilateral direct inguinal herniæ.

STRANGULATED INGUINAL HERNIA

Strangulation of an inguinal hernia occurs at any time during life, and in both sexes. Oblique inguinal herniæ strangulate commonly; the direct variety but rarely owing to the wide neck of the sac. Sometimes a hernia strangulates on the first occasion that it descends; more often strangulation occurs in patients who have worn a truss for a long time, and in those with a partially reducible or irreducible hernia.

In order of frequency, the constricting agent is: (a) the neck of the sac; (b) the external abdominal ring in children; (c) rarely adhesions within the sac.

Contents.—Usually small intestine is involved in the strangulation; the next most frequent is omentum; sometimes both are implicated. For large intestine to become strangulated in an inguinal hernia is of the utmost rarity, even when the hernia is of the sliding variety (p. 1046).

Pathological and Clinical Features are described on pp. 1033-35.

Strangulation during Infancy.—The incidence of strangulation is high, and the ratio of females to males is 5:1. In most cases of strangulated inguinal hernia occurring in female infants the content of the sac is an ovary, or an ovary plus its Fallopian tube.



FIG. 1348.

Maydl's hernia (*syn.* Hernia-in-W) (fig. 1348) is rare. The strangulated loop of the W lies within the abdomen, thus local tenderness over the hernia is not marked. At operation two comparatively normal-looking loops of intestine are present in the sac. After the obstruction has been relieved, the strangulated loop will become apparent if traction is exerted on the middle limbs of the loops occupying the sac.

Treatment of Strangulated Inguinal Hernia.—The treatment of strangulated hernia is usually by *emergency operation*.

If dehydration and collapse are present, intravenous fluid replacement (p. 89) and gastric aspiration for one to three hours are invaluable. It is absolutely essential to make sure that the stomach is emptied just before commencing the anæsthetic. The passing of a large-bore stomach tube is the best way of preventing vomiting, drowning, and cardiac arrest during the induction. The bladder must also be emptied, if necessary by a catheter.

Inguinal Herniotomy for Strangulation.—An incision is made over the most prominent part of the swelling. The external oblique aponeurosis is exposed, and the sac, with its coverings, is seen issuing from the superficial inguinal ring. In all but very large herniæ it is possible to deliver the body and fundus of the sac together with its coverings and (in the male) the testis on to the surface. Each layer covering the anterior surface of the body of the sac near the fundus is incised, and if possible it is stripped off the sac. The sac is then incised, the fluid therein is mopped up or aspirated very thoroughly, for it may be highly infected. The external oblique aponeurosis and the superficial inguinal ring are divided. Returning to the sac, a finger is passed into the opening, and employing the finger as a guide, the sac is slit up along its length. If the constriction lies at the superficial inguinal ring or in the inguinal canal, it is readily divided by this procedure. When the constricting agent is at the deep inguinal ring, by applying hæmostats to the cut edge of the neck of the sac and drawing them downwards, and at the same time retracting the internal oblique upwards, it may be possible to continue slitting up the sac over the finger beyond the point of constriction. When the constriction is too tight to admit a finger, a grooved director is inserted and the neck of the sac is divided with a hernia knife in an upward and inward direction, i.e. parallel to the inferior epigastric artery, under vision. Once the constricting agent has been divided, the strangulated contents can be drawn down. Devitalised omentum is excised after being securely ligated, by interlocking stitches if bulky. Viable intestine is returned to the peritoneal cavity. Doubtfully viable and gangrenous intestine is dealt with as described on p. 933. If the hernial sac is of moderate size and can be separated easily from its coverings, it is excised and closed by a purse-string suture. When the sac is large and adherent, much time is saved by adopting the principle described for obviating excision of the body of a scrotal hernia (p. 1039). Having tied off the neck of the sac, if the condition of the patient permits, a simple form of herniorrhaphy can be carried out.

Conservative Measures.—These may be instituted during preparation for operation in patients with a previously reducible hernia which has been strangulated for less than four hours, or in an infant, less than six hours. In elderly patients gangrene of the contents of a strangulated hernia frequently occurs early and with few signs. Conservative measures are therefore unsafe in this age group.

Conservative measures consist of:

1. Nursing in a warmed bed to promote muscular relaxation.
2. Elevation of foot of bed to reduce congestion.—
3. Administration of a full dose of premedication e.g. omnopon and scopolamine. Infants are given chloral hydrate.

4. Half an hour after the foregoing the hernia is reviewed. In a few instances the hernia will have reduced spontaneously. In a number of others reduction is easily obtained by *gentle taxis*: the thigh is flexed and internally rotated, in order to relax the pillars of the external ring. The fundus is supported with one hand, while the contents at the neck of the sac are kneaded with the fingers of the other hand, so that the contents which were last extruded are the first to be reduced. In these



patients emergency operation is avoided and elective herniorrhaphy is performed (usually on the next operation list). Alternatively, a pad and spica bandage¹ are applied, and the patient can then arrange a convenient time for operation, or be fitted with a truss if operation is contraindicated.

The same principles are used in the case of an infant. The child is given a sedative and then slung to a Balkan beam² or to the bedrail by his feet (the judgment of Solomon position) for no longer than three hours. In 75 per cent. of cases reduction is effected, and there appears to be no danger of gangrenous intestine being reduced (Irvine Smith).

N.B.—Vigorous taxis has no place in modern surgery, and is mentioned only to be condemned. Its dangers include:

1. Contusion or rupture of the intestinal wall.
2. Reduction-en-masse (fig. 1349). 'The sac together with its contents, is pushed forcibly back into the abdomen; and as the bowel will still be strangulated by the neck of the sac, the symptoms are in no way relieved' (Treves).
3. Reduction into a loculus of the sac.
4. The sac may rupture at its neck and its contents are reduced, not into the peritoneal cavity, but extra-peritoneally.

RECURRENT INGUINAL HERNIA

Although some individual series show a recurrence-rate of under 1 per cent., the general rate of recurrence is nearer 10 per cent. Seventy-five per cent. of recurrent inguinal herniæ are of the indirect variety. In most instances the recurrence takes place within a year, but it may occur much later.

The following summarises the principal causes of recurrence :

1. **Pre-operative.**—Faulty selection of cases (e.g. those with Malgaigne's bulging, (fig. 1346)) or chronic bronchitis.
2. **Operative.**—Faulty technique.
 - (a) Failure to ligate the sac at the neck.
 - (b) Tying stitches too tightly so that intervening tissues are devitalised.
 - (c) Imperfect hæmostasis, predisposing to infection.
 - (d) The use of absorbable sutures in the repair of the inguinal canal.
3. **Post-operative.**—The common causes of a recurrence are (a) infection of the wound; (b) persistent cough; (c) heavy lifting before three months after herniorrhaphy; (d) urethral obstruction; (e) straining at defæcation.

Treatment.—Unless there is some reason why further operation is unlikely to be successful, e.g. persistent cough, re-operation performed carefully with special precautions against recurrence, e.g. nylon darn, fascial graft, tantalum gauze or Dacron mesh, is often indicated.

The Spermatic Cord as a Barrier to Effective Closure of the Inguinal Canal.—In the elderly patient, removal of the testis aids in an effective repair in the case of recurrent inguinal hernia, sliding hernia, and some large direct herniæ. The permission of the patient must always be obtained.



FIG. 1349.—Reduction-en-masse.

¹ Spica, L. = an ear of corn. The individual grains overlap each other, as do the turns of the bandage.

² An overhead beam or bar for applying traction to a fractured femur, first used in the Balkan theatre of war, 1914–1918.

SLIDING HERNIA (*syn.* **HERNIE-EN-GLISSADE**) (Fig. 1350)

As a result of slipping of the posterior parietal peritoneum on the underlying cellular tissue, the posterior wall of the sac is not formed of peritoneum alone, but by the sigmoid colon and its mesentery on the left, the cæcum on the right and, sometimes, on either side by a portion of the bladder. It should be clearly understood that the cæcum, appendix, or a portion of the colon *wholly within* a hernial sac does not constitute a sliding hernia. A small bowel sliding hernia occurs once in 2,000 cases; a sacless sliding hernia once in 8,000 cases.

Ætiology.—Usually a sliding hernia does not originate as such, but rather, in the course of time, a hernia (nearly always an oblique in the comparatively young, and more commonly a direct inguinal hernia in later life) becomes a sliding hernia. At first there is a gradual increase in the size of the hernia, but this alone cannot cause it to slide. The presence of a congenital anatomical variation in the attachment of the sigmoid and an insecure attachment of the parietal peritoneum to the under-

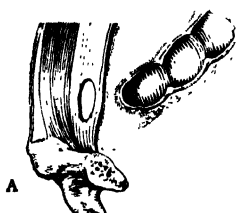


FIG. 1350A.—The stage set for a sliding hernia.

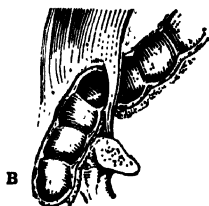


FIG. 1350B.—Fully developed left sliding hernia.

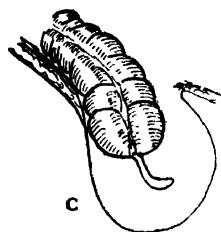


FIG. 1350C.—Right-sided sliding hernia.

lying cellular tissues causes the viscus to drag its anchor (fig. 1350B). Increasing flabbiness of the abdominal musculature and obesity are accessory factors. When a sliding hernia occurs on the right side (fig. 1350C) it does so only in one of the 5 per cent. of individuals who have a cæcal mesentery (Ryan). When a sliding femoral hernia occurs the postero-internal wall of the sac is composed of a portion of the urinary bladder.

Clinical Features.—A sliding hernia occurs almost exclusively in males. Five out of six sliding herniæ are situated on the left side; bilateral sliding herniæ are exceedingly rare. The patient is nearly always over forty, the incidence rising with the weight of years. There are no clinical findings that are pathognomonic of a sliding hernia, but it should be suspected in every large globular inguinal hernia descending well into the scrotum.

Occasionally large intestine is strangulated in a sliding hernia; more often non-strangulated large intestine is present behind the sac containing strangulated small intestine.

Treatment.—A sliding hernia is impossible to control with a truss, and as a rule the hernia is a cause of considerable discomfort. Consequently operation is indicated, and the results generally are good.

Operation.—It is unnecessary to remove any of the sliding hernial sac provided it is freed completely from the cord and the abdominal wall, and that it is replaced deep to the repaired fascia transversalis. In most instances it is desirable to perform orchiectomy (p. 1235) in order to effect a secure repair. No attempt should be made

to dissect the cæcum or colon free from the peritoneum under the impression that there are adhesions in which case peritonitis or a fæcal fistula resulting from necrosis of a devascularised portion of the bowel may occur. This is specially liable to occur on the left side, as vessels in the meso-colon may be injured.

FEMORAL HERNIA

Femoral hernia is the third most common type of hernia (incisional hernia comes second). It accounts for about 20 per cent. of herniæ in women, and 5 per cent. in men. The overriding importance of femoral hernia lies in the facts that it cannot be controlled by a truss, and that of all herniæ it is the most liable to become strangulated.

Surgical Anatomy.—The femoral canal occupies the most medial compartment of the femoral sheath, and is shaped like a truncated cone. It extends from the femoral ring above to the saphenous opening below. It is 1.25 cm. ($\frac{1}{2}$ inch) long, and $\frac{1}{2}$ inch wide at its base, which is directed upwards. The femoral canal contains fat, lymphatic vessels, and the lymph node of Cloquet. It is closed above by the septum crurale, a condensation of extraperitoneal tissue pierced by lymphatic vessels, and below by the cribriform fascia.

The femoral ring (fig. 1351) is bounded:

Anteriorly by the inguinal ligament.

Posteriorly by Astley Cooper's (ilio-pectineal) ligament, the pubic bone, and the fascia over the pectineus muscle.

Medially by the concave knife-like edge of Gimbernat's (lacunar) ligament (fig. 1351), which is also prolonged along the ilio-pectineal line as Astley Cooper's ligament.

Laterally by a thin septum separating it from the femoral vein.

Throughout an operation for the repair of a femoral hernia, *on the lateral side*, the femoral vein, with the internal saphenous vein emptying into it, must be protected. *On the medial side* of the neck of the sac great care must be taken not to injure the bladder, particularly since a portion of the bladder may form part of the wall of the sac (a sliding femoral hernia).



FIG. 1351.—The right femoral ring from within. The dotted structure is an abnormal obturator artery.

Sex Incidence.—The female to male ratio is about 2:1, but it is interesting that whereas the female patients are frequently elderly, the male patients are usually between thirty to forty-five years. The condition is more prevalent in women who have borne children than in nulliparæ.

Pathology.—A hernia passing down the femoral canal descends vertically as far as the saphenous opening. Because of the attachment of the superficial fascia to the lower part of the circumference of the saphenous opening, the hernial sac is directed forwards, pushing before it the cribriform fascia; it then curves upwards towards the inguinal ligament (fig. 1352). While it is confined to the inelastic walls of the femoral canal the hernia is necessarily narrow, but once it escapes through the saphenous opening into the loose areolar tissue of the groin, it expands, sometimes considerably. A fully distended femoral hernia assumes the shape of a retort

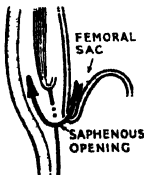


FIG. 1352.—The path taken by a femoral hernia.

Jules Germain Cloquet, 1790–1883. Professor of Clinical Surgery, Paris.

Sir Astley Cooper, 1768–1841. Surgeon, Guy's Hospital, London.

Antonio Gimbernat y Arbos, 1784–1816. Professor of Anatomy and Surgeon to the Santa Cruz Hospital, Barcelona.



FIG. 1353.

(fig. 1353), and its bulbous extremity may be above the inguinal ligament. By the time the contents have pursued so tortuous a path they are usually irreducible and apt to strangulate.

Clinical Features.—Femoral hernia is very rare before the fifteenth year. Between twenty and forty years of age the prevalence rises, and continues to old age. The right side (fig. 1354) is affected twice as often as the left, and in 20 per cent. of cases the condition is bilateral. The symptoms to which a femoral hernia gives rise are less pronounced than those of an inguinal hernia; indeed, a small femoral hernia may be unnoticed by the patient or disregarded for years, until perhaps one day it strangulates. Adherence of greater omentum sometimes causes a dragging pain, but the most usual complaint in an uncomplicated femoral hernia is the presence of a swelling. On the whole a femoral hernia is small and, especially in the obese, is liable to escape detection unless on clinical examination it is sought specifically. Occasionally a large sac is present.

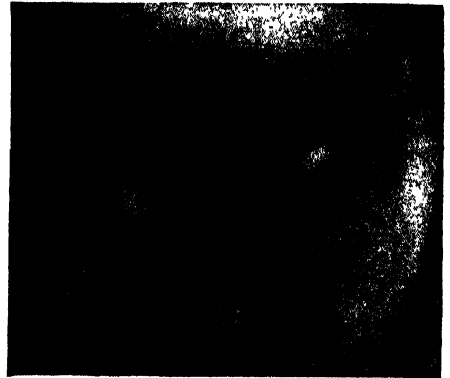


FIG. 1354.—The patient has a left inguinal and a right femoral hernia.

Differential Diagnosis

A Femoral Hernia has to be distinguished from:

(1) *An Inguinal Hernia.*—The neck of an inguinal hernia lies above the line of the medial end of the inguinal ligament and its attachment to the pubic tubercle. The femoral hernia lies below this line (figs. 1354, 1355). Occasionally the fundus of a femoral hernia sac overlies the inguinal ligament (fig. 1356). Furthermore, when

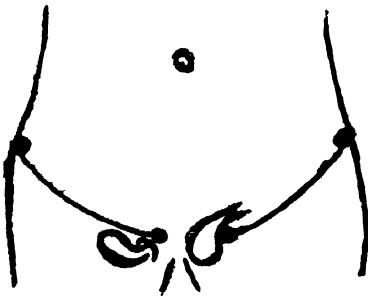


FIG. 1355.—The essentials of differential diagnosis between a femoral and an inguinal hernia (see text).



FIG. 1356.—An irreducible femoral hernia with its fundus overlying the inguinal ligament.

the tip of the little finger is insinuated into the superficial inguinal ring (with the patient standing), and the inguinal canal is found to be empty, the swelling cannot be an inguinal hernia.

(2) *A Saphena Varix* (fig. 1357).—A saphena varix is a saccular enlargement of the termination of the long saphenous vein and it is usually accompanied by other signs of varicose veins. The swelling disappears completely when the patient lies

down, while a femoral hernia sac usually is still palpable. In both there is an impulse on coughing. A saphena varix will, however, impart a fluid thrill to the examining fingers when the patient coughs, or when the saphenous vein below the varix is tapped with the fingers of the other hand. Sometimes a venous hum can be heard when a stethoscope is applied over a saphena varix.

(3) *An Enlarged Femoral Lymph Node*.—If there are other enlarged lymph nodes in the region the diagnosis is tolerably simple, but when Cloquet's lymph node alone is affected the diagnosis may be impossible unless there is a lead, such as an infected wound or abrasion on the corresponding limb or on the perineum.

(4) *Lipoma*.

(5) *A Femoral Aneurysm*.—The intrinsic signs of an aneurysm are described on p. 125.

(6) *A Psoas Abscess*.—There is often a fluctuating swelling—an iliac abscess—which communicates with the swelling in question (fig. 1358). Examination of the spine and an X-ray will settle the diagnosis.

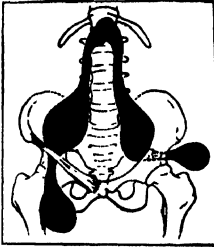


FIG. 1358.—A psoas abscess appearing beneath the inguinal ligament may simulate a reducible femoral hernia. Likewise, when it points in the buttock, a gluteal hernia has been suspected. (After J. F. Calot.)

Cloquet's hernia is one in which the sac lies under the fascia covering the pectineus muscle. Strangulation is likely. The sac may coexist with the usual type of femoral sac (see interparietal inguinal hernia, p. 1058).

Strangulated Femoral Hernia.—It cannot be emphasised too strongly that not only does a femoral hernia become strangulated frequently, but often gangrene develops rapidly. This is accounted for by the narrow, unyielding femoral ring. In 40 per cent. of cases the obstructing agent (fig. 1360)

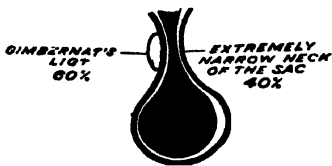


FIG. 1360.

is not Gimbernat's (lacunar) ligament but the narrow neck of the femoral sac itself (Henry Souttar). The frequent occurrence of a *Richter's hernia* (p. 1034) also must be stressed.



FIG. 1357.—Saphena varix.

(7) *A distended Psoas Bursa*.—The swelling diminishes when the hip is flexed, and osteoarthritis of the hip is present.

(8) *Rupture of the adductor longus* (with hæmatoma).—If there is no superficial discoloration, the diagnosis can be very difficult.

Hydrocele of a Femoral Hernial Sac.—The neck of the sac becomes plugged with omentum or by adhesions, and a hydrocele of the hernial sac results (fig. 1359).

Laugier's femoral hernia is a hernia through a gap in the lacunar (Gimbernat's) ligament. The diagnosis is based on the unusual medial position of a small femoral hernial sac. Nearly always the hernia has been strangulated.

Narath's femoral hernia occurs only in patients with congenital dislocation of the hip and is due to lateral displacement of the psoas muscle. The hernia lies hidden behind the femoral vessels.



FIG. 1359.—Hydrocele of a femoral hernial sac. The patient previously had ascites, which abated under treatment.

TREATMENT OF FEMORAL HERNIA

The constant risk of strangulation is sufficient reason for urging the patient to have herniorrhaphy performed. A *femoral truss* is highly unsatisfactory in that it becomes displaced when the thigh is flexed (unless the hip-joint should be arthrodesed), and strangulation is thereby encouraged.

Operative Treatment.—The low operation (Lockwood), the high operation (McEvedy), and the inguinal operation (Lotheissen) all have their advocates. In all cases the bladder *must* be emptied immediately before commencing the operation.

The Low Operation (Lockwood).—The sac is dissected out below the inguinal ligament via a groin-crease incision. It is essential to peel off all the anatomical layers which cover the sac. These are often thick and fatty. After dealing with the contents (e.g. freeing adherent omentum) the neck of the sac is pulled down, ligated as high as possible and allowed to retract through the femoral canal. The canal is closed by suturing the inguinal ligament to the ilio-pectineal line using three unabsorbable sutures (e.g. monofilament nylon.)

The High (McEvedy) Operation.—A vertical incision is made over the femoral canal and continued upwards above the inguinal ligament. Through the lower part of the incision the sac is dissected out. The upper part of the incision exposes the inguinal ligament and the rectus sheath. The superficial inguinal ring is identified, and an incision 2.5 cm (1 inch) above the ring and parallel to the outer border of the rectus muscle is deepened until the extraperitoneal space is found.

By gauze dissection in this space the hernial sac entering the femoral canal can be easily identified. Should the sac be empty and small, it can be drawn upwards; if it is large, the fundus is opened below, and its contents, if any, dealt with appropriately before delivering the sac upwards from its canal. The sac is then freed from the extraperitoneal tissue and its neck is ligated. An excellent view of the iliopectineal ligament is obtained and the conjoined tendon is sutured to it with non-absorbable sutures (fig. 1361).

An advantage of the operation is that if resection of intestine is required, ample room can be obtained by opening the peritoneum. The disadvantage of the operation is that if infection occurs incisional hernia is not very unusual.

Lotheissen's Operation.—The inguinal canal is opened as for *inguinal* herniorrhaphy. The transversalis fascia is incised to the medial side of the epigastric vessels and the opening is enlarged. The peritoneum is now in view; one must be certain that it is the peritoneum and not the bladder or a diverticulum thereof. The peritoneum is picked up with dissecting forceps, and incised. It is now possible to ascertain if any intraperitoneal structure is entering

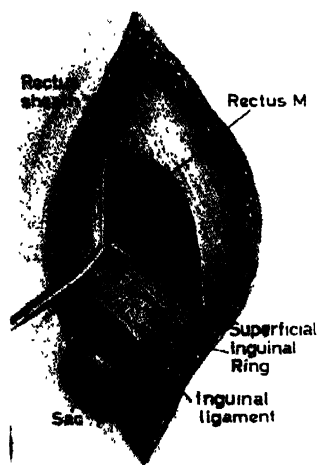


FIG. 1361.—McEvedy's operation.

the femoral sac. Should the sac be empty, hæmostats are placed upon the edges of the opening into the peritoneum, and by gauze dissection the sac is withdrawn from the femoral canal. An empty sac can be delivered easily; in the event of the sac being occupied, the technique described below for strangulation should be followed.

The Lotheissen Repair.—The conjoined tendon is sutured to the iliopectineal line to form a shutter. While protecting the external iliac vein with the forefinger, unabsorbable sutures are passed through the periosteum and Cooper's ligament overlying the iliopectineal line (fig. 1362). The retractor having been removed, the

long ends of the sutures are passed from within, outwards, through the conjoined tendon, and tied, thus approximating the conjoined tendon to the iliopectineal line. If there is any tension, a Tanner's slide (p. 1042) will facilitate this step. The incised external oblique is sutured.

Strangulated Femoral Hernia.—It is necessary to modify some of the steps of the Lotheissen operation. As soon as the external oblique has been exposed, the inferior margin of the wound is retracted strongly, thereby displaying the swelling. The coverings of the sac are incised and peeled off, until the sac, dark from contained bloodstained fluid, is apparent. The sac is incised, and the fluid that escapes is mopped up with great care. The retractor is removed and the operation is continued above the inguinal ligament in the same way as described already. Once the peritoneum has been opened above the inguinal ligament, one can peer within and see exactly what is entering the sac. With suitable retraction, Gimbernat's ligament is sought, and it can be avulsed by firm pressure applied by the surgeon's thumb. Alternatively, by means of a bistoury guided by a director, this ligament is nicked in one or two places. Should the obstruction lie in a narrow neck of the sac, the beak of a hæmostat is insinuated, and with great care the neck is stretched¹. The contents of the sac are delivered, and dealt with *secundum artem*, p. 1039.



FIG. 1362.—Method of placing the deep sutures (see text). The finger is protecting the femoral vein.

UMBILICAL HERNIA

Exomphalos (*syn.* omphalocele) occurs once in every 6,000 births; it is due to failure of all or part of the mid-gut to return to the cœlom during early fetal life. Sometimes a large sac ruptures during birth (fig. 1363). When the sac remains unruptured, it is semi-translucent, and although very thin it consists of three layers—an outer layer of amniotic membrane, a middle layer of Wharton's jelly, and an inner layer of peritoneum. There are two varieties of exomphalos:



FIG. 1363.—Exomphalos. The delicate sac burst during delivery.

Inadvertently a loop of small intestine or a Meckel's diverticulum has been included in the ligature applied to the base of an umbilical cord containing this protrusion.

Exomphalos Major.—The umbilical cord is attached to the inferior aspect of the swelling (fig. 1365), which contains small and large intestine, and nearly always a portion of the liver. Half the cases belong to this group.



FIG. 1364.—Exomphalos minor.

Exomphalos Minor.—The sac is relatively small and to its summit is attached the umbilical cord (fig. 1364).



FIG. 1365.—Exomphalos major.

¹ An abnormal obturator artery is present either on the medial or the lateral side of the neck of the sac in 28 per cent. of cases.

Thomas Wharton, 1614–1673. Physician, St. Thomas's Hospital, London.

Treatment : (a) *Exomphalos Minor*.—It is necessary only to twist the cord, so as to reduce the contents of the sac through the narrow umbilical opening into the peritoneal cavity, and to retain them there by strapping applied firmly. In spite of a sero-purulent discharge from beneath it, on no account must the strapping be removed for fourteen days (Denis Browne).

(b) *Exomphalos Major*.—Operation within the first few hours of life is the only hope, otherwise the sac will burst. In order to prevent further distension of the contents of the sac, the infant should not be fed. A few newborn infants with a ruptured sac have survived following immediate operation and antibiotic therapy. A drip blood-transfusion is desirable.

Operation.—It must be realised that most of the contents of the sac have never been housed within the abdominal cavity; consequently that cavity is unduly small, and to attempt to replace the contents of the sac is like endeavouring to put 2 lb. of sugar into a 1 lb. bag—a feat that so often results in respiratory embarrassment, compromise of venous return, and possibly intestinal obstruction. It is necessary to create flaps of skin by undermining the subcutaneous tissue on either side, so that the flaps can be brought together over the sac. If necessary relaxing incisions (fig. 15) must be made in the loins to permit closure. For several days following the operation it is advisable to carry out aspiration through an indwelling gastric tube, in order to relieve or prevent distension. If the patient survives the construction of this protective cutaneous coverage, repair of the hernia can be delayed for months, or even years. At the second operation, it is surprising to find that the peritoneum and the muscles can be drawn together and closed in layers.

Congenital Umbilical Hernia.—On rare occasions a well-developed umbilical hernia is present at birth; the condition is believed to be due to intra-uterine epithelialisation of a small exomphalos.

Umbilical Hernia of Infants and Children.—This is a hernia through a weak umbilical scar. The ratio of males to females is 2:1. Usually the hernia is symptomless, but sometimes increase in the size of the hernia by

crying causes pain, which makes the infant cry the more. Small herniæ are spherical; those that increase in size tend to assume a conical shape (fig. 1366) and are present apart from crying. Obstruction or strangulation below the age of three years is extremely uncommon.

Treatment.—Conservative treatment is successful in about 93 per cent. of cases.

Masterly Inactivity.—When the hernia is symptomless, reassurance of the parents is all that is necessary, for in a very high percentage of cases the hernia will be found to disappear spontaneously during the first few months of life.

Herniorrhaphy.—In cases where masterly inactivity fails, operation is required, and it should be carried out, preferably, about the age of two years.



FIG. 1366. — Infantile umbilical hernia.

Operation.—In infants a small curved incision is made immediately below the umbilicus (fig. 1367). The skin cicatrix is dissected upwards, and the neck of the sac is isolated. After ensuring that the sac is empty of contents, it is either inverted into the abdomen or it is ligated by transfixion and excised. The defect in the linea alba is closed with two unabsorbable sutures.

Para-umbilical Hernia of Adults (syn. supra-, infra-umbilical hernia).—It should be noted that in adults the hernia does not occur through the umbilical scar. It is a protrusion through the linea alba just above the umbilicus or, occasionally, just below that structure (fig. 1368). As it enlarges, it becomes rounded or oval in shape (fig. 1369) with a tendency to sag downwards, viz.:

Para-umbilical herniæ increase steadily in size and frequently attain very large dimensions (fig. 1370). The neck of the sac is often remarkably narrow as compared with the size of the sac and the volume of its contents, which consist of greater omentum often accompanied by small intestine and, alternatively or in addition, a portion of the transverse colon. In old-standing cases the sac sometimes becomes loculated due to adherence of omentum to its fundus.



FIG. 1367.—The incision, 8mm. from the hernia. (After L. F. Watson.)



FIG. 1368.—Small.



FIG. 1369.—Large.
Para-umbilical hernia

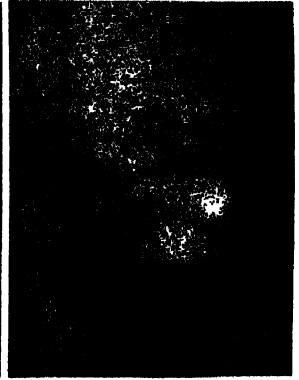


FIG. 1370.—Very large.

Clinical Features.—Women are affected five times more frequently than men. The patient is usually corpulent and between the ages of thirty-five and fifty. Increasing obesity, with flabbiness of the abdominal muscles, and repeated pregnancy are important factors. These herniæ soon become irreducible because of omental adhesions within the sac. A large umbilical hernia causes a local dragging pain by its weight. Gastro-intestinal symptoms are common and are probably due to traction on the stomach or transverse colon. Often there are transient attacks of intestinal colic due to subacute intestinal obstruction. In old-standing cases intertrigo of the adjacent surfaces of the skin is a troublesome complication.

Treatment.—Untreated, the hernia increases in size, and more and more of its contents become irreducible. Eventually, strangulation may occur. Therefore without undue delay operation should be advised in nearly all cases. If the patient is obese and the hernia is symptomless, operation can be postponed with advantage until weight has been reduced. The laxity of the abdominal wall during the early puerperium makes it a most opportune time to perform umbilical herniorrhaphy.

Mayo's Operation.—A transverse elliptical incision is made around the umbilicus. The subcutaneous tissues are dissected off the rectus sheath to expose the neck of the sac. The neck is incised to expose the contents. Intestine is returned to the abdomen. Any adherent omentum is freed and ligated by transfixion if it is bleeding. Excess adherent omentum can be removed with the sac if necessary. The sac is then removed and the peritoneum of the neck closed with catgut. The aponeurosis on both sides of the umbilical ring are incised transversely for 2.5 cm (1 inch) or more—sufficiently to allow an overlap of 5 or 7.5 cm (2 or 3 inches). Three to five mattress sutures are then inserted into the aponeurosis as shown in fig. 1371. When this row of mattress sutures has been tied, the overlapping upper margin is stitched to the sheath of the rectus abdominis and the midline aponeurosis. It is important to denude this area of fat before stitching the



FIG. 1371.—Mayo's operation for umbilical hernia.

flap in position. In fat patients, who ooze blood and liquid fat, a drain is provided at each end of the wound. The subcutaneous fat and skin are then approximated with deep sutures. If the patient has a tendency to bronchitis, it is wise to prescribe antibiotic therapy and breathing exercises.

A few operators, dissatisfied with the results of Mayo's operation (which is acclaimed generally to be satisfactory), prefer to employ the fascial flap operation depicted in fig. 1372.

Additional Lipectomy.—In patients with a para-umbilical hernia associated with a large pendulous, fat-laden abdominal wall the operation can, with great advantage, be combined with lipectomy by fashioning the incisions to embrace a larger area of the fat-laden superficial layers of the abdominal wall.

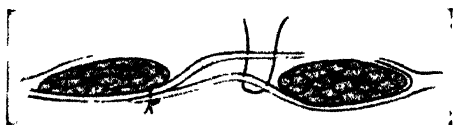


FIG. 1372.—Method of repairing a gap in the linea alba. (After Charles Wells, F.R.C.S.)

Strangulation is a frequent complication of a large para-umbilical hernia in adults. Owing to the narrow neck and the fibrous edge of the linea alba, gangrene is liable to supervene unless early operation is carried out.

Operation.—In early cases the operation does not differ from that for non-strangulated cases. Gangrenous contents are dealt with as in other situations. If a portion of the transverse colon is gangrenous, it should be exteriorised by the Paul-Mikulicz method (fig. 1214) and the gangrenous portion excised. If the ring is large enough to transmit the colon unhampered, it is left alone; otherwise it is enlarged. It is important that the small intestine be thoroughly scrutinised as a small loop may have been trapped and slipped back when the constriction was relieved. If non-viable gut is overlooked, peritonitis quietly supervenes, and the symptoms are ascribed to 'post-operative discomfort'. The condition of the patient steadily deteriorates until she succumbs after a few days (Franklin).

INCISIONAL HERNIA (*syn.* VENTRAL HERNIA; POST-OPERATIVE HERNIA)

Ætiology.—An incisional hernia usually starts as a symptomless, partial disruption of the deeper layers of a laparotomy wound during the immediate or very early post-operative period. It is more than probable that such an event will pass unnoticed, for the skin stitches remain intact. A sero-sanguinous discharge from a laparotomy incision is often a signal of dehiscence.

(p. 1029), and calls for systematic palpation of the immediate proximity of the incision, with the examiner duly masked and gloved. Resuture of the deeper disrupted layers of the incision obviates the more difficult repair of an established hernia later on. Incisional hernia occurs most often in obese individuals, and a persistent post-operative cough and post-operative abdominal distension are its precursors. There is a high incidence of incisional hernia following operations for peritonitis because, as a rule, the wound becomes infected. The accommodation of a necessary drainage tube in a stab incision, as opposed to placing such a tube through the laparotomy wound, reduces the frequency of its occurrence.

The concept that the hernia occurs during late convalescence is erroneous. Provided the main nerves to the abdominal musculature have not been damaged, a *soundly healed* laparotomy scar will withstand the buffeting of any walk of life, including repeated pregnancy.

Clinical Features.—Incisional hernia presents no difficulty in diagnosis. There are great variations in the degree of herniation. The hernia may occur through a small portion of the scar, often the lower end. More frequently there is a diffuse bulging of the whole length of the incision. A post-operative hernia, especially one through a lower abdominal scar, usually increases steadily in size, and more and more of its contents become irreducible. Sometimes the skin overlying it is so thin and atrophic that normal peristalsis can be seen in the underlying coils of intestine. Attacks of sub-acute intestinal obstruction are common, and strangulation is liable to occur at the neck of a small sac or in a loculus of a large one.

Treatment.—*Palliative.*—An abdominal belt is sometimes satisfactory, especially in cases of a hernia through an upper abdominal incision.

Operation.—Many procedures are advocated. Three only will be outlined here.

Pre-operative Measures.—In order to obtain a lasting repair, very special preparation is required. If the patient is obese, reduction by dieting should precede the operation. To attempt to return the contents of a very large hernia to the main abdominal cavity if they have not been there for several years is to court danger, unless weight reduction has been effected. In these circumstances, not only is there a risk of failure of the hernioplasty, but there is a greatly increased risk of paralytic ileus from visceral compression, and of pulmonary complications from elevation of the diaphragm.

The 'Keel' Operation (Rodney Maingot).—In this operation, the hernial sac is not opened. It is dissected out and pushed back into the abdomen, and by a series of inverting and pleating layers of unabsorbable sutures, the abdominal deficiency is closed. This repair, viewed as a cross-section, looks something like the keel of a ship (fig. 1373). The incidence of post-operative ileus and distension is less than that occurring after operations in which the hernial sac is opened. The technique includes: (a) A wide excision of skin and scar tissue overlying the sac. Special care has to be taken not to damage any bowel which is often adherent to the underside of the sac, (b) an extensive mobilisation of skin-flaps and fat, and exposure of healthy and strong aponeurosis around the neck of the sac, (c) inversion of the sac into the



FIG. 1373. — Cross-section of the 'keel' operation.

abdomen, pleating it with strong interrupted unabsorbable sutures, care being taken not to damage any underlying bowel. A second, third, or fourth layer of sutures are inserted until, finally, the healthy margins of the aponeurosis are brought firmly together. Any degree of tension in this final layer can be averted by a few relaxation incisions in the aponeurosis, parallel to the suture line.

When the scar is paper-thin and devoid of normal skin, Ibrahim advises that no attempt should be made to separate dermal scar from sac—both should be inverted as one.

Cattell's Operation.—In this operation the hernial sac is dissected out with equal care, but then it is formally opened, the viscera are reduced, and the repair starts from the inside margins of the sac (fig. 1374). During the freeing of the contents

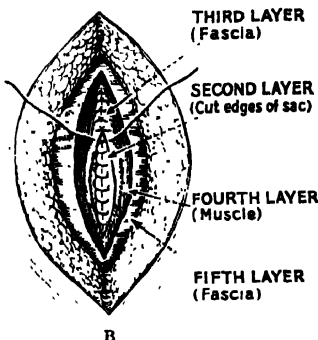
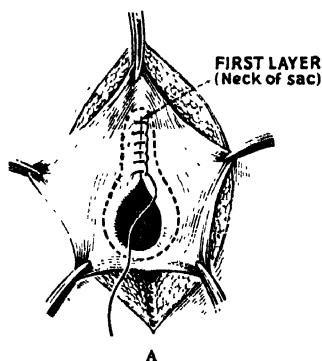


FIG. 1374.—A. The closure of the hernial defect from inside the sac. Dotted line indicates the excess sac. B. Approximation of the third layer. (After R. B. Cattell.)

of the sac any adherent omentum can be ligated and divided, leaving a portion of it attached to the fundus of the sac and skin. The layers of repair are as follows: The peritoneum and the abdominal wall are approximated at the neck with an interlocking suture of thick chromic catgut, including all layers of the abdominal wall that are attached to the hernial ring (fig. 1374A). An incision is made

through the sac as shown by the dotted line in fig. 1374A. (2) The cut edges of the base of the sac are approximated with unabsorbable sutures. An elliptical incision is made 2 cm. lateral to the previous suture line. (3) The medial borders of the incision are approximated. The lateral edges of the fascia are freed from the overlying muscles for some distance, and this fascial layer is approximated with interrupted sutures at the upper and lower ends of the wound. This is done to relieve tension on the muscles. (4 and 5) The muscles and the remaining fascial layer are approximated by alternating stitches (fig. 1374B). Tension-relaxing incisions may be required and should be placed well laterally.

Tantalum Gauze, Plastic Fibre Mesh or Net closures.—The sac is dealt with in either of the methods just described. The deficiency in the abdominal wall can very easily be bridged without tension by laying and sewing on a sheet of tantalum gauze, or a mesh or net made of Nylon or Dacron, cut to well over-size and tacked down to the aponeurosis.

Careful haemostasis and meticulous asepsis are essential during these operations. Post-operative collections of serum can be removed by drainage, using plastic tubing led, via skin punctures lateral to the wound, into closed suction drainage bottles (e.g. Redi-vac).

Post-operative Treatment.—Continuous gastric decompression should be employed, and nothing by mouth allowed until the bowels have functioned. Early ambulation is to be discouraged, but exercises, especially of the legs, are to be encouraged. The patient should not resume strenuous exercise for three months.

EPIGASTRIC HERNIA (*syn.* FATTY HERNIA OF THE LINEA ALBA)

An epigastric hernia occurs through the linea alba anywhere between the xiphoid process and the umbilicus, usually midway between these structures. Such a hernia commences as a protrusion of extra-peritoneal fat through the linea alba, where the latter is pierced by a small blood-vessel. Sometimes more than one hernia is present.

A swelling the size of a pea consists of a protrusion of extraperitoneal fat only (fatty hernia of the linea alba (figs. 1375 and 1376)). If the protrusion enlarges, it drags a pouch of peritoneum after it, and so becomes a true epigastric hernia. The mouth of the hernia is rarely large enough to permit a portion of a hollow viscus to enter it; consequently, either the sac is empty or it contains a small portion of greater omentum.

It is probable that an epigastric hernia is the direct result of a sudden strain tearing the interlacing fibres of the linea alba. The patients are often manual workers between thirty and forty-five years of age.

Clinical Features:

(a) *Symptomless*.—A small fatty hernia of the linea alba can be felt better than it can be seen, and may be symptomless, being discovered only in the course of routine abdominal palpation.



FIG. 1376.—Epigastric hernia.

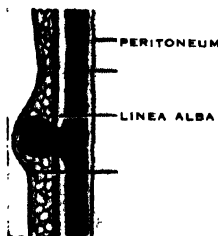


FIG. 1375.—Fatty hernia of the linea alba.

(b) *Painful*.—Sometimes such a hernia gives rise to attacks of local pain, worse on physical exertion, and tenderness to touch and tight clothing, possibly because the fatty contents become nipped sufficiently to produce partial strangulation.

(c) *Referred Pain* (Peptic ulcer cases).—It is not uncommon to find that the patient, who may not have noticed the hernia, complains of pain suggestive

of a peptic ulcer. On the other hand, a patient suffering from a gastric or duodenal ulcer may attribute all his symptoms to an epigastric hernia. In many cases of epigastric hernia there is coexistent upper abdominal disease.

Treatment.—If the hernia is giving rise to symptoms, operation should be undertaken.

Operation.—An adequate vertical or transverse incision is made over the swelling, exposing the linea alba. The protruding extraperitoneal fat is cleared from the hernial orifice by gauze dissection. If the pedicle passing through the linea alba is slender, it is separated on all sides of the opening by blunt dissection. After ligating the pedicle, the small opening in the linea alba is closed by unabsorbable sutures. When a hernial sac is present it is opened and any contents reduced, after which the sac is excised before repairing the linea alba.

DIVARICATION OF THE RECTI ABDOMINIS

Divarication of the recti abdominis is seen principally in elderly multiparæ. When the patient strains, a gap can be seen between the recti abdominis through which gap the abdominal contents bulge. When the abdomen is relaxed the fingers can be introduced between the recti.

Treatment.—An abdominal belt is all that is required.

A similar condition is met with in babies, only the divarication exists above the umbilicus. No treatment is necessary; as the child develops a spontaneous cure results.

RARE EXTERNAL HERNIÆ

Interparietal Hernia (*syn.* Interstitial Hernia).—An interparietal hernia has a hernial sac that passes between the layers of the anterior abdominal wall. The sac may be associated with, or communicate with, the sac of a concomitant inguinal or femoral hernia. Lack of knowledge of this condition is the cause of misdiagnosis and mismanagement.

Varieties (fig. 1377):

1. *Properitoneal* (20 per cent.).—Usually the sac takes the form of a diverticulum from a femoral or inguinal hernia, but these can be quite distinct from the properitoneal hernia.

2. *Intermuscular* (60 per cent.).—The sac passes between the muscular layers of the anterior abdominal wall, usually between the external oblique and internal oblique muscles. The sac is nearly always bilocular, and is associated with an inguinal hernia.

3. *Inguino-superficial* (20 per cent.).—The sac expands beneath the superficial fascia of the abdominal wall or the thigh.

Ætiology.—It is believed that sometimes an interparietal hernia is congenital, but it may be acquired due to the wearing of a truss or to the blocking of the superficial inguinal ring by (1) an incompletely descended testis, (2) hydrocele of the cord, or (3) a hydrocele of the canal of Nuck. The inguino-superficial type is commonly associated with an incompletely descended testis.

Clinical Features.—In all varieties, the incidence in the male is higher than in the female, viz. 3:1 in the properitoneal and intermuscular varieties, and 14:1 in the inguino-superficial variety. Most patients present with intestinal obstruction, due to obstruction or strangulation of the hernia. In these circumstances the cause of the obstruction should be apparent in the intermuscular and inguino-superficial varieties, but in the properitoneal variety, as no swelling is likely to be apparent, delays in diagnosis occur and consequently the mortality in this variety is high.

Treatment.—Operation is imperative because of intestinal obstruction. In all cases of inguinal and femoral hernia a finger should be introduced into the sac with the object of detecting an unsuspected intraparietal loculus.

A **Spigelian hernia** is a variety of interparietal hernia. The Spigelian fascia is the aponeurosis intervening between the muscular part of the transversus muscle and the rectus sheath. A Spigelian hernia is one occurring through the Spigelian fascia, commonly at the level of the arcuate line (fig. 1378). The fundus of the sac, clothed by extraperitoneal fat, may lie beneath the internal oblique muscle, where it is virtually impalpable. More often it advances through that muscle and spreads out like a mushroom between the external and internal obliques and gives rise to a more evident swelling. The patient is often corpulent, and usually over fifty years of age, men and women being affected equally. Typically, a soft, reducible mass will be encountered lateral to the rectus muscle and below the umbilicus. After reduction a small oval defect can often be felt and because of the rigid fascia surrounding its neck, strangulation may occur. In a stout female, to differentiate a strangulated Spigelian hernia from a twisted ovarian cyst or a hæmatoma due to the tearing of the inferior epigastric artery may be impossible.

Treatment.—Operation should be advised as repair is simple. The external oblique aponeurosis is split. After isolating the sac, dealing with any contents, and ligating and excising it, the transversus, internal oblique, and external oblique muscles are approximated in layers.

Adrian van der Spiegel, 1678–1685. Professor of Anatomy and Surgery, Padua.

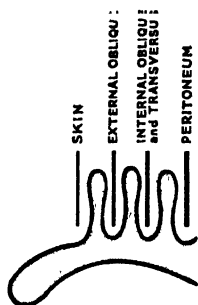


FIG. 1377.—The planes that an interparietal hernia may occupy.



FIG. 1378.—The usual site for a Spigelian hernia.

Lumbar Hernia.—Most primary lumbar herniæ gain exit through the inferior lumbar triangle of Petit (fig. 1379), bounded below by the crest of the ilium, laterally by the external oblique, and medially by the latissimus dorsi. Some come through the superior lumbar triangle, which is bounded by the twelfth rib above, medially by the sacrospinalis, and laterally by the posterior border of the internal oblique. An extensive incisional lumbar hernia sometimes follows an operation upon an infected kidney, in which the wound suppurated post-operatively.

Differential Diagnosis.—A lumbar hernia must be distinguished from: (a) A cold abscess pointing to this position. (b) Phantom hernia due to local muscular paralysis (fig. 1380). Lumbar phantom herniæ can result from any interference with the nerve-supply of the affected muscles (e.g. poliomyelitis).

Treatment.—A primary lumbar hernia, being small, does not entail an extensive operation for its repair. On the other hand, a post-operative lumbar hernia may be large, and the defect is impossible to repair unless fascial flaps are used. When necessary, the area can be reinforced still further by stitching in place a piece of tannum gauze or Dacron mesh.

Perineal hernia (*syn. hernia through the pelvic floor*) is not uncommon in the ageing dog—a reducible swelling appears immediately lateral to the anus. In human beings, except following excision of the rectum, perineal hernia is rare. Several varieties are encountered:

FIG. 1380.—Phantom hernia following anterior poliomyelitis.



FIG. 1380.—Phantom hernia following anterior poliomyelitis.

(a) *Antero-lateral perineal hernia* occurs in women and passes through an opening anterior to the transversus perinei muscle to enter the labium majus. It is more frequent than

(b) *Postero-lateral perineal hernia*, which passes through the levator ani to enter the ischio-rectal fossa. These herniæ seldom strangulate.

Treatment.—A combined operation is generally the most satisfactory. The hernia is exposed by an incision directly over it. The sac is opened and its contents are reduced. The sac is cleared from surrounding structures and the wound is closed. With the patient in semi-Trendelenburg position, the abdomen is opened and the mouth of the sac is exposed. The sac is inverted, ligated, and excised, and the pelvic floor is repaired as adequately as possible.

(c) *Median sliding perineal hernia* is a complete prolapse of the rectum (p. 998).

(d) *Post-operative hernia through a perineal scar* may occur after excision of the rectum. The onset may be at any time after the operation, but is more common after a year or so. Many asymptomatic herniæ of this kind are overlooked because the patient is not examined in the erect posture. When symptoms are present they include considerable pain on sitting, a feeling of perineal pressure when walking, and sometimes a swelling in the perineum. It can be repaired by excising the scar, freeing and reducing the contents into the peritoneal cavity, closing the opening in the peritoneum, and approximating the edges of the levator ani, if sufficient of this muscle can be identified. A flap of the gluteus maximus muscle and fascia is freed, and sutured in such a way as to form a sling to support the closed peritoneum.

Obturator Hernia.—The hernia, which passes through the obturator canal, occurs six times more frequently in women than in men. Most of the patients are over sixty years of age.

The swelling is liable to be overlooked because it is covered by the pectineus. It seldom causes a definite swelling in Scarpa's triangle, but if the limb is flexed, abducted, and rotated outwards, sometimes the hernia becomes more apparent. The leg is usually kept in a semi-flexed position and movement increases the pain. In more than 50 per cent. of cases of strangulated obturator hernia pain is referred along the obturator nerve (fig. 1381) by its geniculate branch to the knee. On vaginal or rectal examination the hernia sometimes can be felt as a tender swelling in the region of the obturator foramen.



FIG. 1379.—Inferior lumbar hernia.

Cases of obturator hernia which present themselves have usually undergone strangulation, which is often of the Richter type (p. 1034)

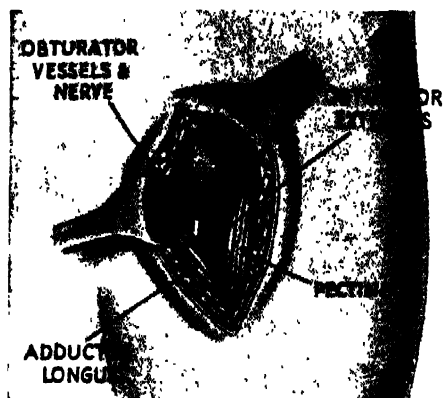


FIG. 1381.—The relationships of an obturator hernia. (After Sir Cecil Wakeley, F.R.C.S.)

Treatment.—(a) Perform lower laparotomy (on the side of the lesion, if known). Confirm the diagnosis and then adopt full Trendelenburg's position; (b) the constricting agent is the obturator fascia. Taking every precaution to avoid spilling infected fluid from the hernial sac into the peritoneal cavity, this fascia can be stretched to allow reduction by inserting suitable forceps, e.g. cholecystectomy forceps, through the gap in the fascia and opening the blades with care. If incision of the fascia is required, it is made parallel to the obturator vessels and nerve; (c) the contents of the sac are dealt with *secundum artem*; (d) the broad ligament is stitched over the opening to prevent recurrence; (e) the abdominal wall is closed.

Gluteal and Sciatic Herniæ.—A *gluteal hernia* passes through the greater sciatic foramen, either above or below the piriformis. A *sciatic hernia* passes through the lesser sciatic foramen.

Differential diagnosis must be made between these conditions and (a) A lipoma or fibro-sarcoma beneath the gluteus maximus. (b) A tuberculous abscess. (c) A gluteal aneurysm.

All doubtful swellings in this situation should be explored by operation. The swelling is approached by splitting the fibres of the gluteus maximus. After isolating the hernial sac, opening it, and dealing appropriately with any contents, the neck of the sac is ligated, and the sac is excised. A flap of fascia raised from the covering of the piriformis muscle is used to close the opening through which the hernia extruded. Like an obturator hernia, occasionally a gluteal or sciatic hernia is discovered in the course of laparotomy for intestinal obstruction.

CHAPTER 44

URINARY SYMPTOMS. INVESTIGATION OF THE URINARY TRACT. ANURIA

URINARY SYMPTOMS

THREE symptoms, a veritable triple alliance, accompany most urinary affections. They are pain, frequency, and hæmaturia.

Pain associated with affections of the urinary tract embraces :

Renal pain is usually a dull ache situated mainly in the costo-vertebral angle, but also in the upper and outer quadrant of the abdomen.

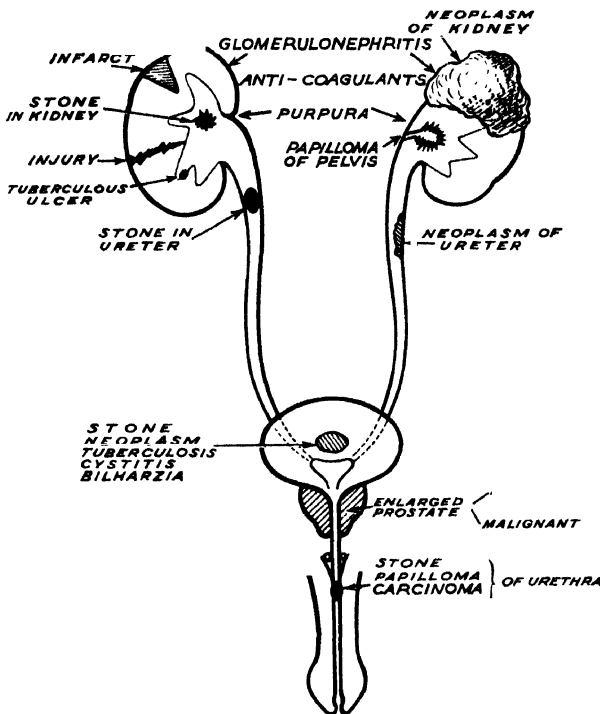


FIG. 1382.—The more common causes of hæmaturia.

Frequency.—For example, in prostatic disease, of great significance is the number of times the patient has to rise at night to micturate.

Hæmaturia.—Blood in the urine, however transient, is a symptom that must never be ignored. Painless hæmaturia is so often a herald of new-growth in some part of the urinary tract that a complete urinary investigation

Ureteric pain is the well-known colic¹ passing from the loin to the groin.

Vesical pain varies from slight suprapubic discomfort to strangury, which is agonising pain referred to the external urinary meatus, accompanied by an intense desire to micturate, but resulting in the expulsion (by straining) of only a few drops of blood-stained urine.

Prostatic and seminal vesicular pain is deep-seated in the rectum; it is often referred to the perineum, but sometimes to the suprapubic region or one or both iliac fossæ.

Urethral pain is scalding, and occurs during micturition.

¹ Renal colic is a term that is deeply rooted. Pain passing from loin to groin should be called ureteric colic, for obviously that is more accurate.

In the Male.—If the prepuce is present, it is retracted and all smegma is removed by washing with soap and water followed by swabbing with a mild antiseptic solution.

Cocaine is dangerous, absorption via the prostatic urethra into the circulation sometimes causing collapse and even death. 1 per cent. lignocaine with 0.25 per cent. chlorhexidine gluconate as a local antiseptic in a jelly base (Lidesthesin) is the safest. About 15 ml. is squeezed into the urethra and retained for five minutes with the aid of a penile clamp placed proximal to the glans.

In the Female.—The vulva is washed with soap and water followed by swabbing with a mild antiseptic solution. The labia minora are held apart and the local anæsthetic jelly is injected into the urethra.

Bouginae is described on p. 1195, and **Catheterisation** on p. 1166.

Cystoscopy.—By cystoscopy, the interior of the bladder is inspected and the ureteric orifices observed. If 7 ml. of a 0.4 per cent. solution (adult dose) of *indigo-carmin* are injected intravenously, the excretion of the dye down the ureters can be watched (fig. 1385); normally it should appear within four or five minutes. Delay of excretion of the dye on one side is indicative of unilateral

FIG. 1385.—Discharge of indigo-carmin down a right normal ureter. Cystoscopic appearance.

urinary obstruction or disease, while delay on both sides suggests bilateral renal impairment. By employing a catheterising cystoscope, ureteric catheters can be passed up each ureter (fig. 1386), and specimens of urine collected



FIG. 1386.—A ureteric catheter about to enter the left ureteric orifice. Cystoscopic view.

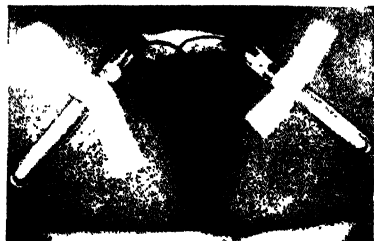


FIG. 1387.—Specimens of urine being collected after ureteric catheterisation. The test-tubes are capped with rubber finger-stalls.

from each kidney (fig. 1387). Such specimens are examined chemically and bacteriologically, and much information is obtained thereby.

Retrograde (syn. instrumental) pyelography is employed when a clearer definition than that afforded by the intravenous pyelogram is required, e.g. suspected renal neoplasm or early tuberculosis, or to collect urine from each kidney separately for differential functional tests. The ureters are catheterised, and the patient is conveyed to the radiological table. A plain radiograph is taken and developed before injecting the medium in order to ensure that the ureteric catheters are not too advanced. A sterile 12½ per cent. solution of sodium iodide can be used, but for even better definition Pyelectan retrograde (Glaxo Labs.), which is a 20 per cent. solution of iodoxyl, can be employed and has the advantage of being entirely non-irritating. A pyelograph syringe which fits any ureteric catheter is filled with 10 ml. of the solution, and the injection is made up the catheter slowly. If pain is complained of,

the injection is stopped. An initial film after 3 to 4 ml. have been injected will outline a normal pelvis (fig. 1388). Should a large hydronephrosis be present, more medium will be required to obtain clear definition. By this method it is possible to inject the pelves of both kidneys at the same sitting, but in cases of poor renal function it is essential to make each examination independently. In either event, after the X-ray exposure has been made, as much of the medium as possible is aspirated from the renal pelvis.

Cystography: (a) *Excretory*.—The cystographs which accompany the later films of excretory pyelography may not be sufficiently dense to give a clear delineation of pathological conditions in the bladder.

(b) *Retrograde* is employed principally to confirm the presence and dimensions of a vesical diverticulum. A rubber catheter is passed and the urine in the bladder is evacuated. If a diverticulum is suspected, the patient lies on his face so as to favour drainage of urine from the diverticulum. A plain radiograph is taken to exclude a calculus in the diverticulum. The bladder is then filled with medium until the patient feels a desire to micturate. An excellent contrast medium for cystography is a 25 per cent. solution of iodoxyl (B.P.). A radiograph is taken with the bladder full, and again after the bladder has been emptied. If there is a diverticulum present, it will remain full of medium after the medium has left the main cavity (p. 1139). The extent of a very large neoplasm of the bladder can sometimes be demonstrated better by cystography than by cystoscopy.

(c) *Micturating cystograms* are best performed with an X-ray image-intensifier. The function of the bladder neck, and the presence of ureteric reflux of urine, can be observed on a monitoring screen as the patient empties his bladder.

Urethroscopy: (a) *Anterior urethroscopy* is employed when the presence of a urethral stricture (fig. 1382) or strictures is suspected, or in cases of chronic urethritis, in order to exclude or confirm the presence of an infected urethral crypt or a granuloma (a 'soft' stricture). Anterior urethroscopy is conducted under air inflation of the urethra.

(b) *Posterior urethroscopy* permits inspection of the prostatic and membranous urethræ. It is carried out with an irrigating urethroscope. The most notable normal spectacle of posterior urethroscopy is the verumontanum, which presents as an eminence on the floor of the prostatic urethra. On the summit of this projection is the sinus pocularis (fig. 1389). Into the sinus pocularis open the ejaculatory ducts, the orifices of which can be seen but rarely. The verumontanum is reddened and enlarged in cases of chronic vesiculitis (fig. 1390). In cases of chronic prostatitis, one or more of the numerous prostatic ducts, which normally are difficult to visualise, may be seen exuding pus. When the prostate is enlarged, it can be seen as bulging of the floor and lateral walls of the urethra in the vicinity of the internal urinary meatus.



FIG. 1388.—Normal retrograde pyelogram. The definition is much clearer, consequently this procedure is invaluable in confirming doubtful abnormalities visualised by the excretory method.

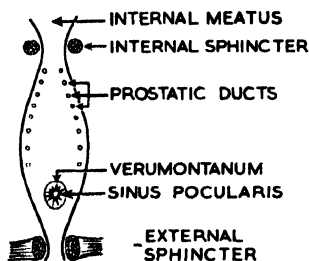


FIG. 1389.—Structures on the floor of the posterior urethra. (After J. C. Ainsworth-Davis, F.R.C.S.)

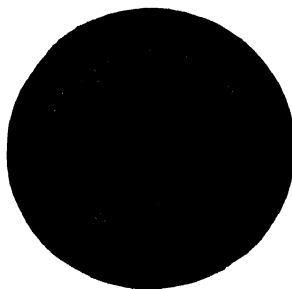


FIG. 1390.—Inflamed swollen verumontanum seen through a posterior urethroscope.

Urethrography.—Urethrography (fig. 1391) is especially valuable

for gaining information concerning the length of a urethral stricture, of dilatation or diverticulum formation above a stricture, or of failure of the medium to pass a stricture. It also reveals dilated prostatic ducts in chronic prostatitis and especially in tuberculous prostatitis. It is valuable for determining the presence of contraction of the bladder neck. The one contra-indication to its use is the presence of urethral hæmorrhage. The medium employed must be chosen with great care, for should there be a breach in the continuity of the lining mem-



FIG. 1391.—Normal urethrograph.

brane of the urethra, the medium will enter the circulation.

Lipiodol brings with it the danger of oil embolus and this medium should never be employed. Even worse is to inject barium emulsion, and deaths have occurred from its use in cases of ruptured urethra.

The medium must therefore be harmless should some of it enter the circulation, and the injection must not be rapid or forceful. Perabrodil (80 per cent.) complies with these stipulations as it can be injected intravenously. For general purposes the most satisfactory medium is Umbradil viscous V. This is a jelly which is squeezed from a tube into the butt end of a urethral syringe. It is injected easily and contains the local anæsthetic lignocaine. The injection is made most satisfactorily by employing Knutsson's apparatus which has a penile clamp attached (fig. 1392).

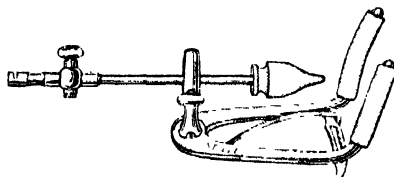


FIG. 1392.—Knutsson's apparatus for urethrography.

Renal Arteriography (fig. 119)

Renal arteriography can provide information unobtainable by retrograde pycelography. To be enabled to visualise the renal arterial architecture is of diagnostic value in the following conditions.

1. Hypertension, if unilateral ischæmia (e.g. due to renal artery stenosis) is suspected.
2. Renal neoplasm. 3. Renal cyst. 4. Chronic pyelonephritis. 5. Aneurism of the renal artery.

There are two methods of performing renal arteriography.

(a) **Translumbar aortography** is carried out under general anæsthesia. A needle is passed into the abdominal aorta just above the renal arteries (level of L.I.), the contrast medium is injected and four films are taken in rapid succession. To minimise

accidents a small quantity of medium is injected and a film taken to ensure that the needle is in its correct position (p. 120).

(b) **Retrograde Arteriography.**—A Seldinger's needle is used (fig. 120). The needle is passed into the femoral artery. The stylette is removed. A spiral guide wire (a 'leader') with a supple tip is passed into the artery and the needle is removed. The artery is compressed to avoid bleeding and polythene tubing is threaded on to the leader and so guided through the skin and arterial puncture into the artery. The polythene tube is advanced to the desired level, a small quantity of Hypaque being injected to render it opaque to X-rays. The medium is then injected. *Selective Renal Arteriography* can be achieved by using a hooked polythene tube, and guiding it into the renal artery (fig. 1393).



(a)



(b)

FIG. 1393.—Selective renal arteriography. (a) normal kidney. (b) renal cyst (p. 1077).

In either method not more than 30 ml. of Hypaque (65 per cent.), one of the least toxic media, should be injected. If these stipulations are adhered to, accidents are minimised. The dangers of the procedure are (a) tubular necrosis of the kidneys, (b) paraplegia, often temporary, probably due to contrast medium producing spasm of the vessels of the spinal cord, (c) elevation of a plaque of atheroma and possibly the production of a dissection in the aortic wall.

Operation should not be performed for at least one week after renal angiography.

Perirenal insufflation of oxygen to delineate a renal or adrenal swelling is described on p. 575.

Renal tomography is sometimes used in combination with other radiographic procedures as it can help in doubtful cases to differentiate between tumours and cysts.

ANURIA (*syn.* SUPPRESSION OF URINE)

Oliguria should be defined as an excretion of less than 300 ml. (11 ounces) of urine in twenty-four hours. Anuria is an absence of excretion for twelve hours.

The most helpful classification of anuria is into pre-renal, renal, and post-renal. Although suppression of urine is commonly and conveniently referred to as anuria, except in post-renal (ureteric obstruction) cases the suppression is seldom absolutely complete.

PRE-RENAL ANURIA

The blood pressure in the glomeruli is normally about 90 mm. of mercury; when the systolic blood pressure falls below 70 mm., filtration from glomeruli ceases. If the glomeruli are diseased, a higher pressure (up to 100 mm. of mercury) may be inadequate to maintain filtration. The causes of pre-renal anuria are traumatic shock, severe hæmorrhage, spinal anæsthesia, extensive

burns, dehydration from vomiting, diarrhoea, or excessive sweating, and cardiac failure.

Treatment.—Blood transfusion in the case of hæmorrhage, the treatment of shock (p. 79) if that be the cause of the fall in the blood-pressure, or in cases of dehydration the administration of dextrose-saline solution intravenously often restores urinary excretion. If hypotension is long maintained, damage to the renal epithelium results, and the condition passes on to one of renal anuria.

ACUTE RENAL ANURIA

Acute renal anuria results from damage to, or ischæmia of, the renal tubular epithelium (fig. 1394). The principal causes met with in surgical practice are:

1. Severe shock (hypotension) lasting two hours or more.
2. Incompatible blood transfusion.
3. Ultra-acute pyelonephritis, occurring with retention of bladder urine.
4. The crush syndrome (p. 80).
5. Concealed accidental hæmorrhage and abortion.
6. Certain poisons. The most important are (a) media used for aortography, (b) the toxin of eclampsia, (c) chemicals—mercury salts, carbon tetrachloride.
7. Acute pancreatitis.
8. Operations on jaundiced patients.
9. Bacteræmic shock.

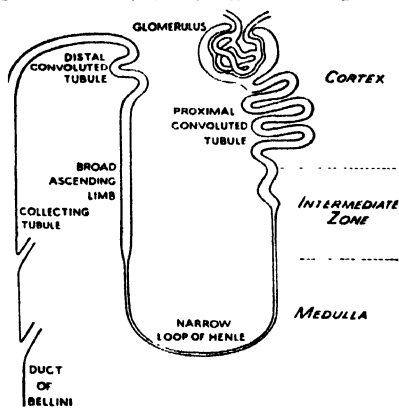


FIG. 1394.—Necrosis of the proximal convoluted tubule produces immediate anuria. (After H. L. Sheehan.)

When renal anuria is reversible, and it is treated correctly, the condition is divided into three phases: 1. *The oliguric phase.* 2. *The phase of diuresis.* 3. *The phase of recovery.*

Clinical Features.—The average duration of the oliguric phase is ten to twelve days. Dark urine in small amounts and with a specific gravity of 1.010 is passed, or will be found on catheterisation. Anorexia is an early symptom which is followed by hiccough. Within four or five days there is copious effortless vomiting. Abdominal distension is common. Untreated, or treated incorrectly, the blood urea mounts by 20 to 30 mg. daily (fig. 1395). The systolic blood-pressure may be elevated—200 mm. Hg. after two or three days.

Still untreated, or treated incorrectly, about the sixth day increasing drowsiness, thirst, a dry skin, and a dry brown tongue are characteristic findings. The respiratory and pulse-rates become slower and irregular, and Cheyne-Stokes'

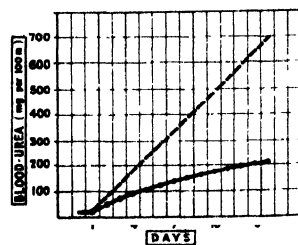


FIG. 1395.—Dotted line: rise of blood urea in untreated anuria. Solid line: same when treated by fluid limitation and protein-free diet. (After G. M. Bull.)

Lorenzo Bellini, 1643–1704. Professor of Anatomy, Pisa.

Friedrich Gustav Jacob Henle, 1809–1885. Professor of Anatomy at, successively, Zürich, Heidelberg, and Göttinge

John Cheyne, 1777–1836. Physician-General to the Forces in Ireland.

William Stokes, 1804–1878. Regius Professor of Medicine, Dublin.

respirations are often in evidence. The final stages are usually characterised by muttering delirium followed by coma.

Treatment: Special Early Measures.—(a) If there is any doubt as to the cause, post-renal (obstructive) anuria must be eliminated by plain radiography and ureteric catheterisation (p. 1166). (b) In cases of incompatible blood transfusion, renal damage can sometimes be prevented by provoking a diuresis within an hour of the reaction.—500 ml. of Rheomacrodex (p. 77) or mannitol (p. 77) is given intravenously. If no urine is secreted (the bladder is emptied naturally—or via a catheter), all fluids are restricted forthwith.

In all other circumstances of acute renal anuria it is imperative not to force fluids.¹ The important substances in the genesis of uræmia are not urea and other end products of nitrogen metabolism, but water and electrolytes, such as potassium. If patients receive 3,500 ml. or more per day, 75 per cent. die with signs of water overload, and evidence of pulmonary or cerebral œdema at necropsy. Another reprehensible practice is to prescribe potassium citrate for a patient on the threshold of suppression of urine; it has caused many deaths from hyperkalæmia.

Treatment in Practice.—The first three essentials are:

1. *Water Balance.*—In the absence of vomiting, the elimination of water by an anuric patient is limited to extra-renal routes, i.e. lungs, skin, and fæces. In a temperate climate this loss is 600 to 1,000 ml. per day. As it is estimated that there is a daily production of 400 ml. of water from the oxidation of body fat and proteins, the daily fluid intake of an anuric patient must be limited to 500 ml. plus an amount equal to that of the water vomited or recovered by gastric aspiration.

Additions must be made for diarrhœa and excessive sweating: regarding the latter, an extra 200 ml. is allowed for each degree of the patient's temperature above 100° F. (37·8° C.). When the weather is exceedingly hot, the allowance is increased, but even in these circumstances it should rarely exceed 1,000 ml. per twenty-four hours.

Bedside evaluation—thirst, moisture of the tongue, and the skin—are excellent guides of adequate hydration.

2. *Electrolytic Balance.*—In the absence of diarrhœa and vomiting, the only effective channel for excretion of electrolytes is by the kidneys. Consequently, in anuria the administration of electrolytes often leads to gross disturbances of electrolytic balance; in particular, hyperpotassæmia is liable to cause sudden death from cardiac arrest. *Therefore the patient should not receive any electrolytes until diuresis recommences.*

3. *Nitrogen Metabolism.*—The end-products of protein katabolism, other than urea, are possibly toxic. Furthermore, by the breakdown of both exogenous and endogenous protein, potassium is liberated. For these reasons it is essential to prohibit protein intake altogether, and to reduce endogenous nitrogen metabolism as much as possible. A high carbohydrate intake depresses endogenous nitrogen metabolism.

¹ According to Harrington, the pioneer in water closets, it does no good to keep trying to flush a lavatory when it is blocked.

The Protein-free, Mineral-free, Strictly-limited-fluid Regimen.—

The object is to provide 2,000 calories per day, and at the same time to limit fluid intake as detailed above. This is a diet upon which the patient can subsist without discomfort for periods up to three weeks (Bull). According to circumstances, the regimen can be varied:

(a) When the patient can take fluid by mouth without vomiting or undue nausea, a daily intake of 500 ml. of 40 per cent. lactose, given in small frequent amounts, is likely to be acceptable because the solution tastes less sweet than dextrose.

(b) When the patient is anorexic, nauseated, and prone to vomit, a plastic gastric tube is passed transnasally and the 500 ml. of 40 per cent. dextrose or lactose is gravitated by the drip method. A plastic tube is chosen because its relative stiffness makes it less likely to be expelled if the patient vomits. As far as possible the vomitus must be collected and measured, for obviously it is highly important to supplement the loss with precision. To stop the drip for two to four hours at a time helps some patients to retain the solution. Alternatively, 50 to 100 ml. of doubly centrifuged cream is added to the dextrose solution. This enables the solution to be tolerated by some patients who otherwise would eject it.

(c) When the patient is unable to retain the fluid gravitated into the stomach, or toxæmia or a head injury makes him stuporous and therefore liable to inhale vomitus, there is but one alternative—to give the 40 per cent. dextrose solution intravenously. The modern method is to give this clot-provoking solution into the inferior or superior vena cava by a polythene tube passed from the long saphenous or cubital vein respectively.

In order to discourage thrombosis, heparin (1,000 units per 500 ml.) is added to the infusion. Even so post-mortem examination has shown intravenous clotting in a high percentage of cases. For this reason the intravenous route should be employed only in cases of absolute necessity. Some advise passing the polythene tube but a few inches into a peripheral vein and to change the vein if clotting occurs. By gravitating the fluid in this way it is believed that the menace of pulmonary embolism is lessened.

Vitamins.—Irrespective as to whether the alimentary or the intravenous route is employed, the daily vitamin intake should be identical with that of a patient subsisting wholly on parenteral feeding (pp. 89 to 94).

Prevention of Infection.—Anuric patients become infected easily, therefore all who come in contact with the patient should be gowned and masked. A nasal swab should be taken from the patient at the onset of anuria. If *Staphylococcus aureus* is cultured, a suitable antibiotic should be given at once. Penicillin, 1 mega unit per twenty-four hours, can safely be given but all other antibiotics are cumulative even when given in greatly reduced doses. Erythromycin 1 G. daily is the least harmful.

Watching for Hyperkalæmia.—Serial electrocardiographs are a better check to the development of this complication than reliance on plasma potassium determinations (p. 88).

Diuretic Phase.—In reversible lesions, usually about the eighth day, the epithelium of the lower nephrons regenerates sufficiently to prevent resorp-

tion of the glomerular filtrate, and a little urine is passed. This is a most hopeful sign, and given correct management and previously healthy kidneys (Causes 1 to 7, p. 1068) an increasing twenty-four-hourly output during the subsequent week is usual. When diuresis commences, an amount of water equal to the output of urine for the previous twenty-four hours is added to the daily allowance. It is more difficult to estimate an appropriate allowance of electrolytes, for the restitution of renal electrolyte-regulating function returns slowly. A heavy loss of sodium and potassium may occur and must be replaced. Only when renal excretion reaches 1 litre per day does the blood-urea level commence to fall. Once diuresis exceeds 1 litre in twenty-four hours, the intragastric tube is removed and the patient is fed on a high caloric, low-protein diet containing an adequate daily amount of mineral salts.

Other methods of treatment in special circumstances :

1. **The Artificial Kidney.**—The principle of the artificial kidney is that of dialysis across a membrane which is permeable to crystalloids and not to colloids, with the patient's blood on one side of the membrane and a saline bath on the other; excessive crystalloids will leave the blood and enter the bath, which is constantly flowing. The indications for dialysis are: (a) Serum potassium above 7.0 mEq./litre, (b) Serum bicarbonate level below 10 m Eq./litre, (c) Gross overhydration, or (d) a blood urea above 400 mg. per cent. Fits or coma may also be indications.

The artificial kidney requires a trained team to operate the machine, hence it is available only in a few centres. It is therefore necessary to describe other methods of ridding the patient of excessive electrolytes, particularly potassium.

2. **Peritoneal Dialysis.**—This method has been used with success. The simplest method and the one least likely to give rise to peritonitis is to insert a polythene tube into the peritoneal cavity by means of a trocar and cannula. Into the peritoneal cavity is gravitated moderately slowly a hypertonic solution (20 per cent. dextrose solution with 6 G. sodium chloride per litre added). The fluid is retained by clipping the tube for two hours and then allowing it to flow out. The process is continued for up to forty-eight hours. Thus the peritoneum acts as a dialysing membrane.

3. **Ion Exchange Resins.**—If the patient does not vomit, then ion exchange resins in the sodium phase have been used successfully to withdraw potassium ions into the intestine in exchange for sodium ions. Five grammes of Resonium, dissolved in the patient's allowance of dextrose solution, is given eight times a day. The resins are of no avail given *per rectum*.

4. **Exchange Transfusion.**—The principal indication is in the initial phase of anuria following incompatible blood transfusion, the object being to remove free hæmoglobin, hæmolytic toxins, and damaged erythrocytes.

POST-RENAL (*syn.* OBSTRUCTIVE) ANURIA

Calculous anuria arises in one of the following ways :

1. A calculus becomes impacted in the ureter of the only functioning kidney, the other kidney being congenitally absent, previously removed, or destroyed by disease.

2. Both ureters become obstructed by stones.

3. A calculus blocks one ureter, and the contralateral organ, seldom completely normal, also ceases to function (reflex anuria).

Clinical Features.—Usually the patient is a man between forty and sixty years of age with a long history of urinary lithiasis, but in about 20 per cent. of cases calculous anuria arises without previous symptoms.

Onset.—Typically an attack of renal colic precedes the anuria, the onset of which is sudden. This pain may disappear and be replaced by a constant severe pain in the loin. In a few cases there is little, if any, pain, and the anuria is preceded by several days of increasing oliguria. The latter variety is usually due to a superimposition of pyelonephritis on kidneys (or a single existing kidney) which have been functioning poorly on account of partial calculus obstruction.

Stage of tolerance usually lasts from three to six days. The patient feels comparatively well and may continue his work. During this time the blood urea mounts steadily, and as it does so headache, sleeplessness, constipation, and lassitude supervene. By the end of this stage the blood urea is often over 200 mg. per cent.

Stage of uræmia follows, and is characterised by the same clinical features described in late renal anuria.

Examination.—The obstructed kidney is tender and there is guarding by the overlying muscles. A large palpable kidney is probably functionless and the seat of a hydronephrosis or pyonephrosis; a recently obstructed kidney is never enlarged.

Action to be taken:

(1) Pass a catheter to exclude retention. A few ounces of blood-stained urine may be obtained.

(2) Plain X-ray may show the obstructing calculus, but more often visualisation is difficult. This is because the small size of the stone (it is seldom larger than an orange-pip), makes it difficult to see against superimposed bone and intestinal gas shadows. Sometimes a large branched renal calculus is displayed on the functioning side.

(3) Cystoscopy. Pass a catheter up both ureters. This may pass or dislodge the stone and the emergency is over. Leave the catheter in position

to drain, and when the general condition of the patient is satisfactory, remove the calculus by open uretero-lithotomy. (Just prior to this, further radiographs must be taken to confirm the site of the stone. An I.V.P. is permissible and helpful if the blood urea has fallen to within normal limits.) Occasionally at cystoscopy a stone is seen wedged in the ureteric orifice (fig. 1396) whereupon attempts should be made to dislodge the stone with a ureteric catheter, by diathermy of the orifice, or by insertion of a Dormia stone-catching basket (fig. 1441).

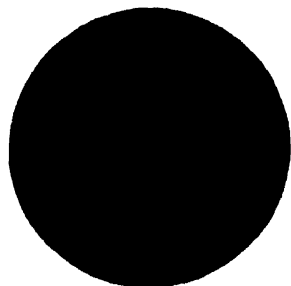


FIG. 1396.—Stone impacted in the ureteric orifice as seen by cystoscopy.

(4) Operation—if a catheter will not pass. In this case the stone should be removed by open operation. If no stone is visualised by the X-ray, then the kidney on the side which was most painful

should be explored. Pyelostomy, or nephrostomy (fig. 1397) may be necessary. If the kidney explored is found to be hopelessly diseased, nephrostomy must be performed on the other side.

(5) When the obstruction has been relieved either by ureteric catheterisation or operation, and until diuresis is established, the general treatment is similar to that described on p. 1069. In cases where nephrostomy has been necessary, preparations should be made for blood transfusion, for sometimes the hæmorrhage from the incised oedematous kidney is excessive.

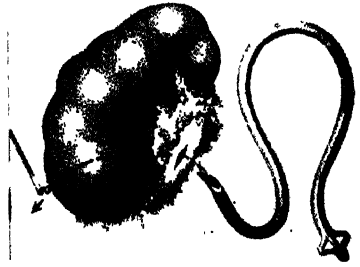


FIG. 1397.—Cabor's method of performing nephrostomy.

Anuria due to Sulphonamide Crystalluria.—Sulphapyridine, sulphadiazine, and occasionally sulphathiazole in the presence of an acid medium are changed into acetyl salts which are insoluble. Crystals (fig. 1398) are deposited in the kidney tubules and ureters. The condition is now infrequent, and occurs mainly in patients with partial obstruction to the kidneys, and in those who are dehydrated.



FIG. 1398.—Acetylated sulphapyridine crystals resemble small wheat sheaves.

Treatment.—When a patient undergoing sulphonamide therapy develops renal colic and oliguria, the drug should be withheld and the high fluid intake further increased. Should anuria supervene, firm deep massage is carried out on each ureter, from above downwards. The lower ends of the ureters are then massaged per rectum. If this is not followed by the passage of urine, there should be no delay in performing cystoscopy and attempting to catheterise the ureters. Provided the catheters can be inserted, the kidney pelvises are washed out with 2·5 per cent. sodium bicarbonate solution. The crystalline mass can, on occasions, be dislodged with a stone dislodger (p. 1099). As an alternative means to

cystoscopic manœuvres, many favourable results have followed splanchnic block anæsthesia. If these measures are unsuccessful, unilateral or bilateral pyelostomy must be performed.

Anuria due to Accidental Ligation of the Ureters.—This is a hazard mainly of hysterectomy. Bilateral pyelostomy should be performed in the first instance. Only when the patient is out of immediate danger should an operation to reconstruct or implant the ureters be performed (p. 1082).

Anuria due to Involvement of both Ureters in a Neoplastic Process, e.g. Carcinoma of the Cervix.—Excluding terminal cases, the only treatment is transplantation of ureters into the bowel or bilateral nephrostomy.

Anuria due to Involvement of Ureters in Retroperitoneal Fibrosis (p. 1099).

Hugh Cabot, 1872–1945. Surgeon, The Mayo Clinic, Rochester, U.S.A.

CHAPTER 45

THE KIDNEYS AND URETERS

Embryology.—A bud from the lower end of the mesonephric (Wolfian) duct grows backwards and upwards behind the peritoneum to the lumbar region (fig. 1399). The stalk of the bud forms the ureter and its dilated extremity the kidney pelvis. Sometimes the bud is duplicated or the stalk becomes bifurcated, giving rise to congenital anomalies (see below). From six weeks to five months, the primitive renal pelvis divides repeatedly to form generations of collecting tubules, e.g. 1st

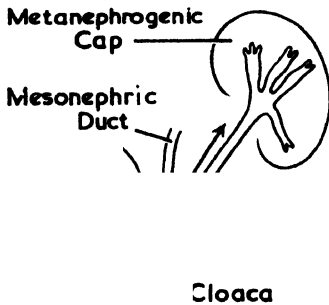


FIG. 1399.—The development of the kidney.

generation, 2nd generation, etc. Each collecting tubule is capped by mesoblast, which becomes the glomerulus and the convoluted tubule. The first three or four generations persist only for a short period as cystic structures before they degenerate and disappear. It is the continued persistence of these provisional structures that gives rise to the cysts of congenital cystic kidneys. If only one cyst fails to degenerate, a solitary cyst of the kidney results.

The foetal kidney is at first lobulated, but in the human the lobules become welded together by the growth of a new cortex beneath the capsule. In some mammals, e.g. oxen and bears, foetal lobulation is retained throughout life.

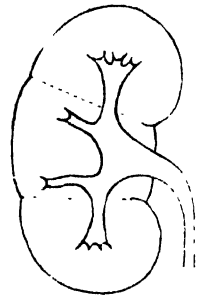


FIG. 1400.—The three divisions of the kidney render resection (partial nephrectomy) of the upper or lower division a practical proposition. (After Carl Semb.)

Surgical Anatomy.—The parenchyma of each kidney is drained by seven pairs of calyces, three in the upper and two each in the middle and lower segments (fig. 1400). Each of the three segments represents an anatomical and physiological unit with its own blood supply, an important point in view of the increased indications for partial nephrectomy. (Carl Semb).

Surgical Physiology.—Ciné-pyelography indicates that there are sphincters at the junction of the minor and major calyces, and at the pelvi-ureteric junction. Each segment of the calyceal system fills before its contents are passed into the next chamber. Should this mechanism be under nervous control (and some believe that it is) it is an argument in favour of renal pedicle sympathectomy for the cure of non-mechanical hydronephrosis.

CONGENITAL ABNORMALITIES OF THE KIDNEY

Absence of One Kidney.—Sometimes pyelography reveals only one functioning kidney, and at cystoscopy only one ureteric orifice is present, alternatively a ureter and pelvis are present on the non-functioning side, but the parenchyma is almost or entirely absent. In either case the functioning kidney is hypertrophied. An absent or congenitally atrophic kidney is present in about 1:1,400 individuals.

Renal ectopia occurs once in 1,000 cases. The kidney is arrested in some part of its normal ascent, usually at the brim of the pelvis. As a rule the kidney of the opposite side is present and in its normal position. The left kidney is ectopic far more often than the right; the reason is unknown.

When an ectopic kidney becomes inflamed, the tender lump to which it gives rise frequently causes supreme difficulty in diagnosis; for instance, when right-sided it is liable to be mistaken for an appendix abscess.

Horse-shoe Kidney.—The most medial subdivisions of the primary mesonephric bud of each side fuse and the kidneys fail to ascend completely. The adrenal glands, being developed separately, are in their normal positions. The abnormality (found once in every 1,000 necropsies) is more common in the male. Usually the bridge joining the lower poles lies in front of the fourth lumbar vertebra. Fusion occurs very early, when the embryo is but thirty to forty days old, at which time the two masses of mesoblast destined to form the kidneys lie very close together. Exceptionally, it is the upper poles that are fused.

Clinical Features.—Horse-shoe kidneys are notoriously prone to become diseased, largely because the ureters are angulated as they pass over the fused isthmus (fig. 1401). This produces urinary stasis; consequently simple infection, tuberculosis, and calculus formation are common complications.



FIG. 1402.—Pyelogram of a horse-shoe kidney. Only rarely are all the calyces directed towards the spinal column. (A. Jacobs, F.R.C.S., Glasgow.)

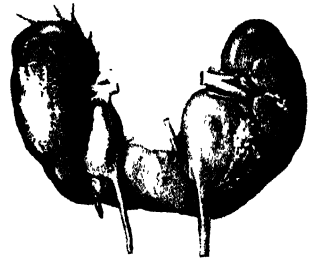


FIG. 1401.—Horse-shoe kidney. Note the ureters passing in front of the fused lower poles.

Although a fixed mass below the umbilicus may suggest a horse-shoe kidney, the final diagnosis is established by pyelographic data. The most characteristic finding is that the lowest calyx on each side is reversed in position (i.e. directed towards the vertebral column. Rarely most, or all, of the calyces are reversed (fig. 1402). In a large percentage of cases the ureters curve like a flower vase. While horse-shoe kidney is not a contraindication to pregnancy, urinary complications are more frequent.

Treatment.—In cases giving rise to pain, or when non-specific infection is present, division of the isthmus is usually curative. The operation can be conducted extra-peritoneally, but a trans-peritoneal approach gives a much better view of the renal arteries and veins, and it is easier to decide whether a nephropexy is desirable. The area can be drained extra-peritoneally. When one half of a horse-shoe kidney is irreparably diseased, e.g. calculi or neoplasm, heminephrectomy should be performed provided the remaining half has adequate function.

Unilateral fusion (*syn.* crossed dystrophia) is rare. Both kidneys are situated in one loin. One kidney, carrying its own blood-vessels and with the ureter opening into the bladder in a normal position, crosses the middle line, and its upper pole fuses with the lower pole of the normally placed kidney. In this instance both kidney pelves are situated one above the other medial to the renal parenchyma (unilateral long kidney). In some cases the pelvis of the crossed kidney faces laterally (unilateral S-shaped kidney (fig. 1403)).

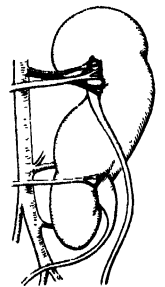


FIG. 1403.—Unilateral S-fusion of the kidneys.

Congenital Cystic Kidneys (*syn.* Polycystic Kidneys).—In 18 per cent. of cases there is a congenital cystic liver;

occasionally the pancreas and lung are affected similarly. The disease is hereditary and can be transmitted by either parent (see Embryology, p. 1074).

Pathology.—The organs may become enormously enlarged. The surface gives an appearance of many bubbles. On section the renal parenchyma is riddled with cysts of varying sizes (fig. 1404), some containing clear fluid, others thick brown material, and still others coagulated blood.

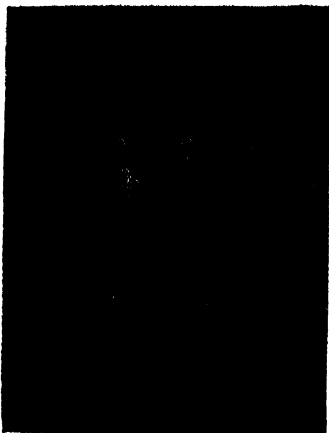


FIG. 1404.—A congenital cystic kidney removed on account of profuse hæmaturia.

Clinical Features.—**In the Fœtus.**—The kidneys may be so large as to obstruct labour. A quarter of the cases are stillborn or die shortly after birth.

In the Infant.—The bilateral swellings of congenital cystic kidney must be distinguished from those of bilateral Wilms's tumour (p. 1114). In cases of congenital cystic kidneys presenting during this period, renal rickets often develops a few years before the final uræmia.

Between the ages of three and thirty, cases of congenital cystic kidneys are encountered but rarely.

In the Adult.—The condition is slightly more common in women than in men. There are six clinical features :

(a) **Renal Enlargement.**—The large knobby kidneys when discovered in the course of a routine examination can hardly be mistaken. Sometimes congenital cystic kidneys are revealed only at laparotomy for some other condition. **Unilateral Renal Swelling.**—One kidney contains larger cysts than the other, and the physical signs are similar to those of a renal neoplasm.

Patients with congenital cystic kidneys pass abundant urine of low specific gravity (1.010 or less) containing a slight trace of albumin but neither casts nor cells. On cystoscopy there is often considerable delay in the excretion of indigo-carmin, even in cases with a normal or only slightly elevated blood urea.

(b) **Pain** is due either to the weight of the organ dragging upon its pedicle or to tension within the cysts. Often the pain is a dull ache in the loin ; sometimes it takes the form of renal colic, when a calculus, which is not rare in this condition, should be suspected.

(c) **Hæmaturia.**—In about 25 per cent. of cases, as a result of over-distension, one of the cysts ruptures into the renal pelvis and causes hæmaturia. Usually moderate hæmaturia lasts for a few days, and recurs at varying intervals ; sometimes it is profuse.

(d) **Infection.**—The most common complication of congenital cystic kidneys in adult life is pyelonephritis.

(e) **Hypertension.**—About 75 per cent. of patients with congenital cystic kidneys above the age of twenty have hypertension. Why some escape this

complication is not clear. Possibly the high blood pressure is due to a separate genetic factor frequently linked with congenital cystic kidneys.

(f) *Uræmia*.—The patient complains of anorexia, headache, and indefinite gastric symptoms, and is frequently given a placebo, until persistent symptoms demand a complete clinical examination. Later drowsiness and vomiting occur. Signs of uræmia often commence suddenly during middle life; only one quarter of patients with this condition survive the age of fifty-five years. Severe anæmia is common.

Pyelography.—Excretory pyelography is the best way of confirming the diagnosis, but when the concentration of Hypaque is insufficient to cast a clear shadow, retrograde pyelography is necessary. Only one side should be injected, the other being deferred for a week lest uræmia be induced thereby. The shadows of the kidneys are enlarged in all directions. The renal pelvis is elongated and may be compressed. The calyces are stretched over the cysts and are often narrow (like the legs of a spider) or bell-like (fig. 1405).

Treatment :

(a) *Expectant*.—Routinely the patient should drink a large quantity of water, have a low-protein diet, and take iron to prevent anæmia. Infection, which is common, should be treated by an appropriate drug.

(b) *Operative*.—By relieving pressure on the remaining renal parenchyma, Rovsing's operation, if performed early, improves renal function. In many instances, life is prolonged five years or more.

Rovsing's Operation.—One kidney and then the other is exposed. The cysts are incised with a narrow-bladed scalpel, first on the convex border, then on the posterior surface and, finally, as the kidney becomes more manageable in size, the peritoneum is peeled away and the anterior surface is dealt with similarly.

So improved by bilateral Rovsing's operation was one of our patients who was admitted in a state of uræmia, that two and a half years later he was serving in the Life Guards ! Rovsing's operation should be performed, not as a last resort, but as soon as possible after the condition is recognised.

Solitary Renal Cyst.—While the term 'solitary' serves to distinguish the condition from congenital cystic disease of the kidneys, it is found that not infrequently one or two similar but smaller cysts are also present.

Ætiology.—The origin of the cyst may be identical (but on a smaller scale) with that of congenital cystic kidney (p. 1075) ; or it could arise from bygone trauma or infection causing blockage of a tubule. The mature age at which the cyst is usually found is in favour of the latter supposition.

Clinical Features.—There are no pathognomonic symptoms or signs. Sometimes there are no symptoms until a swelling is noticed, at others there is a dull ache in the loin. The cyst can become infected and if a swelling is palpable, and particularly if the swelling is tender, the creditable diagnosis of pyonephrosis will be made. A spontaneous hæmorrhage into the cyst occurs from time to time, in which case sudden renal pain is likely to be experienced. Occasionally the cyst presses on the pelvi-ureteric junction and causes urinary symptoms.



FIG. 1405.—Polycystic kidney. Pyelographic appearance. Note length of kidney and elongated bell-like calyces stretched over cysts.

Radiology.—(a) Pyelography reveals a filling defect of one or more calyces simulating the deformity produced by a neoplasm, but typically more rounded. (b) It is possible to distinguish a renal cyst from a renal neoplasm by selective renal arteriography (fig. 1393).

Treatment.—Exploration should be advised in every case. Renal cysts can be caused by a tumour blocking tubules and interfering with the vascular supply to the same area of the kidney. Having displayed the kidney the operation is varied according to circumstances.

(a) *Kirwin's Method.*—When the cyst is blue-domed and aspiration reveals clear fluid, that portion of the cyst wall lying above the surface is excised. The remaining portion of the cyst is swabbed with phenol in glycerine equal parts, followed by the application of 95 per cent. alcohol. The cavity is filled with perinephric fat and the edges of the cavity are approximated. The phenol destroys the lining membrane.

(b) *Partial nephrectomy* is performed if the contents of the cyst are blood-stained (one-third of such cysts contain a papilliferous neoplasm).

(c) *Nephrectomy* is performed if there are indications that the cyst is a malignant degeneration of a neoplasm, e.g. irregular induration at the periphery of the cyst.

Differential Diagnosis.—In sheep-raising districts, *hydatid cyst of the kidney* is common. On the right side (if hydatid disease is suspected) the swelling is liable to be mistaken for a hydatid cyst of the liver. Occasionally the patient complains of passing 'grape skins' (ruptured daughter cysts) in the urine. The treatment is excision of the cyst, but in many cases the cyst is so large that nephrectomy is advisable.

Aberrant renal vessels are found on the left side more frequently than on the right; in females more often than males; unilateral examples are three times more common than bilateral.

Some hold that aberrant renal vessels are a cause of hydronephrosis (p. 1084 and fig. 1142); others believe that aberrant vessels in themselves are seldom a cause of that condition, but that their presence accentuates a hydronephrosis existent on account of another reason (fig. 1418).

Usually the aberrant artery is small; occasionally it is comparatively large, supplying one-quarter of the renal parenchyma. Division will cause infarction of the corresponding portion of renal tissue. On the other hand, an obstructing aberrant renal vein can be divided with impunity, because the venous collateral circulation is very generous.

CONGENITAL ABNORMALITIES OF THE RENAL PELVIS AND URETER

Duplication of a renal pelvis is the most common congenital anomaly of the upper renal tract and is found in about 4 per cent. of patients. It is usually unilateral, and is somewhat more common on the left side than on the right. The upper renal pelvis is comparatively small (fig. 1406) and



FIG. 1406.—Pyelogram showing a kidney with a double pelvis.



FIG. 1407.—Double ureter. Retrograde pyelogram.

drains the upper group of calyces; the larger lower renal pelvis drains the middle and lower groups of calyces.

Duplication of a Ureter.—Double ureters are present in addition to double renal pelvises in about 3 per cent. of cases submitted to pyelography. The ureters often join, usually in the lower third of their course (fig. 1407), and have a common orifice into the bladder. Less frequently the ureters open independently into the bladder (fig. 1408), in which case the ureter draining the upper pelvis crosses its fellow, and opens below and medial to it.

Clinical Features.—In many instances a double renal pelvis or a double ureter is found by chance in the course of an investigation of the urinary organs. A double pelvis is more liable to become the seat of infection, calculus formation, or hydronephrosis than a normal pelvis. Initially such disease is confined to one part of the duplication. In cases of complete duplication of the ureter the lower ureteric orifice is sometimes the site of congenital atresia.

Treatment.—Ureteric meatotomy can be performed in some early cases of stricture of a ureteric meatus. Difficulty is encountered in cases of Y-shaped bifid ureter, because a ureteric catheter cannot be made to enter the smaller orifice. Heminephrectomy with removal of its ureter is eminently satisfactory when disease normally requiring total nephrectomy is confined strictly to one half of a kidney that has a double pelvis. The exception to the rule is, of course, a neoplasm.

Ectopic Ureteric Orifice.—Should a second ureteric bud arise from the mesonephric duct later than usual, the orifice of the accessory ureter is prone to occupy a grossly abnormal position. This is a rare anomaly. In both sexes the existence of double ureter is determined by excretory pyelography.

In the female an ectopic ureter opens either into the urethra below the sphincter urethræ (fig. 1409) or into the vagina, and causes an intractable incontinence of urine.

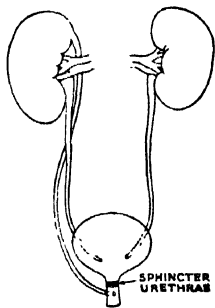


FIG. 1409.—Ectopic ureter in a female.

The diagnosis can nearly always be made from the history alone. A girl or woman who has dribbled for as long as she can remember, despite the fact that she has a desire to void, and does urinate, has an ectopic ureteric orifice. The demonstration of this orifice is often extremely difficult, because it is guarded by a valve. The diagnosis may be established by giving an intravenous injection of indigo-carmin, and placing one swab into the vestibule and another in the vagina; the one coloured blue signifies the position of the ectopic orifice. Sometimes laparotomy is the only way of finding or excluding the additional ureter.

In the male the aberrant opening may be situated at the apex of the trigone, in the posterior urethra, in a seminal vesicle, or in an ejaculatory duct. As in all these diverse situations, the ectopic ureteric orifice is situated above the sphincter urethræ, the male patient is continent, but recurrent acute or chronic pyelonephritis is prone to supervene in the renal tissue served by the ectopic ureter. Posterior urethroscopy, after the intravenous injection of indigo-carmin, is the only means of ascertaining that a male patient has an ectopic ureter.

Treatment.—*In the female* excision of that segment of the kidney served by the ectopic ureter is usually advisable, because it is so often hydronephrotic and chronically infected. In the rare event of the urine draining from it being sterile, the ectopic ureter can be implanted into the bladder.

In the male—Often no treatment is required. Should urinary infection supervene, heminephrectomy is indicated.



FIG. 1408.—Complete duplication of the left ureter as seen through the cystoscope.

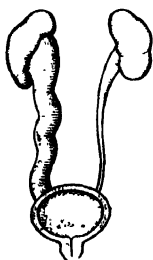


FIG. 1410.—Unilateral congenital mega-ureter.

is likely however, and the condition is better treated by open excision of the lower 1 to 2 cm. of ureter, followed by the formation of a 'nipple' ureterostomy with a long intra-mural course for the ureter. If this fails, total excision of the ureter and replacement with ileum (fig. 1411), or wrapping the ureter in a tube of ileum after removal of the mucosa has been recommended. When the renal parenchyma is severely damaged, nephro-ureterectomy is curative. Occasionally in bilateral cases presacral neurectomy is successful. Bilateral meatotomy is likely to prolong life, but eventually increasing renal failure and pyelonephritis prove fatal.

Post-caval Ureter.—The right ureter passes behind the inferior vena cava (fig. 1412) instead of lying to the right of it. This may cause obstructive symptoms. Should these symptoms warrant it,

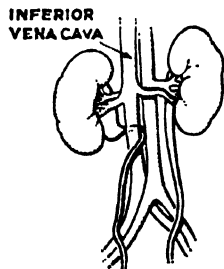


FIG. 1412.—Post-caval ureter.

the ureter can be divided near the bladder, withdrawn behind the inferior vena cava, and reimplanted into the bladder. **Congenital atresia of a ureteric orifice** is a precursor of a ureterocele and is found in children undergoing cystoscopy for increased frequency of micturition or renal colic. Ureteric meatotomy (p. 1099) often remedies the condition.

Ureterocele is due to congenital atresia of a ureteric orifice which causes a cystic enlargement of the intramural portion of the ureter. Usually the wall of the cyst is composed of mucous membrane only; infrequently the muscle coat is included. This condition may be discovered in childhood; often it is not recognised until adult life. Women are more often affected than men, and in 10 per cent. of cases the condition is bilateral. A prominent symptom is increased frequency of micturition, but there may be renal colic and hæmaturia. In long-standing cases secondary infection is often present. The cystoscopic findings are characteristic: when the wall consists of mucous membrane there is a translucent cyst over which blood-vessels radiate (fig. 1413); in the rare variety containing muscle in the wall, the cyst is opaque. In either case the sac is seen to enlarge rapidly with each efflux of urine, and then slowly to collapse. The pyelographic findings are characteristic (fig. 1414).

Treatment.—Many cases are symptomless and no treatment is required. Hydronephrosis, stone, or recurrent infection may necessitate treatment. Cauterising a hole through the wall of the cyst with a diathermy electrode is a simple and satisfactory procedure. A micturating cystogram should be performed three months later to exclude urinary reflux. In advanced unilateral cases associated with a hydro- or pyonephrosis and dilatation of the whole of the ureter, nephrectomy is called for.

Congenital mega-ureter may be unilateral (fig. 1410) or bilateral, and in late stages is accompanied by hydronephrosis. Often the condition is symptomless until infection has occurred. If, however, the patient experiences pain, a diagnosis can be made before the onset of infection. The ureteric orifice on the affected side is normal in size and shape, but it is immotile, the efflux being a continuous trickle instead of intermittent ejections. A ureteric catheter passes easily. Excretory pyelography shows the ureter greatly dilated.

Treatment.—In infected cases, per-cystoscopic meatotomy and drainage of the dilated ureter with a ureteric catheter for about six weeks has been found to be effective in clearing up the infection. Reflux afterwards

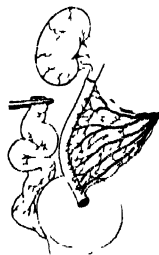


FIG. 1411.—Substitution of a segment of ileum for the mega-ureter, which is shown attached to the bladder.

is likely however, and the condition is better treated by open excision of the lower 1 to 2 cm. of ureter, followed by the formation of a 'nipple' ureterostomy with a long intra-mural course for the ureter. If this fails, total excision of the ureter and replacement with ileum (fig. 1411), or wrapping the ureter in a tube of ileum after removal of the mucosa has been recommended. When the renal parenchyma is severely damaged, nephro-ureterectomy is curative. Occasionally in bilateral cases presacral neurectomy is successful. Bilateral meatotomy is likely to prolong life, but eventually increasing renal failure and pyelonephritis prove fatal.

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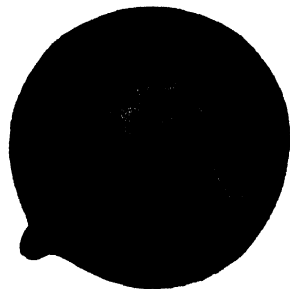


FIG. 1413.—Right-sided ureterocele. (After J. C. Ainsworth-Davies, F.R.C.S., London.)

INJURIES TO THE KIDNEY

In civil life blows or falls upon the loin are the most fruitful sources of such injuries, while blows from in front and crushing accidents add their quota (road traffic accidents). Hæmaturia following minor injuries should suggest the possibility of pre-existing renal abnormality, e.g. stone, hydronephrosis, or tuberculous.

The degree of injury varies considerably from a small subcapsular hæmatoma to a complete tear involving the whole thickness of the kidney (fig. 1415); in addition, the kidney may be partially or wholly avulsed from its pedicle. Tears of the renal parenchyma follow the lines of the uriniferous tubules. The whole of one pole may be detached.

The injury is extraperitoneal, except very occasionally in children in whom there is little perinephric fat; in these cases the peritoneum, being intimately related to the kidney, is liable to be torn in addition to the renal capsule, allowing blood and, perhaps, urine to escape into the peritoneal cavity.

Clinical Features.—Rarely is there superficial bruising, but there are likely to be local pain and tenderness.

Hæmaturia is a cardinal sign of a damaged kidney, but it may not make its

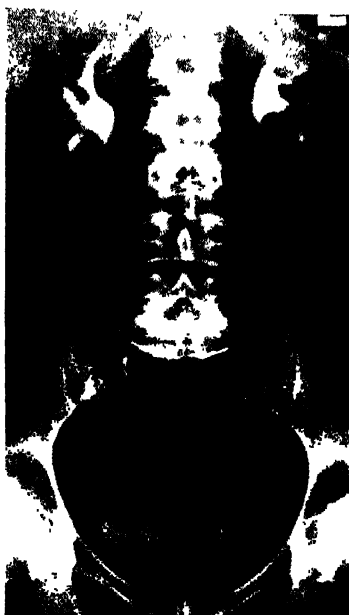


FIG. 1414.—Intravenous pyelographic appearance of ureteroceles. Typical adder-head appearance of lower ureters which remain filled with Hypaque (diatrizoate).

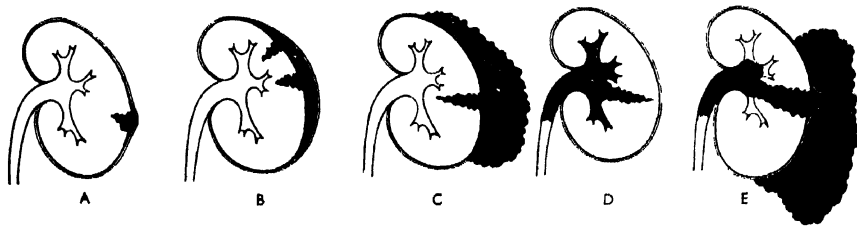


FIG. 1415.—Various types of renal injuries: A, small subcapsular hæmorrhage; B, large subcapsular hæmorrhage; C, cortical laceration with perinephric hæmatoma; D, medullary laceration with bleeding into the renal pelvis; E, complete rupture. (After P. Adams.)

appearance until some hours after the accident. If the hæmorrhage is profuse, it may be followed by clot colic.

Severe Delayed Hæmaturia.—Sudden profuse hæmaturia can occur between the third day and the third week after the accident in a patient who appears to be progressing favourably. It is due to a clot becoming dislodged.

Meteorism.—In many cases of renal injury, abdominal distension comes on about twenty-four to forty-eight hours after the accident. In all

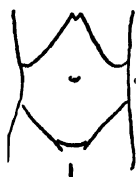


FIG. 1416.

probability it is caused by a retro-peritoneal hæmatoma implicating splanchnic nerves.

A *perinephric hæmatoma* should be suspected if there is even a slight flattening of the normal contour of the loin (fig. 1416).

MANAGEMENT AND TREATMENT

Conservative treatment is usually successful and must be instituted at once. However, it must be remembered that injury to other organs may be associated with injury to the kidney.

1. The patient must rest flat in bed until macroscopic hæmaturia has been absent for one week.
2. Morphine should be given for pain, and as a sedative.
3. Hourly pulse and blood-pressure chart must be kept.
4. Give a sulphonamide or antibiotic to prevent infection of the hæmatoma.
5. Save a sample of each specimen of urine passed.
6. Group and cross-match blood. A transfusion may be required for shock or continuing hæmorrhage.
7. An intravenous urogram (I.V.P.) should be obtained urgently to show that the other kidney is normal.

Under this regimen the hæmaturia should diminish, pain and guarding decrease, and pulse and colour of the patient improve.

Exploration is necessary in 20 to 25 per cent. and is undertaken if : 1. There are signs of progressive blood loss. 2. A swelling develops in the loin. 3. Signs of peri-renal infection develop. The aim is to conserve the kidney or part of it if possible.

Should the necessity for an urgent operation arise and facilities for pyelography not be available, the presence of a functioning kidney on the contralateral side can often be confirmed by chromo-cystoscopy (p. 1064). When the kidney is found to be ruptured in several places, or the kidney pedicle is damaged, nephrectomy must be undertaken. Small tears can be sutured over Oxyel or a piece of detached muscle, either of which promotes hæmostasis. Larger single rents in the middle of the kidney are best dealt with by performing nephrostomy through the rent and suturing the kidney on either side of the tube. If laceration is confined to one pole of the kidney, partial nephrectomy may be practicable.

When a sole existing kidney is sufficiently damaged to necessitate exploration, it must be repaired. Failing this, the wound is packed firmly with gauze in the hope that not only will the bleeding be controlled but that the ruptured kidney may heal.

In all cases the peritoneum should be opened to exclude damage to other organs.

When there are signs of intraperitoneal hæmorrhage or peritonitis, laparotomy should be performed. Repair of a lacerated kidney by the transperitoneal route should be followed by careful closure of the posterior parietal peritoneum and drainage of the kidney through the loin. Antibiotic therapy is commenced.

In the case of severe intraperitoneal rupture of the kidney, abdominal nephrectomy

is undertaken, provided the contralateral kidney is present, an easy matter to ascertain when the peritoneal cavity is open.

Multiple Injuries.—Simultaneous splenectomy and left nephrectomy have been carried out successfully when both organs have been ruptured. The mortality of cases of rupture of the kidney with damage to the liver or hollow organs, often complicated by fractured ribs, pelvis, or vertebræ, is high.

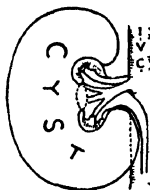


FIG. 1417.—A pararenal pseudo-hydronephrosis.

Complications.—1. Clot retention in the bladder can be cleared with a plastic catheter and a glass syringe.

2. Pararenal pseudo-hydronephrosis (fig. 1417) may occur in the course of a few weeks, due to a combination of a complete tear of the renal cortex and ureteric obstruction.

3. Hypertension may follow fibrosis of the kidney, usually after three months or more. Nephrectomy will be necessary.

4. **Aneurism of a renal artery** is a rare complication of injury to a kidney. The most frequent symptom is pain in the renal region, and, when the aneurism is large, a non-tender swelling will be felt. Only occasionally is it pulsatile, and a bruit may be heard. An X-ray may show a shadow in relation to the pelvis of the kidney. An I.V.P. demonstrates that the swelling is extrarenal with the renal pelvis displaced laterally. Aortography will confirm the diagnosis. Attacks of hæmaturia occur, and are probably due to congestion of the renal parenchyma. The aneurism may rupture into the peritoneal cavity or into the perirenal tissues, with fatal results.

Treatment by nephrectomy and excision of the aneurism has been undertaken successfully on a number of occasions. In a few cases a saccular aneurism has been resected with preservation of the renal vessels and the kidney.

INJURIES TO THE URETER

Rupture of a ureter can occur as the result of an accident causing hyperextension of the spine. The clinical diagnosis is impossible until a diminished quantity of urine is passed, followed by a swelling in the loin or iliac fossa. An I.V.P. shows a diffuse shadow below the kidney on the injured side.

Injury of one or both ureters during a pelvic operation is considerably more common than the foregoing and occurs most often during the course of a difficult hysterectomy. Preliminary catheterisation of the ureters prevents such accidents, for with catheters within them, the ureters can be felt and seen unmistakably. The accidents include division of the ureter, ligating it, crushing it in forceps or removing a portion of its wall.

Injury recognised at the Time of the Operation.—The uretero-vesical continuity should be restored by one of the methods described below, unless the patient's condition is poor, when ligation of the proximal end of the ureter is the best course. If the patient rallies within two days, temporary nephrostomy is carried out, and a reparative operation undertaken later.

Injury not recognised at the Time of the Operation:

(a) **Unilateral Injury.**—There are three possibilities:

1. **No Symptoms.**—If one ureter is ligated securely with unabsorbable material and the contralateral kidney and ureter are normal, in 68 per cent. of cases the kidney on the obstructed side undergoes silent atrophy. Thus the accident remains unsuspected unless the patient, some time later, undergoes a urological examination.

2. **Lumbar pain**→*hydronephrosis*→*pyonephrosis* occurs in less than a half of the remainder. Excretory pyelography reveals no excretion on the side of the lesion. To avoid infection of the developing hydronephrosis nephrostomy should be undertaken early. A reparative operation can be undertaken later.

3. **A urinary fistula develops** through the abdominal incision or, following pan-hysterectomy, through the vagina. Again, a temporary nephrostomy may be performed, and a reparative operation postponed until œdema and infection have abated after an interval of about six weeks.

(b) **Bilateral Injury.**—In cases of anuria following ligation of both ureters during hysterectomy an attempt is made to pass ureteric catheters, when both ureters will be found to be occluded. The wound should be explored and ligatures removed.

Repair of the Injured Ureter

1. If the ends are clean cut and no length is lost, each end should be split for 1.5 cm. and the edges united as a flat ribbon (fig. 1557). A soft latex T-tube is left across the gap for two weeks. This is to be preferred to end-to-end suture which frequently results in stricture formation.

2. In division low down, it may be possible to reimplant the ureter into the bladder.

3. *Boari's Operation*.—A flap of bladder is turned up to replace the lower ureter.

4. Occasionally, where conservation of all renal tissue is vital, replacement by a segment of ileum is necessary.

Nephrectomy may be the best course for example in the elderly patient with a malignant condition.

HYDRONEPHROSIS

A hydronephrosis is an aseptic dilatation of the whole or part of the kidney due to a partial or intermittent obstruction to the outflow of urine.

A hydronephrosis may be unilateral or bilateral.

Unilateral hydronephrosis is due to some form of *ureteral* obstruction, often at the pelvi-ureteric junction; any length of ureter above the obstruction is dilated also. The causes are:

Extramural.—1. In a large number of cases of pelvic hydronephrosis an artery, or a vein, at the lower pole obstructs the upper ureter. In many

instances this is not an aberrant vessel, but a normal inferior renal artery or vein that has been displaced downwards by the enlarged renal pelvis. This displaced vessel, while adding considerably to the rapid increase in the size of the hydronephrosis, is not the cause thereof (fig. 1418).

2. Involvement of ureter by growth outside it, e.g. carcinoma of the cervix, prostate, rectum, colon, or cæcum.

3. Idiopathic retroperitoneal fibrosis (p. 1099).

Intermural.—1. Congenital stenosis, physiological narrowing, or achalasia at the pelvi-ureteric junction.

2. Congenital atresia of the ureteric orifice (p. 1080).

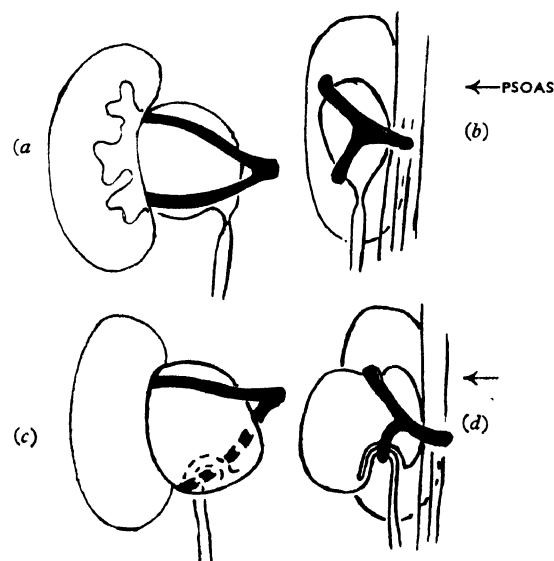


FIG. 1418.—'Congenital' hydronephrosis with obstruction at pelvi-ureteric junction. (a) Initial arrangement. (b) Lateral view of the hilum. (c) and (d) show pelvis of kidney prolapsing forward between branches of the renal artery.

3. Inflammatory stricture of the ureter following removal of a stone that was lodged in one portion of the ureter for a considerable time, or tuberculosis of the ureter. Stricture of the ureter also follows uretero-ureteric anastomosis or trauma to the ureter during a pelvic operation.

4. Neoplasm of a ureter, or the bladder involving a ureteric orifice.

Intramural.—A calculus in the ureter or small calculus in the renal pelvis. The latter often gives rise to intermittent hydronephrosis.

Bilateral hydronephrosis is generally the result of some form of *urethral* obstruction, but it can also be caused by one of the lesions described above occurring on both sides.

When due to lower urinary obstruction, the cause may be :

(a) *Congenital*:

1. Atresia of the urethral meatus or, very rarely, phimosis.
2. Congenital valves of the posterior urethra, or congenital contracture of the bladder neck.

(b) *Acquired*:

1. A neoplasm of the bladder which involves both ureteric orifices.
2. Prostatic enlargement or carcinoma ; acquired contracture of the bladder neck.
3. Carcinoma of the cervix (occasionally carcinoma of the rectum), involving both ureters.
4. Inflammatory or traumatic urethral stricture. Phimosis.

When the obstruction lies in the urethra, muscular hypertrophy of the bladder occurs, and the ureters, by reason of this hypertrophy, become partially obstructed in the intramural portion of their course.

Pathology.—The pathological changes differ in some respects according to whether the kidney has an extrarenal or an intrarenal pelvis (fig. 1419), the former

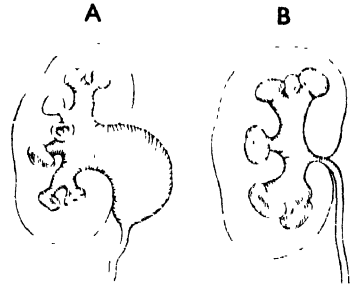


FIG. 1419. — Hydronephrosis occurring in an (A) Extrarenal, (B) Intrarenal pelvis of the kidney, the former being more liable to be obstructed by a blood-vessel.



FIG. 1420.—Renal type of hydronephrosis. Most of the renal parenchyma has been destroyed. Calculi are present in the lower pole.

being much more common. In a kidney with an extrarenal pelvis, at first the pelvis alone becomes dilated (pelvic hydronephrosis) (fig. 1418). As time goes on, if the obstruction is not relieved the calyces become increasingly dilated and the renal parenchyma is progressively destroyed by pressure atrophy. In a kidney with an intrarenal pelvis the destruction of the parenchyma (fig. 1420) occurs more rapidly. In either case, it may be a matter of years before the diagnosis is made, by which time the kidney may be merely a lobulated sac containing pale uriniferous fluid of low specific gravity. On bisecting the specimen longitudinally a series of compartments representing the dilated calyces will be found. Rarely is the renal parenchyma destroyed in its entirety, although in advanced cases the amount remaining is very small.

edge of the pelvic flap by a continuous suture of 0000 plain catgut (fig. 1424 C). A ureteric catheter is passed into the ureter for a short distance; this is merely a temporary expedient to keep the deep sutured layer out of the way while the superficial edges are drawn together (fig. 1424 D) in the same manner as were the deep. The catheter is withdrawn just before the anastomosis is completed.

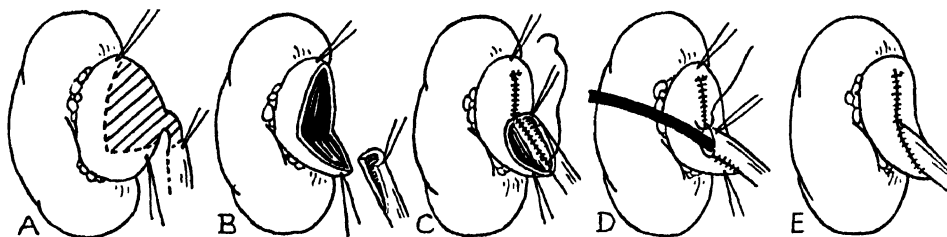


FIG. 1424.—The Anderson-Hynes plastic reconstruction of a hydronephrosis. Note the placement of the stay sutures.

Intubated Ureterostomy (Davis).—Longitudinal incision of a narrowed segment of ureter, leaving a T-tube across the split to allow regrowth round the tube is a simple and effective treatment when the pelvis is not greatly enlarged.

Renal pedicle sympathectomy (syn. denervation of the kidney) is often successful in relieving pain associated with small hydronephroses when no cause for the condition can be discovered. Function will improve and calyceal dilatation may be reduced. This is most likely in the young. The technique involves the mobilisation of the kidney and the careful stripping of all connective tissue and sympathetic fibres from the hilum of the kidney, the vessels, the renal pelvis, and upper 2.5 cm. of the ureter.

RENAL CALCULUS

Ætiology.—The subject is a complicated one, and the following is an epitome of current opinion. (Compare with the ætiology of gallstones, p. 825.)

1. **Dietetic.**—A deficiency of vitamin A causes a desquamation of epithelium. The cells form a nidus around which the stone is deposited. From a study of economic conditions in districts where urinary calculi are common, it is evident that the inhabitants suffer from dietetic imbalances.

2. **Altered Urinary Solutes and Colloids.**—In hot climates the concentration of solutes will rise. It has been postulated that any reduction of the urinary colloids which adsorb solutes, or excess of muco-proteins which may chelate¹ calcium, form an insoluble complex.

3. **Decreased Urinary Output of Citrate.**—The presence of citrate in the urine (normal 60 mg. per 100 ml.) tends to maintain in solution otherwise relatively insoluble calcium phosphate and carbonate. The excretion of citrate is under hormonal control and decreases during menstruation.

4. **Renal Infection.**—Infection favours the formation of urinary calculi. Both clinical and experimental stone formation is common when the urine is infected with a urea-splitting streptococcus, staphylococcus, or proteus. The predominant bacteria found in the nuclei of urinary calculi are a staphylococcus and *Esch. coli*.

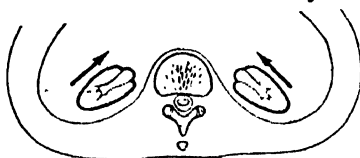


FIG. 1425.—In prolonged recumbency the fact that the urine has to travel uphill favours urinary stasis and consequent stone formation. (After A. C. McEachern.)

5. **Inadequate Urinary Drainage.**—Stones are prone to occur in patients with obstruction to the free passage of urine.

6. **Prolonged immobilisation** from any cause, e.g. paraplegia, is prone to result in skeletal decalcification and an increased output of calcium in the urine. This, combined with the mechanical effects of recumbency on renal drainage (fig. 1425), favours the deposition of calcium phosphate calculi. In uninfected cases spontaneous dissolution sometimes occurs.

¹ Chelate—to seize hold of. Chêṭē, Gr. = the prehensile claw of a crab or scorpion.

7. **Hyperparathyroidism**, although rare, occurring perhaps in 0·2 per cent. of cases, should always receive consideration. In cases of multiple or recurrent urinary calculi this cause should be eliminated by biochemical tests (p. 571). Hyperparathyroidism results in a great increase in the elimination of calcium in the urine. It has been aptly remarked that these patients pass their skeleton in their urine. A parathyroid adenoma should be removed before the urinary calculi are treated.

8. **Randall's Plaque. Microliths.**—Randall showed that the initial lesion in many cases of renal calculus is an erosion at the apex of one of the renal papillæ. On this erosion are deposited urinary salts = Randall's plaque. It has further been shown that minute concretions (microliths) occur normally in the renal parenchyma and Carr postulates that these particles are carried away, like carbon particles from the bronchi, by lymphatics. Should some of the renal lymphatic vessels become blocked by inflammatory exudate, a subendothelial calculus may form. Later the endothelium becomes ulcerated, leaving the calculus in contact with the urine.

Varieties.—Stones found in the kidney belong to one of the following types, a mixture of the first two being especially common.

Oxalate Calculus (Calcium Oxalate).—Popularly known as the mulberry stone, it is covered with sharp projections (fig. 1426). These cause the kidney to bleed, and altered blood is precipitated on the surface of the stone.

FIG. 1426.—Two oxalate calculi. The larger one removed from the right kidney and the smaller from the left of the same individual. The larger is black, owing to altered blood. The smaller is beginning to be discoloured around its sharp projections. These specimens illustrate clearly the process by which the oxalate calculus changes its complexion.



An oxalate calculus, which is usually single, casts an exceptionally good shadow radiologically, and this is fortunate, for often by virtue of its rough surface it gives rise to symptoms when comparatively small. A calcium oxalate stone is very hard, and on section is laminated concentrically. 'Envelope' crystals can be identified in the urine, viz.———



FIG. 1427.—Phosphatic calculus (actual size). Note its branched nature (staghorn calculus).

Phosphatic calculus (usually calcium phosphate, although sometimes combined with ammonium magnesium phosphate and, rarely, composed of the latter only) is smooth and dirty white. In an alkaline urine it enlarges rapidly, and often fills the renal calyces, taking on their shape (staghorn calculus, fig. 1427). Because it is smooth, a phosphatic calculus gives rise to few symptoms until it has attained a large size. By reason of its size rather than its density, it is demonstrated readily by X-rays.

Uric acid and urate calculi are hard and smooth and, because they are usually multiple, they are typically faceted. Their colour varies from yellow to reddish brown. Pure uric acid calculi are not opaque to X-rays,

but absolutely pure uric acid calculi are uncommon; the majority contain enough calcium oxalate crystals to render them opaque. Calculi of ammonium and sodium urate are sometimes found in children. Such stones are yellow, soft, and friable, and unless they contain impurities they do not cast an X-ray shadow.

Cystine calculi are wont to appear in the urinary tract of patients with cystinuria and are sometimes encountered in young girls. Cystinuria is *not* an inborn error of metabolism; it results from a greatly diminished or absent resorption of cystine from the renal tubules.



FIG. 1428.—Cystine crystals from the urine of a patient with cystinuria.

Cystine crystals are hexagonal, white, translucent, and appear only in acid urine (fig. 1428). Cystine calculi are usually multiple and may assume a cast of the renal pelvis and calyces (fig. 1429). These calculi are soft, like beeswax, and pink or yellow when first removed. On exposure the colour changes to a greenish hue. They are radio-opaque owing to the sulphur they contain.

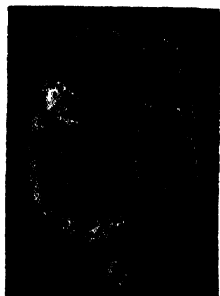


FIG. 1429.—Cystine calculus which has destroyed the renal parenchyma. (Dr. L. C. Hermite, Sheffield.)

Xanthine calculi are extremely rare. They are smooth and round, brick red in colour, and show a lamellar structure.

Indigo calculi are curiosities. Blue in colour, they are derived from indican.

Clinical Features.—Fifty per cent. of patients with renal calculus present between the ages of thirty and fifty. The male/female ratio is 4:3.

The symptoms are not stereotyped and sometimes the diagnosis remains obscure until a radiological examination has been made.

Quiescent Calculus.—Some stones, especially those composed mainly of phosphates, lie dormant for a long period, during which time there is progressive destruction of the renal parenchyma, and uræmia may be the first indication. Secondary infection usually supervenes.

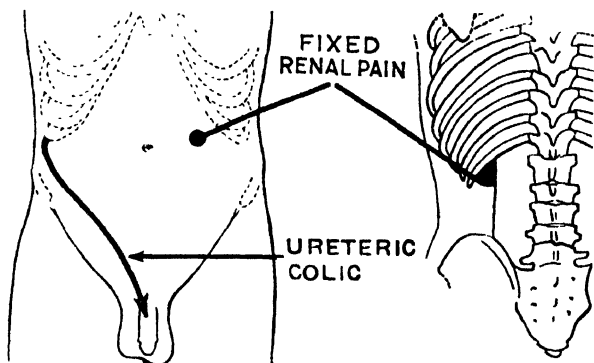


FIG. 1430.—The usual distribution of renal pain.

Pain is the leading symptom in 75 per cent. of cases.

Fixed renal pain is located in the renal angle posteriorly (fig. 1430), in the hypochondrium anteriorly, or in both situations simultaneously. It is often worse on movement, particularly on walking upstairs.

Ureteric¹ colic is an agonising pain passing from the

¹ Pain passing from loin to groin should be called ureteric colic, *not* renal colic.

loin to the groin, coming on suddenly, causing the patient to draw up his knees and roll about. It is often accompanied by vomiting, profuse sweating, and strangury¹. The pulse quickens, and as the attack progresses the temperature becomes sub-normal. Bouts of colic often recur for several hours, and occasionally continue for more than twenty-four hours. The condition is often due to a stone entering the ureter, but also occurs when a stone in the renal pelvis temporarily blocks the pelvi-ureteric junction. Colic may also be caused by the passage of a shower of oxalate crystals, e.g. after eating an excess of strawberries or rhubarb.

Abdominal Examination.—During an attack of ureteric colic there is rigidity of the lateral abdominal muscles but not, as a rule, of the rectus abdominis. After the attack has passed off, abdominal examination is often negative. In patients with a dull, fixed pain usually there is tenderness over the affected kidney, especially if it is infected. Only when a calculus-containing kidney becomes the seat of a hydronephrosis or pyonephrosis of considerable size does it give rise to a palpable swelling.

Hæmaturia.—Infrequently, profuse hæmaturia is a leading, or the only symptom. As a rule hæmaturia occurs in small amounts (enough to render the urine 'smoky') during or after an attack of pain.

Pyuria.—Infection of the kidney is liable to supervene, and pus will be found in the urine in varying amounts. When a large phosphate calculus is present, the passage of turbid urine, which on examination is found to contain pus cells, is sometimes the leading feature.

INVESTIGATION OF A SUSPECTED CASE OF RENAL CALCULUS

Radiography.—Before the examination the bowels should be emptied by giving a *vegetable* laxative, for minerals in the intestine cast a shadow (fig.



FIG. 1431.—The two shadows in the region of the right kidney were mistaken for calculi. They were due to enteric-coated pills of ammonium chloride. (After H. L. Kretschmer.)

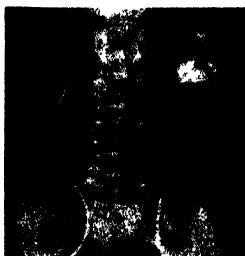


FIG. 1432.—Thought to be gall-stones, proved by ureteric catheter, etc., to be renal calculi.

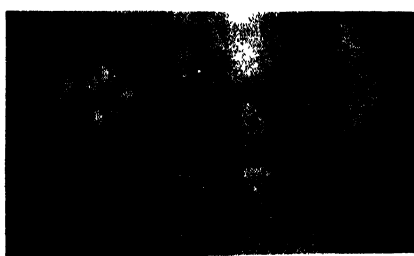


FIG. 1433.—Plain radiograph showing horse-shoe kidney full of stones. The patient, a man of forty-two, stated that he had enjoyed good health until one week previously.

1431). Radiographs of both kidneys, ureters, and the bladder are taken. When a renal calculus is branched, there is no doubt concerning the shadow it casts. If a shadow *may* be a calculus in the kidney, exposures are made

¹ Strangury is the passage of a few drops of urine, often blood-stained, after painful straining.

during full inspiration and full expiration. If the opacity moves with the kidney, and measurements from the lower pole of the kidney to the opacity remain constant, then the shadow is intrarenal.

The following structures and substances from time to time cast a shadow which at first sight may appear to be a renal calculus :

- (i) A calcified lumbar or mesenteric lymph node.
- (ii) A gall-stone or a concretion in the appendix.
- (iii) Drugs (e.g. fersolate) or foreign bodies in the alimentary canal.
- (iv) Phleboliths.
- (v) The ossified tip of the twelfth rib.
- (vi) A chip fracture of the transverse process of a lumbar vertebra.
- (vii) Calcified tuberculous lesion of the kidney.
- (viii) A calcified suprarenal gland.

A doubtful shadow in relation to the kidney can often be proved to be extrarenal by a lateral radiograph. When the shadow can be shown to lie anterior to the bodies of the vertebræ, *ipso facto* it is anterior to the kidney. Such is the finding in calcified mesenteric lymph nodes, gall-stones, and opacities in the alimentary tract.

Excretory pyelography is of value in confirming that the opacity is intrarenal, in determining in which part of the kidney the stone is situated (fig. 1434), and in revealing the function of the other kidney.

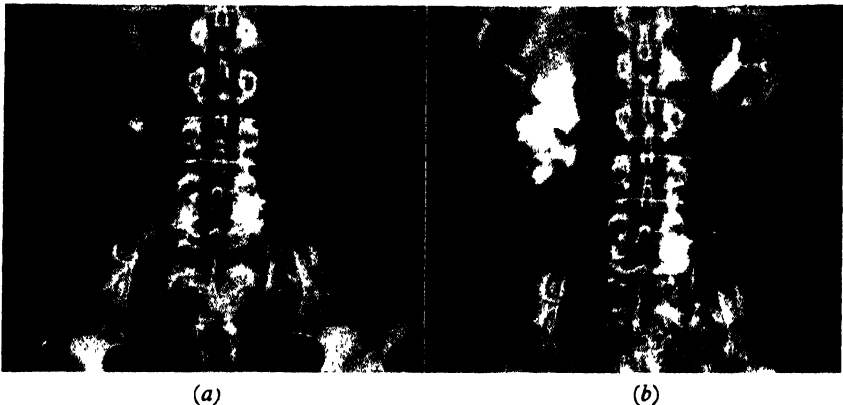


FIG. 1434.—(a) Plain X-ray showing a renal calculus. In which part of the kidney is it situated? (b) Same case. Excretory pyelogram. The stone is in the pelvi-ureteric junction. Pyelolithotomy is indicated.

The presence of a non-opaque calculus also can be demonstrated by pyelography, for it causes a filling defect in the shadow cast by the medium. A similar defect is seen in papilloma of the renal pelvis (p. 1118). Retrograde pyelography may be required to give a clearer delineation.

Cystoscopy.—A urethral stricture or prostatic obstruction will be revealed by instrumental examination. Unless cystitis is present, the bladder wall looks normal. The urinary efflux from the affected side is increased if the stone is irritating the renal pelvis, or decreased when the pelvi-ureteric junction is blocked or when the parenchyma is severely diseased.

Treatment of Ureteric Colic (see p. 1097).

OPERATIONS FOR THE REMOVAL OF RENAL CALCULI

Pre-operative Treatment.—If urinary infection is present, antibiotic treatment is instituted, and continued after operation as necessary. A bacteriology swab for culture should be taken of urine in the renal pelvis.

Operation—General Remarks.—All the operations about to be described have certain features in common. Via a lumbar incision (p. 1119) the kidney is exposed and delivered on to the surface. In the small number of cases when delivery is impossible, the choice of procedure is more limited and the operation more difficult. An X-ray showing the calculus or calculi clearly should be displayed in an illuminated viewing-box in the operating theatre. The stone or stones removed should coincide in every respect with the X-ray findings. Failure to account for some part of the shadow calls for a further search.

Often it is advisable to X-ray the exposed kidney (using a special film encased in a sterile plastic cover) on the operating table. This ensures that no stone or portion of stone within the kidney has been overlooked, and reduces the number of so-called recurrences.

At the conclusion of all the operations to be described the kidney is replaced in its bed, and the lumbar wound closed with drainage of the perinephric space. If nephrostomy has been performed, the tube is either brought through the lumbar incision or through a stab incision.

Pyelolithotomy is indicated when a stone (it is usually solitary) lies in the renal pelvis, provided the pelvis is extrarenal. In nearly 50 per cent. of cases a renal calculus is so situated. The posterior wall of the renal pelvis is dissected free from its surrounding fat. The kidney is grasped in the left hand so that the tips of the index and middle fingers lie beneath the renal pelvis, making it more prominent, while the thumb anteriorly prevents the stone slipping into one of the calyces. An incision is made on to the stone in the long axis of the renal pelvis. The stone is removed with gall-stone or special forceps, care being taken to avoid breaking it. If pyelography showed a stone in a calyx and the renal pelvis is large, the stone can be located by the little finger introduced into the renal pelvis: should the stone be accessible, it can be grasped in forceps and removed by this route. After a stone has been removed via the renal pelvis, a bougie is passed through the pelvi-ureteric junction in order to dilate a possible stricture, and at the same time any adhesions present in this neighbourhood are freed, or an obstructing vein is divided between ligatures. If the kidney is known to be uninfected, the incision in the renal pelvis is closed by interrupted sutures of fine plain catgut. If the kidney is grossly infected, nephrostomy (p. 1073) is performed before closing the incision in the renal pelvis.

Nephrolithotomy is indicated (1) when a calculus or calculi lie within an intra-renal pelvis, or the pelvis cannot be displayed because the pedicle is short, or because of adhesions due to a previous operation; (2) when the calculus can be palpated through the cortex; (3) when there is a branched calculus, and the state of the opposite kidney precludes nephrectomy.

An incision into the renal parenchyma is accompanied by considerable hæmorrhage, which is lessened by digital compression of the renal vessels maintained by an assistant. Also with a view to reducing hæmorrhage, the renal parenchyma is incised with a diathermy knife just behind and parallel to the most prominent part of the convex border of the kidney (Brödel's line). As the terminal branches of the anterior and posterior renal arteries meet along this line, no large artery is divided, thereby

The first operation of nephrolithotomy was performed by Ambroise Paré (1509–1590). His patient was a criminal condemned to death by hanging, instead of which Paré removed two stones from his kidney. Fifteen days later the patient was cured, secured his remission, and was given a grant of money.

Max Brödel, 1870–1941. *Artist of Pathology and Founder and Director of the Department of Art as Applied to Medicine (the first school of its kind in the world), Johns Hopkins University, Baltimore, U.S.A.*

minimising the amount of subsequent cortical necrosis. The incision, usually about 1 inch (2.5 cm.) long, is made over that calyx containing the stone or stones, which are removed with lithotomy forceps. If the kidney is uninfected, the renal incision can be closed by interrupted catgut sutures passing through the kidney substance but not penetrating the calyx, and tied over a piece of oxycel or muscle to enhance hæmostasis. If infection is present, nephrostomy is carried out by placing a small self-retaining catheter through the incision into the renal pelvis. The incision is then closed on either side of the tube in the manner just described.

Pyelonephrolithotomy is used to remove a stone situated deeply in a calyx, especially if it is present in addition to one in the renal pelvis, and in cases of stones situated in more than one calyx. The pelvis is opened, the little finger is introduced and the calyx containing the stone is palpated. If pressure is exerted on the corresponding convex border of the kidney, even a small stone can usually be palpated. Otherwise, the kidney is punctured with a round-bodied needle so that the point grates on the stone. An incision is made through the parenchyma, and with the finger pressing on the calculus from within, it is grasped by lithotomy forceps introduced through the cortical incision, and withdrawn. If there are stones in other calyces the procedure may be repeated, making two, or even three, cortical incisions. The incisions are closed in the manner already described. If drainage of the interior of the kidney is necessary, the catheter is introduced through the cortical incision or, if there are more than one, through the most dependent.

Partial Nephrectomy.—When the stone is in the lowermost calyx—a fairly common position—and there is considerable damage to the calyx, as in multiple stones, or ‘grit’ in the renal substance, then lower partial nephrectomy is the best operation (p. 1120). If the damaged calyx is not removed, its dependent position encourages further stone formation.

Nephrectomy (p. 1119) is indicated when the contralateral organ has been proved to be perfectly healthy and the stone or stones have destroyed much of the renal parenchyma. Large branched calculi are notorious for recurring, so nephrectomy is often the best treatment in such cases.

Post-operative Treatment.—In non-infected cases a prophylactic course of Sulphatriad is given for a week, together with alkalis and a high fluid intake. If a nephrostomy tube has been inserted, it is managed in the same way as that described on p. 1073. In infected cases the interior of the kidney is irrigated with saline solution through the nephrostomy tube frequently, until the bleeding has ceased, and thereafter two or three times a day with solution G (see below). The tube is retained for at least fourteen days. These measures, by dissolving phosphatic debris, greatly reduce the incidence of recurrence.

Hæmorrhage sometimes occurs, usually about the fourth day after an operation which necessitates incising the renal parenchyma. Blood transfusion and sedatives are usually sufficient to tide the patient over this complication. Should hæmorrhage continue, however, the lumbar wound must be reopened. In cases where the contralateral organ is healthy, nephrectomy is the best method to adopt. In other circumstances, placing a strip of Oxycel in contact with the cut renal parenchyma of the reopened kidney, and resuture, together with nephrostomy, may prove successful.

Treatment of Renal Calculi by Dissolution.—Most renal calculi, because they are irreversible colloidal compounds, cannot be dissolved. The possible exceptions are (a) non-infected recumbency calculi of calcium phosphate undergo dissolution with a high fluid intake; (b) small cystine stones dissolve in an alkaline urine. Sodium citrate mixture (p. 1102) is taken three or four times a day to maintain a urinary pH of between 7.6 and 8.0; (c) small phosphate calculi can be dissolved by bringing them in direct contact with solution G:

Solution G :	(Monohydrous citric acid	32.3 G.
	Anhydrous magnesium oxide	3.8 G.
	Anhydrous sodium carbonate	4.4 G.
	Distilled water	ad 1,000 ml.

Treatment of Bilateral Renal Calculi.—Usually the kidney with the better function is operated upon first, the operation on the contralateral side being postponed for two or three months. Exceptions to this rule are (a) if there is pain on one side, that side is operated upon first, for pain usually

signifies that the stone is obstructing the outflow of urine from the kidney, the function of which will become increasingly impaired; (b) if there is a pyonephrosis on one side this should be treated by nephrectomy, or, if the patient's condition is poor, by nephrostomy. In cases of bilateral staghorn calculi without infection, especially in the elderly, it is probably wiser not to operate. The patient should drink large amounts of fluid.

PREVENTION OF RECURRENCE

Frère Jacques, that famous lithotomist of the Middle Ages, used to say, "I have removed the stone, but God will cure the patient." With the advance of knowledge our responsibilities do not end with the removal of the stone; provision must be made to prevent recurrence. Recurrent calculi can be divided into two varieties:

(a) *False Recurrence*.—Even a tiny fragment overlooked at the time of the operation is liable to become the nucleus of a new stone.

(b) *True Recurrence*.—The stone should be analysed so that by a proper diet and vitamin intake the patient may render his urine unfavourable to the formation of a similar stone. There should be a high fluid intake in all cases, regardless of the chemistry of the stone. Hyperparathyroidism must be excluded.

Uric-acid and Urate Calculi.—Those meats, offal, and fish which are rich in purine should be avoided. Sufficient sodium citrate and sodium bicarbonate are given to keep the urine slightly alkaline.

Calcium Oxalate Calculi.—Rhubarb, strawberries, plums, spinach, and asparagus, which are rich in oxalate, should only be taken if they are accompanied by cream or milk, as then oxalates are precipitated as an insoluble calcium salt in the intestine, and are not absorbed. At all times a diet adequate in calcium and magnesium is necessary; calcium oxalate is rendered less soluble in the presence of magnesium ions.

Phosphate Calculi.—Excessive alkalinity of the urine should be treated by giving ammonium chloride or another urinary acidifier. Phosphorus in the diet must be restricted to a maximum of 1,400 mg. per diem. Aluminium gel, 40 ml. t.d.s., p.c. and at bedtime, drastically reduces the incidence of recurrence.

Cystine Calculi.—Sulphur-containing proteins such as eggs, meat, or fish are restricted, and proteins with a low sulphur content substituted. Carbohydrates and fats are unrestricted. The urine must be kept alkaline permanently, and to this end sodium citrate and sodium bicarbonate are given in sufficient quantities, the patient testing the reaction of his urine night and morning.

Recumbency Calculi.—In patients doomed to long recumbency, sodium phytate taken by mouth forms insoluble calcium phytate, thus removing by the bowel one source of hypercalciuria.

Infection.—In all types of calculi accompanied by infection, no effort should be spared to render the urine sterile and keep it sterile.

Obstruction.—If there is any factor that causes urinary stasis, be it a ureteric stricture, an enlarged prostate, or a urethral stricture, this should be remedied.

URETERIC CALCULUS

A stone in the ureter nearly always has its birth in the kidney. When it enters the ureter the calculus is rounded or oval in shape. Should it become arrested in its descent to the bladder, in time it becomes enlarged and elongated, resembling in shape a date stone (fig. 1435). In 90 per cent. of cases the stone is single.

Clinical Features.—When a stone descends into the ureter there is an attack of ureteric colic, which is repeated at longer or shorter intervals, until the stone is ejected into the bladder or becomes impacted in the ureter.



FIG. 1435.—Ureteric calculus removed by operation.

There are five sites of anatomical narrowing of the ureter where a stone may be arrested (fig. 1436).

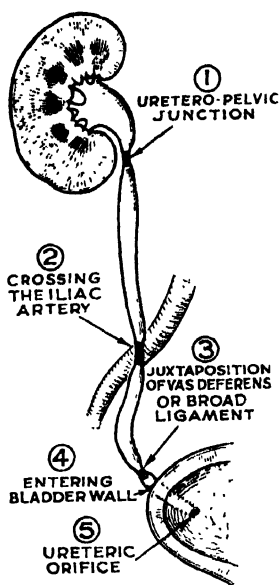


FIG. 1436.—Normal anatomical narrowings of the ureter. (After C. C. Higgins.)

is referred to the tip of the penis in the male. In both sexes there is strangury. In more than 50 per cent. of cases the stone is passed spontaneously.

Impaction.—When the stone becomes impacted the attacks of colic pass off and give place to a dull pain, usually in the iliac fossa, for impaction usually occurs in the pelvic portion of the ureter. The pain is increased by exercise, and relieved by rest. Such pain varies in intensity and is often associated with backache due to distension of the renal pelvis. Cessation of pain sometimes occurs by the stone forming for itself a false diverticulum in the wall of the ureter by a process of pressure necrosis; subsequent perforation of the ureter occurs but rarely. Severe renal pain persisting for one to two days and then gradually subsiding suggests that complete obstruction has occurred. If an I.V.P. performed two weeks later shows no function, the stone must be removed, otherwise complete atrophy of the kidney will occur.

Hæmaturia.—Some degree of hæmaturia occurs after an attack of ureteric colic, and it lasts for a few hours or a day. It is sometimes so slight as to require microscopic identification.

Abdominal Examination.—There is tenderness and often rigidity in some part of the course of the ureter. On rare occasions a stone in the lower end of the ureter can be felt on rectal or vaginal examination. The principal difficulty on the right side is distinguishing the symptoms and signs produced by a ureteric calculus from those of acute appendicitis. The presence of blood in the urine does not necessarily rule out appendicitis, for an inflamed

Ureteric Colic.—A stone in the upper third of the ureter produces symptoms identical with those of a stone blocking the pelvi-ureteric junction (p. 1090). When a calculus enters the lower third of the ureter, the colic it produces often commences anteriorly at a lower level than the kidney, and is frequently accompanied by pain referred along the two branches of the genito-femoral nerve, to the testis in the male (fig. 1437), the labium majus in the female, and to the anterior surface of the thigh in both sexes. The testis usually becomes retracted by spasm of the cremaster, and tender, the tenderness persisting for some time after the colic has ceased.

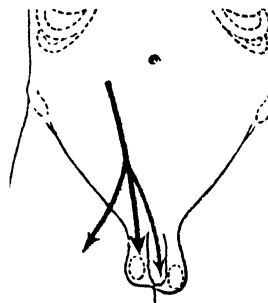


FIG. 1437.—Radiations of ureteric colic produced by a stone in the lower third of the ureter.

appendix lying in juxtaposition to the ureter can give rise to ureteritis which may cause a slight increase of red cells in the urine.

Radiography.—Occasionally a typical elongated shadow in the line of the ureter renders the diagnosis undoubted ; more often either no shadow is seen, owing to the small size of the stone and its obscuration by intestinal gas shadows, or a shadow is seen which may or may not be a ureteric calculus.

Excretory Pyelography.—In, and for some time after, an attack of renal colic there is either no excretion or greatly delayed excretion on the affected side. Films taken later, even up to forty-eight hours after the intravenous injection of Hypaque (diatrizoate) may outline the ureter down to the site of obstruction (fig. 1438). A stone not visible in the plain X-ray may become outlined in the ureter by the contrast solution. After a stone has been impacted for a week or more, a varying degree of hydronephrosis and dilatation of the ureter is often apparent.



FIG. 1438.—Excretory pyelograph showing stone in the line of the ureter.

If the pyelograms show normal excretion on the affected side, then the patient has not got renal colic.

Cystoscopy.—When a calculus is in the upper part of the ureter, the ureteric orifice shows no abnormality. When a calculus has reached the lower third of the ureter, or sometimes when it is in the middle third, the ureteric orifice becomes patulous and its immediate vicinity bespattered with minute petechial hæmorrhages. As the calculus descends to just above the bladder wall, these hæmorrhagic spots coalesce to form larger, bright-red extravasations. The stone having entered the intramural portion of the ureter, the ureteric orifice becomes grossly œdematous. Finally, the pouting calculus may be seen (fig. 1396).

The passage of a ureteric catheter provides much information, and in conjunction with radiography confirms the diagnosis. Usually a catheter fails to pass the obstruction and a radiograph shows the tip of the catheter abutting on the lower margin of the calculus. However, when the urinary efflux is increased, a catheter sometimes passes into the renal pelvis without hindrance, or, if arrested, after partial withdrawal and reinsertion (fig. 1439).



FIG. 1439.—Stone in the ureter. Catheter passed alongside the stone.

Retrograde pyelography is seldom required. The only indications for its employment are when there is doubt concerning a shadow being a ureteric calculus and excretory pyelography shows absence or ineffective delineation of the corresponding renal pelvis and the ureter. It sometimes proves a means of displaying the presence of a ureteric calculus non-opaque to X-rays.

Treatment. **1. Expectant.** Provided the urine is sterile, most small calculi will pass naturally. This may take many months. Large quantities of fluid should be drunk, and short courses

of anti-spasmodic drugs may help. Progress of the stone is observed by plain X-rays every six to eight weeks, and occasional pyelography to see that the function and appearance of the kidneys are satisfactory.

2. Intervention may be required if:

- (a) Repeated attacks of colic produce no advance of the stone.
- (b) The stone is enlarging but not moving.
- (c) Complete obstruction of the kidney occurs.
- (d) The urine is infected.
- (e) The stone when first seen is thought to be too large to pass.

Instrumental Treatment: Ureteric Catheterisation

Before and after instrumentation, a soluble sulphonamide with an alkaline mixture should be given.

A small stone half a centimetre or less in diameter arrested in any part of the ureter often passes after ureteric catheterisation. The catheter may be left in position for twenty-four to forty-eight hours, and 1 ml. of sterile liquid paraffin injected up the catheter before it is removed. This often results in the stone being passed within a few hours or days. Five per cent. procaine injected into the ureter at the time of passing the catheter, and at intervals, often controls pain and overcomes spasm. In the presence of severe renal infection, if the catheter can be manipulated beyond the stone, the drainage afforded often prevents the development of a pyonephrosis.

Operative Treatment

Uretero-lithotomy.—The patient should be submitted to radiography just prior to the operation, in order to detect any change in the position of the calculus.

(a) **Stone Impacted in the Upper Third of the Ureter.**—An incision similar to that for exposing the kidney (p. 1119) is made; it can be prolonged downwards if necessary.

(b) **Stone Impacted in the Lower Two-thirds of the Ureter, but above the Spine of the Ischium.**—With the patient in the dorsal position, an incision is made from a point $1\frac{1}{2}$ inches (3.75 cm.) above the anterior superior iliac spine to the middle of the inguinal ligament. The flank muscles are divided in the direction of their fibres. The peritoneum is displaced medially by gauze dissection until the dilated ureter is found adhering to its under-surface. The stone is sought by palpation, and, if possible, is milked upwards or downwards to a convenient point for extraction. The ureter is incised longitudinally and the stone withdrawn by a scoop or forceps. If the stone is impacted firmly, the only course is to incise the ureter directly over it, a practice to be avoided whenever possible, for the mucous lining is ulcerated at the site of impaction, and healing is impaired. The incision in the ureter is closed by sutures of 0000 plain catgut passing through the muscular coats only. The lower end of the wound is drained down to the incision in the ureter.

(c) **Stone Impacted in the Juxtavesical or Intramural Portions of the Ureter.**—A midline, sub-umbilical extraperitoneal approach is employed, with the patient in Trendelenburg's position. The peritoneum is stripped from the dome of the bladder and from the side wall of the pelvis. A dissection is then made between the posterior surface of the bladder and the wall of the pelvis. Often at this stage the stone will be felt. If this is not possible, the ureter must be identified where it crosses the iliac vessels, and traced downwards. Once the stone has been located it is removed as described above.

After removal of a stone at any site, a ureteric catheter or bougie should be passed to prove the patency of the distal ureter.

Ureteric Meatotomy.—The ureteric orifice is the narrowest part of the whole ureter. By enlarging the opening a stone is often enabled to pass (fig. 1440). However, this procedure almost invariably leads to urinary reflux afterwards and should therefore be avoided.

Dormia Basket (fig. 1441).—This is a stone dislodger and can be used for stones in the lower third of the ureter.

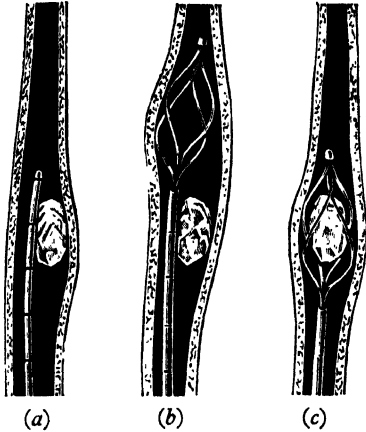


FIG. 1441.—Dormia stone catching basket in use. (a) basket introduced past stone. (b) opened and (c) enclosing stone ready for withdrawal. (Porgès, Paris).

Idiopathic retro-peritoneal fibrosis is rare. The patient, who typically has complained of backache for many months, suddenly becomes anuric. In other circumstances when kidney disease has been suspected pyelography reveals rapidly developing unilateral or bilateral hydronephrosis associated with a rising blood urea. Characteristically the ureters are nearer the midline than normally and this is due to an area of fibrosis centred over the bifurcation of the aorta which strangulates the ureters. The cause is largely unknown, but some cases have been noted following the use of methysergide for the treatment of migraine.

Treatment.—In the anuric patient, if an attempt at ureteric catheterisation is successful, the catheters should be left *in situ* to relieve the obstruction. If this fails, bilateral nephrostomy

is necessary although the use of the artificial kidney (p. 1071) may be beneficial.

In non-urgent cases the involved ureter or ureters should be freed from the fibrous mass by sharp dissection and fixed laterally to the psoas muscles to prevent recurrence. Should this prove impossible an ileal loop ureter should be constructed (p. 1159).



FIG. 1440.—Ogier Ward's electro-surgical ureteric meatotomy. Meatotomy in progress.

INFECTIONS OF THE KIDNEY

Ætiology.—Renal infections arise in the following ways :

1. *Hæmatogenous.*—From infected tonsils, carious teeth, or from cutaneous infections, particularly boils or a carbuncle.

2. *Ascending the peri-ureteral lymphatics* from the cervix or the prostate.

3. *Ascending the lumen of the ureter* from the bladder in long-standing cases of lower urinary obstruction with dilated ureters, when, as a result of vesico-ureteral reflux, infected urine may be carried from the bladder to the renal pelvis. Motile organisms, e.g. *Esch. coli*, ascend along the dilated ureters.

On complete urinary investigation, which should always be undertaken after the acute phase of the infection has subsided, about 30 per cent. of males and a smaller number of females will be found to have an obstructive lesion in some part of the urinary tract. Moreover, with or without such a lesion, a focus of infection in the genital organs may be demonstrable.

Bacteriology.—By far the most common organisms responsible are those of the coliform¹ group. When *Streptococcus faecalis* is present, in nearly 40 per cent. of cases it occurs in association with another organism.

¹ Coliform organisms comprise *Esch. coli*, *paracolon bacilli*, and *Aerobacter aerogenes*.

—Dormia, *Contemporary*. Assistant Professor of Urology, University of Milan, Italy.
Hans Christian Joachim Gram, 1863–1938. Professor of Medicine, Copenhagen.

In the presence of the common *Esch. coli* infection the urine is acid, as it also is in many types of streptococcal infection. Both staphylococci and *Proteus vulgaris* split urea, releasing ammonia, which causes the urine to become alkaline.

PYELONEPHRITIS

Renal infection, by whatever route it originates, is never confined to the renal pelvis. In hæmatogenous infection the renal parenchyma is attacked first, and the infection spreads to the renal pelvis. In ascending infection the bacterial inflammation is not limited to the renal pelvis; it implicates the calyces, the pyramids, and at least some part of the adjacent parenchyma. It is, therefore, more correct to discard the term 'pyelitis' in connection with the latter type of infection, in favour of the more accurate designation 'pyelonephritis'.

ACUTE PYELONEPHRITIS

Acute pyelonephritis is more common in females, especially during childhood, at puberty, soon after marriage ('honeymoon pyelitis'), during pregnancy, and at the menopause. It occurs more often on the right side than on the left. Rarely it is bilateral.

Clinical Features.—While there may be prodromal symptoms of headache, lassitude, and nausea, the onset is usually sudden, often commencing with a rigor and, even, vomiting. There is acute pain in the flank and the hypochondrium. In a few cases the pain radiates from the loin to the groin (ureteric colic). The temperature rises to 102° or 103° F. (38·8° or 39·5° C.), and is remitting. Soon after the onset, increased frequency of micturition, due to cystitis, sets in, and when, as is often the case, the urine is acid, micturition is accompanied by a scalding pain in the urethra. On examination there is tenderness in the hypochondrium and in the angle between the last rib and the sacrospinalis, accompanied by a varying degree of muscular rigidity. In the early stages, when it is imperative to make a correct diagnosis, the urine is clear macroscopically.

In cases of severe bilateral pyelonephritis, particularly when there is an associated obstruction, there are likely to be symptoms of uræmia.

Bacteriological Examination of the Urine.—A mid-stream specimen should be collected in a sterile bottle; the urine is centrifuged and examined microscopically. In early acute pyelonephritis there are typically a few pus cells and many bacteria. When pyelonephritis has been present for twenty-four hours or more, the urine is often cloudy and pus cells abound. Quantitative estimations of the cells and bacteria in the specimen are of real value, whereas vague impressions reported as . . . 'many, some, a few' are usually misleading. In all cases the investigation includes an examination of the sediment stained by Gram's method, culture of the specimen, and an investigation of the sensitivity of the organism or organisms to various antibiotics and chemotherapeutic agents.

Severe Cases.—There are repeated rigors and the temperature rises to 104° or 105° F. (40° or 40·5° C.), often without a corresponding rise in the pulse-rate. There is vomiting, sweating, and great thirst, and the patient looks and feels ill. The blood culture, if the specimen is taken soon after a rigor, often gives a positive result. After some hours the pain is localised in one or, rarely, both loins. Urine is scanty and highly concentrated, and is frequently teeming with coliform organisms and pus cells, in which case

the diagnosis is simplified. In bloodborne infections the organisms and pus cells do not appear in the urine until the infection has spread from the cortex to the medulla—a matter of many hours, or even days. As the abdominal rigidity abates, the enlarged affected kidney may be palpable.

Differential Diagnosis.—When the symptoms and signs are typical the diagnosis is straightforward. In other circumstances there may be difficulty in distinguishing the condition from pneumonia, acute appendicitis, and acute cholecystitis. The urgent need is to differentiate acute appendicitis from right-sided pyelonephritis. The fact that the pain commenced in, and did not pass to the right side, greatly favours the latter condition. Excretory pyelography may prove of assistance, for in early acute pyelonephritis limited to the right kidney the concentration of medium in the renal pelvis and calyces on the affected side is often so poor that no shadow, or a very indefinite shadow, is cast.

Pyelonephritis of Pregnancy.—The condition usually develops between the fourth and sixth months of gestation. It is commoner in women who have had recurrent bouts of cystitis or pyelonephritis in the past. In 90 per cent. of cases the right side alone is affected. The initiating causes are often chronic urethritis or a cervical erosion. The symptoms do not differ from those of pyelonephritis occurring in the non-pregnant. In about 10 per cent. of cases the disease runs a more severe and protracted course, and occasionally it results in abortion or premature birth. In all patients who have had pyelonephritis of pregnancy, periodic examinations of the urine are necessary during the rest of the pregnancy, the puerperium and for several months afterwards, for if the infection has not been eradicated, recrudescence is liable to occur during this period.

Pyelonephritis of infants and children is most common under the age of two years, and is more common in females. Often the infection commences acutely with rigors, the temperature rising to 103° or 104° F. (39·5° or 40° C.) remittently. In infants, more often than not, there are no symptoms referable to the urinary tract but only pyrexia, vomiting, anorexia, and loss of weight. Occasionally attacks of screaming due to colic occur. Slight terminal hæmaturia is sometimes present. In some cases a clue to the origin of the infection is apparent; in females vulvitis extending around the external urinary meatus; in circumcised males atresia with or without meatitis; in the uncircumcised, balanitis. Older children may complain of lumbar pain. In chronic cases increased frequency of micturition and slight intermittent pyrexia are the usual symptoms, but an examination of the urine to account for otherwise unexplained pyrexia may be the means of directing attention to the urinary organs. Seven out of ten boys with pyelonephritis have a congenital deformity of the urinary tract, usually producing some degree of obstruction. In girls, reflux of urine from bladder to kidney is nearly always present.

ACUTE PYELONEPHRITIS ASSOCIATED WITH RETENTION OF BLADDER URINE

Acute Pyelonephritis with retention of bladder urine occurs most frequently in men suffering from prostatic obstruction of stricture or the urethra, and in cases of fracture of the spine with injury to the spinal cord (p. 408). The retention is not necessarily complete and the patient may be able to urinate, but there is a varying amount of residual urine, from a few ounces to several pints. In most cases the infection is bilateral. Often it follows an ascending infection following the passage of a catheter or other instrument, or operations on the lower urinary tract, but it may arise spontaneously. The old name for this condition was 'surgical kidneys', and in the days of unsterile catheterisation it was frequent and dreaded. At post-mortem examination the kidneys are enlarged and miliary abscesses can be seen beneath the capsules. On bisection there are numerous yellow streaks of pus in the parenchyma radiating from the medulla to the convex border. The renal calyces, pelves, and ureters are dilated.

TREATMENT OF ACUTE PYELONEPHRITIS

The essential factors in curing pyelonephritis are *early, correct and prolonged* treatment, and careful follow-up with repeat cultures and white cell counts of the urine after treatment has finished. A full investigation to exclude abnormalities in the urinary tract should be undertaken when the acute attack is controlled.

A mid-stream specimen of urine should be taken and sent for culture and tests of the sensitivity of the organisms to antibiotics. Bed rest is important.

If the urine is acid, as it is in the common coliform infections, alkalinisation of the urine has a most beneficial effect in relieving the symptoms and in inhibiting the growth of these organisms. A mixture containing sodium citrate and sodium bicarbonate is efficacious. Tinct. hyoscyamus, minims 20 (1·2 ml.), can be added to the mixture with advantage to diminish pain.

To alkalinise the urine rapidly an intravenous injection of 10 ml. each of an isotonic solution of sodium lactate and a saturated solution of sodium bicarbonate (ampoules can be obtained from Crookes Laboratories) is very effective in ultra-acute cases.

When pain is severe, an antispasmodic is given and heat is applied to the affected loin. Morphia, in addition, may be necessary sometimes.

The patient should be instructed to imbibe large quantities of bland fluid, at least 5 pints (3 litres) in the twenty-four hours. In severe cases with vomiting or dehydration, intravenous dextrose-saline is given until the dehydration has been rectified and the vomiting has ceased.

While awaiting the bacteriological report and the result of the sensitivity tests, in comparatively mild cases a soluble sulphonamide can be given as directed below, but in severe cases an antibiotic with a wide range of activity, e.g. tetracycline, should be substituted. When the bacteriological report is to hand, the drug of choice should be given.

Three and seven days after commencing the treatment of pyelonephritis the urine must be sent for culture. Even when a full initial course (ten days) of an antibiotic or chemotherapeutic agent has rendered the urine sterile, it is advisable to continue treatment with another drug for a further two to three weeks, or until the hourly white cell excretion rate has fallen to normal levels (less than 200,000 per hour). During this treatment further investigations to rule out abnormalities in the urinary tract should be carried out (p. 1062). Despite the undoubted efficacy of modern antibacterial drugs, none is likely to succeed in sterilising the urine of a patient with an abnormality of the urinary tract. A history of previous urinary infection also reduces the likelihood of a cure, although to a lesser extent (Garrod).

Colomycin and Gentamicin are the best drugs currently available for combating infections with the more resistant strains of *Pseudomonas Pyocyanea*, *Proteus*, and *Klebsiella*.

Chemotherapeutic and Antibiotic Agents

Sulphonamides:

Sulphafurazole (Gantrisin) is capable of high urinary concentration without risk of crystalluria, and is of low toxicity. It is capable of controlling many *Esch. coli* infections and some strains of *proteus* and *Ps. aeruginosa*. The dose is 2 G. *statim* followed

Lawrence Poul Garrod, Contemporary. Professor Emeritus of Bacteriology, University of London (St. Bartholomew's Hospital).
Theodor Albrecht Edwin Klebs, 1874-1913. German bacteriologist.

by 1 G. six-hourly until the temperature has remained normal for three days, and cultures of the urine have proved sterile on three consecutive occasions.

Sulphamethiazole (Urolucosil) has similar properties, but unlike other sulphonamides it is unnecessary to insist on a high fluid intake while the patient is taking this drug. The dose is 0.1 G. five times daily.

Antibacterial Nitrofurans:

Nitrofurantoin (Furadantin) is a synthetic antibacterial agent. Although when taken by mouth the blood levels remain low, the concentration in the urine is significant; hence it is of value only in genito-urinary infections. It is particularly effective against *Esch. coli*, *A. aerogenes* and *proteus*; often it has proved lethal to antibiotic-resistant strains of these organisms. The average dose by mouth is 5 to 8 mg. per kilo (2.2 to 3.6 mg. per lb.) of body weight. One-quarter of this dose is administered immediately after food three times daily and a further dose with cold milk last thing at night. If nausea or vomiting occurs, the dose must be reduced.

Antibiotics:

Broad Spectrum Antibiotics.—While these are effective if employed in accordance with bacteriological findings, when it is necessary to continue treatment for a prolonged period (which is not unusual) a number of dangers loom large. Firstly, there are the toxic properties, producing nausea, vomiting, and diarrhoea. Secondly, there is the alteration in the intestinal micro-flora these antibiotics produce, sometimes leading to moniliasis or staphylococcal enterocolitis (p. 3). Thirdly, the frequency with which resistant variants emerge, often in a comparatively short time, is disturbing.

Streptomycin, if employed, should be given in four-hourly doses of 0.5 G. intramuscularly for not more than forty-eight hours. Attention is again directed to the liability of the eighth cranial nerve to suffer damage if streptomycin is administered in the presence of depressed renal function.

CHRONIC PYELONEPHRITIS

Chronic pyelonephritis is an important, common, and dangerous disease. It is the most frequent cause of death from uræmia. Like its acute counterpart it can be primary, or secondary to abnormality of the urinary tract.

Pathology.—The inflammation is interstitial, and microscopically scars resulting from the destruction of adjacent parenchyma can be seen. These scars are unevenly distributed and nearly always are more evident in one kidney than in the other. Histologically it can be seen that the brunt of the onslaught has been borne by the renal tubules; they are atrophic, often dilated, and sometimes cystic. In time many of them are destroyed and disappear in scar tissue. On the other hand, the glomeruli retain their normal structure until late in the disease.

Chronic pyelonephritis is almost three times as common in females as it is in males. Two-thirds of the affected females are under forty years of age, while 60 per cent. of the males are over forty.

Clinical Features.—It is possible, but unusual, for chronic pyelonephritis to remain asymptomatic until advanced renal insufficiency appears.

Lumbar pain, dull in character, is present in 60 per cent. of cases.

Increased frequency and dysuria are leading symptoms in most cases.

Hypertension is present in 40 per cent. of cases, being more common in primary than in secondary pyelonephritis. It develops slowly and thus is more in evidence in long-standing cases. In 20 per cent. of cases the hypertension is of the accelerated ('malignant') type.

Constitutional symptoms comprising lassitude, malaise, anorexia, nausea,

and headache constitute the main complaint in 30 per cent. of cases. The disease often remains unrecognised in these sufferers, perhaps for years.

Pyrexia. Attacks of pyrexia exceeding 100° F. (37·8° C.) are the main symptoms in 20 per cent. of cases. The need to explain these attacks often leads to a thorough investigation including the urinary tract, and the disease is then brought to light.

Anæmia.—A normochromic anæmia due to unsuspected renal impairment may be the presenting feature.

The Urine. *Proteinuria.*—On account of the late destruction of the glomeruli, contrary to glomerulonephritis, the amount of protein passed in the urine is small (5 G. daily).

Casts are found infrequently.

White cells are numerous. A fresh three-hour urine should be examined and a count of over 200,000 per hour is abnormal—often it may be as high as several million. Ureteric specimens may reveal considerable difference in this respect between the kidneys, and on the ability of the individual kidney to concentrate and acidify urine.

Bacteriological Examination of the Urine.—Commonly found are *Esch. coli*, *Streptococcus faecalis*, *proteus* and *pseudomonas*.

Treatment is not easy. Two principles must be followed: (1) to eradicate predisposing causes such as obstructions or stones, and (2) by repeated cultures and sensitivity tests to control and, it is hoped, ultimately to eradicate the infection with repeated courses of suitable drugs. In secondary cases, even if the cause can be removed, the renal pelvises have frequently lost their musculature and are fibrotic and non-contractile. Moreover, once the parenchyma has been damaged it is a locus of predilection for blood-borne organisms. Consequently chemotherapy and antibiotics confer as a rule only temporary benefit. The longer the infection has persisted, the more difficult is it to eradicate. When prolonged treatment is required, there is a definite place for the following inexpensive drug.

Mandelic acid and its salts are often curative in coliform infections and those caused by *streptococcus faecalis*. Ammonium mandelate 2 G. (or better, mandamine, which is mandelic acid and hexamine combined), together with a capsule of 1 G. ammonium chloride, is given six-hourly. Fluids are restricted to 2½ pints (1·5 litres) in the twenty-four hours. It is essential that the urine be kept acid (pH 5·3 or less). Contraindications to this form of therapy are impairment of renal function and infections producing ammoniacal decomposition of the urine.

In nearly 50 per cent. of patients with pyelonephritis due to infection by one organism only, in whom infection persists in spite of treatment with antibacterial drugs, it is found that the original organism has been replaced by another, e.g. *Streptococcus faecalis*, instead of *Esch. coli*.

It is highly important to eradicate distant foci of infection whenever possible. In occasional cases when the disease is unilateral and associated with the accelerated type of hypertension, nephrectomy is sometimes curative. Partial nephrectomy, removing a badly damaged portion of renal tissue, may be necessary so helping to sterilise the urine and free the patient of pain.

Recurrent Dialysis.—In advanced cases, diminishing renal function may require institution of recurrent dialysis using an artificial kidney. Difficulty in controlling hypertension or renal infection may necessitate bilateral nephrectomy.

Transplantation of a kidney from a donor or a cadaver may be possible (p. 1120).

INVESTIGATION OF A CASE OF PYELONEPHRITIS

The following investigations are carried out in chronic cases a week or more after treatment with antibacterial drugs, and in acute cases after the infection has subsided with treatment. These investigations are to discover any cause of obstruction to the upper or lower urinary tract, and/or a source of infection, which is present most often in the genital organs or the urethra.

(a) *The Clinical Examination is Repeated.*—The kidneys are palpated for tenderness and enlargement, and the bladder for chronic retention of urine. In middle-aged or elderly males the prostate is examined for enlargement or fibrosis. In male children, atresia of the meatus or meatitis is sought. Inspection of the cervix uteri often displays cervicitis or cervical erosion. Examination of the secretion expressed by prostatic massage sometimes accounts for the presence of infection. If no infection is found in these situations, a possible focus in carious teeth or infected tonsils is sought.

(b) *Renal Function Tests.*—To assess the glomerular and tubular function (p. 1062).

(c) *Excretory Pyelography.*—The plain film of the urinary tract may reveal the presence of a urinary calculus. In chronic pyelonephritis, and after an attack of severe acute or recurring acute pyelonephritis, changes in the pyelographic shadow or shadows will be manifest. Some of these are similar to those of early hydronephrosis, but they are more unequally distributed. Although the calyces become flattened or clubbed, this is more in evidence in one part of the kidney than another. Persistent poor definition in one group of calyces is presumptive evidence of chronic infection. In cases of longer standing the renal pelvis becomes slightly dilated, but, unlike hydronephrosis, the dilatation of the calyces predominates. The most important X-ray sign is narrowing of the cortex, often localised, and giving rise to depressed scars seen as notches in the outline, or considerable contracture of one pole.

(d) *Cysto-urethroscopy.*—After the instrument has been passed, residual urine, if any, is measured. Examination of the urethra may show chronic urethritis or the presence of a stricture. In the female urethro-trigonitis is commonly found. Hitherto unconfirmed obstruction to the prostatic urethra by an enlarged middle lobe or contracture of the bladder neck may be demonstrated. Evidence of cystitis is the most common finding. Sometimes the ureteric orifice, or orifices in bilateral cases, is seen pouting, oedematous, or gaping. Delay in efflux is usual on the affected or more affected side. Ureteric catheters are passed and specimens collected from each kidney. These specimens are examined for cells and bacteria. By these means it is ascertained whether the infection is bilateral or unilateral. In male children valves in the posterior urethra, or a contracture of the bladder neck, are sometimes revealed.

(e) *Micturating Cine-cystography,* using a cine-camera coupled to an X-ray image intensifier, may reveal reflux of urine up one or both ureters. This can be a transient phenomena due to infection. It is more commonly a factor in childhood.

HYPERTENSION AND A UNILATERAL RENAL LESION

Renal ischæmia regularly produces increased arterial tension, as ischæmia of the renal parenchyma leads to the formation of a vasopressor substance. Too often cases are labelled essential hypertension without a thorough urological examination. Sometimes in a case of hypertension a unilateral diseased kidney is demonstrated and nephrectomy is followed by permanent lowering of the blood pressure. The most amenable lesion in this respect is chronic pyelonephritis with a short history of high blood pressure. Occasionally patients with a renal neoplasm, or renal calculi with infection, have been permanently benefited, in so far as hypertension is concerned, by removing the diseased kidney. Renal artery stenosis is a cause of hypertension (fig. 119). Direct arterial surgery (p. 121 and p. 135) may be curative.

PYONEPHROSIS

The kidney is converted into a multilocular sac containing pus or purulent urine, with a varying amount of renal parenchyma in its walls. A pyonephrosis can result from infection of a hydronephrosis, follow acute pyelonephritis or, most commonly, arise as a complication of renal calculus (calculous pyonephrosis). As a rule the condition is unilateral. Like hydronephrosis, a pyonephrosis can be open, closed, or intermittent.

Clinical Features.—The classical triad of pyonephrosis is anaemia, fever, and a loin swelling. When the condition arises as an infection of a hydronephrosis the swelling may be a large one. When the pyonephrosis is open, an evening rise of temperature to 99° or 100° F. (37·2° or 37·7° C.) is usual; if the pyonephrosis becomes closed, the temperature is higher. Pyuria, when present, gives rise to cystitis, and sometimes symptoms of that condition are the chief complaint.

Investigation.—A plain X-ray may show a calculus. Excretory pyelography shows the same vagaries as advanced hydronephrosis, in that the appearance of the shadow of the dilated renal pelvis and calyces is long delayed and may be absent. Cystoscopy often reveals cystitis with a purulent efflux from the ureter of the affected side. In closed cases the bladder may be normal and a ureteric catheter either becomes arrested, usually at the pelvi-ureteric junction, or passes the obstruction and gives exit to pent-up purulent urine.

Treatment.—In early cases it is occasionally possible to convert the pyonephrosis into a hydronephrosis by antibiotic drugs, and the passage of a ureteric catheter to drain the infected urine from the renal pelvis. Should the pyonephrosis be due to an impacted stone in the ureter, or a comparatively small stone blocking the pelvi-ureteric junction, the stone should be removed and suitable drainage of the kidney provided. In most other unilateral cases, when any associated anaemia has been rectified, nephrectomy is undertaken. Should the capsule prove to be densely adherent to the surrounding structures subcapsular nephrectomy (p. 1120) is performed. Rarely, when the patient's general condition cannot be improved, nephrostomy is carried out, a measure to be avoided if possible because it renders subsequent nephrectomy more difficult. When a pyonephrosis arises in a single kidney, or the condition is bilateral, permanent nephrostomy may be the only possible measure. Drainage by an 'ileal conduit' to bladder or the skin is another possibility (p. 1159).

CARBUNCLE OF THE KIDNEY

The source of origin of the organism is usually a cutaneous lesion, such as a boil, carbuncle, whitlow, or an abscess of the breast, and the *Staphylococcus aureus* is conveyed to the kidney via the blood-stream. Occasionally a blow on the loin determines the issue.

Morbid Anatomy.—On splitting open the kidney there is a necrotic mass of tissue, usually localised, involving the parenchyma (fig. 1442).

Clinical Features.—There is an ill-defined, tender swelling in the loin, persistent pyrexia and leucocytosis, signs that closely simulate a perinephric abscess. In early cases the urine contains neither pus cells nor organisms, but after a few days staphylococci can sometimes be isolated. Pyelography often shows compression or

obliteration of a group of calyces, an appearance that does not differ from adenocarcinoma of the kidney.

Treatment.—Antibiotics have cured some early cases. When the response to antibiotic therapy is not dramatic the kidney must be exposed, the carbuncle incised,

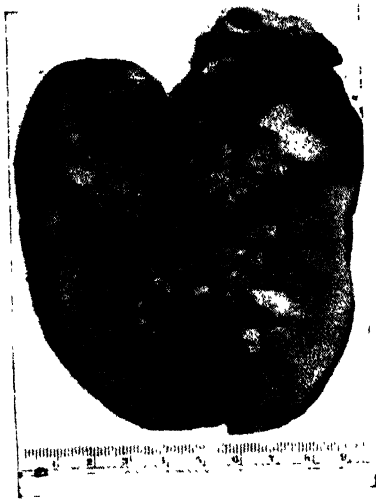


FIG. 1442.—Carbuncle of the kidney.
(J. G. Yates-Bell, F.R.C.S., London.)

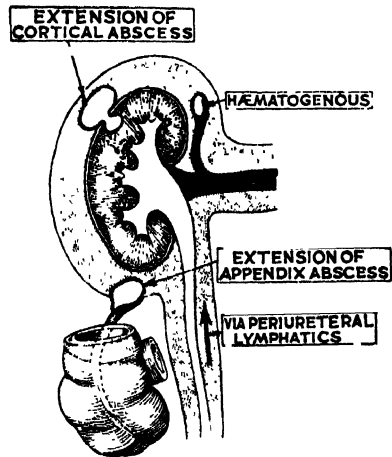


FIG. 1443.—Sources of perinephric abscess.

and the necrotic material removed. Hæmorrhage is not excessive and can be controlled by pressure with a moist warm pack. A drainage tube is passed down to, but not into, the resultant cavity and the wound is closed around the tube. Antibiotics are continued for at least ten days. Primary nephrectomy is indicated only when the major part of the kidney is involved by the carbuncle.



FIG. 1444.—A large perinephric abscess.

PERINEPHRIC ABSCESS

The most common causes are depicted in fig. 1443. Other causes are infection of a perirenal hæmatoma, and perforation of the renal capsule from undue delay in operating upon a pyonephrosis or a renal carbuncle. A tuberculous perinephric abscess can arise from an advanced pyonephrosis or from tuberculosis of a nearby vertebra.

Clinical Features.—The classical symptoms and signs of a perinephric abscess are a high, swinging temperature, rigidity, tenderness, and fullness in the loin (fig. 1444). If the suppuration commences in the lower part of the peri-

nephric fat, local signs present early, but when the suppuration is confined to the upper portion of the perinephric fat which lies beneath the lower ribs, it produces no visible swelling, and even rigidity and tenderness may be absent. The leucocyte count is always raised, often to 20,000 per c.mm. As a rule no pus or organisms are present in the urine.

Radiography shows obscuration of the psoas shadow, with one or more of the following additional signs—scoliosis with concavity towards the abscess, and elevation and immobility of the diaphragm on the affected side.

Mathé's Sign.—Absence of the downward displacement that occurs in the erect posture in every normal kidney. Two radiographs, one in the lying posture and one in the erect posture, during excretory pyelography are required. In established cases the kidney can be shown to be displaced forwards if a lateral view is obtained.

Treatment.—Under antibiotic cover a lumbar incision is made large enough to enable the surgeon to open up pockets both above and below the kidney. The surface of the kidney is palpated for an unruptured cortical abscess, which, if present, should be incised. A specimen of pus having been obtained for bacteriological examination, the wound is closed with ample drainage. Appropriate antibiotic treatment follows.

RENAL TUBERCULOSIS

Ætiology and Pathology.—Tuberculosis of the urinary tract cannot arise except as a blood-borne infection from a distant focus, which, in many cases, is impossible to locate.

When clinically recognisable, in the great majority of cases renal tuberculosis is apparently confined to one kidney. A group of microscopical lesions coalesce and discharge pus and tubercle bacilli along one set of tubules, and the pyramid which gives exit to the discharge becomes ulcerated (fig. 1445 A).

Course : *Tuberculous bacilluria* is a convenient term to designate a very early lesion of the renal cortex when no abnormality in the affected kidney can be detected, even by retrograde pyelography. It indicates the presence of active tuberculosis disease which may progress, and as such must be treated in the same way as the demonstrable lesions.

A *macroscopical lesion* progresses rather slowly, and other similar lesions appear in the same kidney. The infection can be confined to the affected kidney for months or years, causing one or other of the changes depicted in fig. 1445 C to G. More often the disease spreads along the ureter (tuberculous ureteritis) to give rise to tuberculous cystitis in both sexes and prostatitis, vesiculitis and later epididymitis in the male.

In the male, renal and genital tuberculosis can be associated before the bladder and posterior urethra are involved, in which event the genital infection probably occurs in the following way. An urgent desire to micturate causes the internal sphincter to open, while at the same time, because there are no immediate facilities for emptying the bladder, voluntary closure of the external sphincter is called into play. At that moment infected urine is forced under pressure into the mucosal crypts of the prostate and the common ejaculatory ducts (Wells).

Clinical Features.—Renal tuberculosis usually occurs between twenty and forty years of age. Men are affected twice as commonly as women, and the right kidney somewhat more commonly than the left.

Frequency.—The earliest symptom is increased frequency of micturition. Often this is the only symptom. It is progressive, the patient complaining

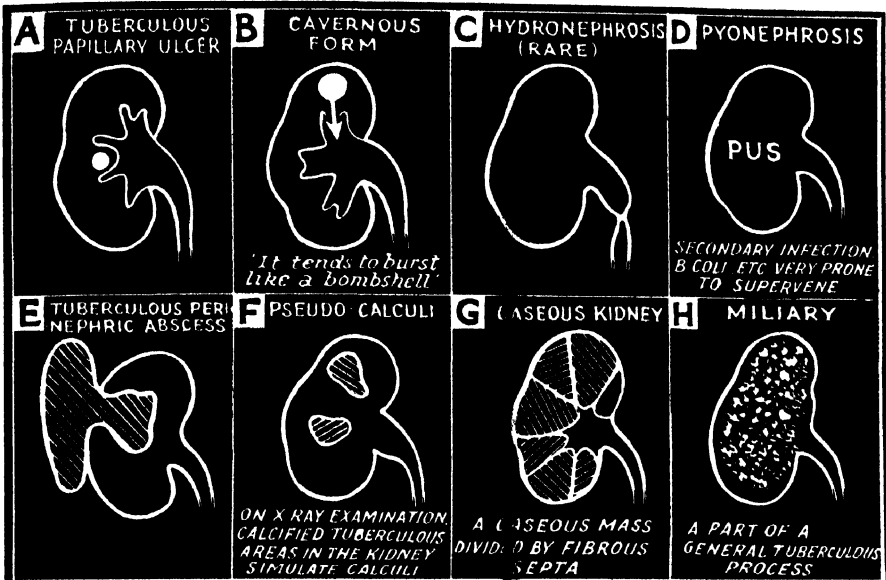


FIG. 1445.—Blackboard sketches of types of tuberculous kidney (macroscopic pathology).¹

that (over a period of months) the frequency has increased both by day and by night.

The causes of the frequency are, in order of sequence:

1. Reflex; the inflamed kidney causes the micturition reflex via the spinal cord to become hypersensitive. It occurs especially at night in a young adult.
2. Tuberculous cystitis (the usual cause at the time of presenting).
3. Secondary infection.
4. Progressive diminution of bladder capacity from fibrosis.
5. Polyuria. The output of the inflamed hyperæmic kidney is increased.
6. Irritation of the bladder by caseous debris passed down the ureter.

Pyuria.—In early cases the urine is pale and slightly opalescent. The presence of pus cells without organisms in an acid urine is very suggestive of tuberculous urinary infection (Bacteriological examination, p. 1110).

Painful Micturition.—Once tuberculous cystitis has set in, micturition becomes increasingly painful. First there is suprapubic pain if the bladder cannot be emptied immediately; later a burning pain accompanies micturition, and when secondary infection has occurred there is superadded agonising pain after micturition referred to the tip of the penis or to the vulva, often associated with terminal hæmaturia (strangury).

Renal pain is often absent, but sometimes the patient complains of a dull ache in the loin.

Hæmaturia.—In 5 per cent. of cases the disease is ushered in with painless hæmaturia occurring from an ulcer situated on a renal papilla. During the hæmorrhage, and after it has ceased, investigations may or may not demonstrate the presence of a renal lesion and tubercle bacilli in the urine.

¹ G is known also as the 'putty kidney' and by the French as the 'cement kidney.' It is nature's method of performing (ineffective!) autonephrectomy.

In the latter event more typical symptoms commence some months later. Apart from a few drops of blood at the end of micturition in cases complicated by severe cystitis, macroscopical hæmaturia occurs only occasionally.

A tuberculous kidney is œdematous and friable, and is therefore more likely to be damaged by trauma than is the normal pliable kidney.

Constitutional Symptoms.—A continuous slight loss of weight is usual. Evening pyrexia to 99° F. (37·3° C.) occurs when the disease is fully established. A high temperature is indicative of a secondary infection or disseminating tuberculosis.

On Examination.—It is unusual for a tuberculous kidney to be palpable

When a patient with renal tuberculosis has an enlarged kidney which can be felt, it is by no means certain that this kidney is the one that is diseased, for compensatory hypertrophy sometimes renders the healthy kidney both large and tender.

The prostate, vesicles, vas, and epididymis should be examined for nodules or thickening, which may indicate tuberculous invasion.

Investigation :

Bacteriological Examination of the Urine.—Prompt examination must be made of a midstream specimen taken first thing in the morning before contaminants can overgrow. When, after staining a specimen of the sediment with Ziehl-Nielsen's stain, acid-fast bacilli are found, it is highly probable that they are tubercle bacilli, but the proof lies in culture or guinea-pig inoculation, either of which is reliable. **Most important—specific therapy should never be started until a positive culture has been obtained.** If this precaution is neglected, an exacting course of treatment may be started for a non-tuberculous condition, and at a later stage it is impossible to prove or disprove the diagnosis.

In few fields of medicine is the diagnosis more exact. The microscope demonstrates tubercle bacilli in 72 per cent., the guinea-pig test in 94 per cent., and the culture medium in 98 per cent. of cases.

A plain radiograph sometimes shows areas of calcification in the kidney

Excretory pyelography reveals fairly typical changes (figs. 1446 and 1447). The earliest manifestation is persistent irregularity and slight dilatation of a calyx from œdema of the neck of the calyx. Gross lesions of the cavernous type are readily apparent. In late cases, the contralateral ureter becomes dilated also; this is not necessarily an indication of disease on the opposite side, for the dilatation may be due to the cicatrised bladder causing obstruction to the ureter in its intramural course.

Cystoscopy.—When increased frequency is the only symptom and it has not been present long, the bladder and the ureteric orifices are normal.

A unilateral cloudy efflux is sometimes observed. Either more frequent efflux or a delay in excretion of indigo-carmin is indicative of the side that is diseased. In 10 per cent. of cases there is an absence of efflux on the affected side, and no pathological changes in the bladder. These are examples of a closed ureter leading to so-called autonephrectomy, or a pyonephrosis.

Edema and pallor around a ureteric orifice are the earliest cystoscopic signs of vesical involvement. Infiltration follows, and the mucous membrane of the affected ureteric orifice pouts. Tubercles appear, usually



FIG. 1446.—Excretory pyelogram showing a small localised lesion. Healing took place under conservative treatment. (*Professor E. Wildbolz, Berne.*)



FIG. 1447.—Excretory pyelogram showing more advanced renal tuberculosis with involvement of the ureter. Nephro-ureterectomy was performed. (*Professor Wildbolz.*)

lateral to the ureteric orifice, and later in the dome of the same half of the bladder. By confluence of a circumscribed group of tubercles, a tuberculous ulcer, which has an irregular periphery, may form. In long-standing cases, as a result of sclerosing peri-ureteritis, which causes shortening of the ureter, the ureteric orifice becomes displaced upwards and its mouth remains open (the so-called 'golf-hole' ureteric orifice). As the disease progresses, the capacity of the bladder steadily diminishes. At any stage, if a secondary infection supervenes, the intense generalised inflammation of the bladder wall obliterates many of the characteristic features of urinary tuberculosis just described above.

Retrograde pyelography may be necessary when investigating a suspicious pyuria if renal function is poor and tubercle bacilli have not been seen in the Ziehl-Nielsen film. Appearances, such as shagginess of a calyx or an abscess of the parenchyma connected to a calyx by a narrow neck, are often displayed by this method when excretory pyelograms appear normal. Retrograde pyelography is also of great assistance in cases of unilateral renal tuberculosis too advanced to concentrate dye given intravenously (fig. 1448). Remember, however, that the diagnosis depends on finding bacilli and *not* on the pyelographic appearances.



FIG. 1448.—Retrograde pyelogram showing advanced tuberculosis of the right kidney and ureter.

Examination of the Lungs.—A radiograph is necessary to exclude active or previous pulmonary tuberculosis.

Treatment.—Streptomycin, P.A.S., and I.N.A.H. are commenced as soon as the diagnosis has been established, and should be continued for a minimum of two years (p. 23). It is essential to have the patient under observation when commencing treatment. The liability to serious side-effects from streptomycin will usually become obvious within the first three weeks. Streptomycin must be given with great care and controlled by serum levels if renal function is impaired. Ideally the first six months, at least, should be spent in a 'sanatorium' where the patient becomes resigned to taking rather unpleasant drugs in large amounts (as other patients are doing

the same), and the problem of the patient who asserts that he is taking the drugs prescribed when in fact he is not, can be overcome.

Prognosis in renal tuberculosis has improved enormously since the advent of chemotherapy and antibiotic treatment, and if the regimen detailed is carried out properly there should be a very low incidence of recrudescence of infection.

Operative Treatment.—Some lesions are surrounded by so much necrotic and fibrous tissue that antibiotic agents do not reach the organisms in sufficient concentration to kill them. Unilateral lesions of this character, and those in which the disease is more advanced, call for operative treatment, the optimum time for which is after four to six months of antibiotic treatment.

According to circumstances one of several operations is carried out. It must be emphasised that conservative surgery should be the aim, and several conservative procedures may prove necessary on one or both kidneys and ureters during the course of the disease. The fact that a horseshoe kidney is involved does not alter this principle, neither does the fact that one kidney may have been removed already.

1. **Renal Cavernotomy** (Hanley).—As the primary lesion of the calyx heals (fig. 1445 A and B) fibrosis tends to cause a stricture at the neck of the calyx. Radiological disappearance of such a cavity should be viewed with suspicion, and usually indicates that there is a closed pyocalyx which remains infected, as its avascularity prevents the drugs from reaching the tubercle bacilli present. Sometimes such a pyocalyx will re-open and discharge its contents some six months after starting treatment, but if this does not occur after a year and a calyx is radiologically 'missing', a drainage operation must be undertaken.

The kidney is approached as described on p. 1119 and after clearing the perinephric fat, the abscess is aspirated with a syringe and wide-bore needle. The roof of the abscess is removed with scissors and its caseous contents swabbed out until healthy granulation tissue is reached. Any bleeding from the cut edges is controlled with fine catgut stitches (fig. 1449). The wound is closed without drainage. As the cavity is closed off from the pelvis (if it is not, the operation is unnecessary), there is no leakage of urine. Multiple abscesses in both kidneys can be dealt with in this way.

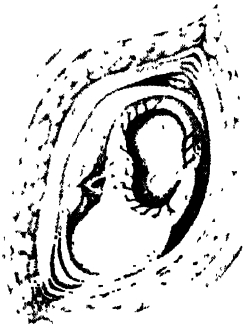


FIG. 1449.—Renal cavernotomy completed. (After H. G. Hanley.)

2. **Nephro-ureterectomy.**—The indication is a unilateral lesion with gross impairment of renal function on the affected side.

In such advanced cases the ureter as well as the kidney usually proves to be diseased, and nephro-ureterectomy (fig. 1451), in which, through a separate incision, the ureter is divided and ligated at its entrance to the bladder before commencing lumbar nephrectomy, is the operation of choice. By total removal of the ureter a possible source of continued infection of the bladder is removed, and the incidence of post-operative wound infection and sinus formation is reduced.

3. **For Hydronephrosis.**—This is due to tuberculous cicatricial contracture and healing of the bladder around the lower end of the ureter or at the pelvi-ureteric junction. A procedure similar to the Anderson-Hynes operation (p. 1087) should be performed.

4. **For Systolic bladder** (syn. 'thimble bladder' (fig. 1493)).—This is due to healing by fibrosis and often leads to extreme frequency of micturition. The best



FIG. 1450.—Typical cavernous lesion upper pole; smaller lesion communicating with lower calyx.



FIG. 1451.—Tuberculous pyonephrosis. Nephro-ureterectomy.

operation is the type of ileo-cystoplasty described by Hanley in which a 6-inch (15-cm.) opened-out loop of terminal ileum is sutured to the bladder, from which the dome has been removed to provide a maximum opening.

ABACTERIAL PYURIA

That sterile pyuria indicates urinary tuberculosis is almost axiomatic. Nevertheless, a small group of cases of abacterial pyuria is definitely non-tuberculous. The symptoms simulate renal tuberculosis very closely, and it is only when repeated examinations of the urine, and in the male the seminal fluid, fail to reveal *mycobacterium tuberculosis* or other bacteria, and cultures and guinea-pig inoculations are negative, that the diagnosis can be made. Pyelography occasionally shows slight dilatation of the renal pelvis and ureter of one or both sides; more usually this examination is negative. Cystoscopy reveals a severe cystitis and often diminished bladder capacity. Occasionally amœbiasis is responsible.

Treatment.—Aureomycin usually cures the condition in three days. In cases resistant to this treatment 0.3 G. of nearsphenamine intravenously weekly for four weeks is often successful. In rare instances, when amœbiasis is responsible, treatment with emetine hydrochloride will eradicate the infection.

NEOPLASMS OF THE KIDNEY

Benign Neoplasms

Adenoma.—Pea-like cortical adenomas are sometimes found at necropsy. They give rise to no symptoms, and are only of academic interest.

Angioma may give rise to profuse hæmaturia (fig. 1452).

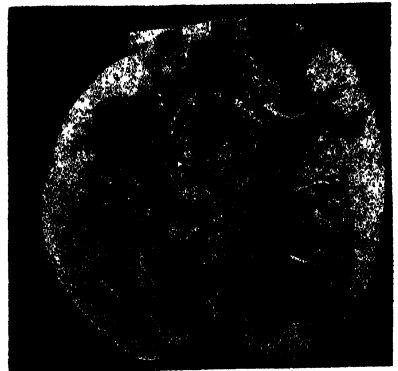


FIG. 1452.—Angioma of the kidney. Nephrectomy for profuse, painless hæmaturia extending over five years.

Truly benign tumours of the kidney are so rare that a good rule is that *all neoplasms of the kidney which can be recognised clinically should be considered malignant and treated as such.*

Malignant Neoplasms

Clinically neoplasms of the kidney are divided into two classes :

In children between the ages of one and seven.

In adults after the age of forty.

Between the ages of seven and forty malignant neoplasms are unusual.

IN CHILDREN

Wilms's tumour (*syn.* nephroblastoma) is a mixed tumour containing epithelial and connective tissue elements arising from embryonic nephrogenic tissue, situated originally in one or other pole of the kidney. In infancy such tumours are very occasionally bilateral. Far more common is a unilateral tumour. They usually occur during the first four years of life.

Pathology.—*Macroscopically.*—On section the neoplasm is a greyish-white or pinkish-white colour; its consistence varies with the rapidity of the growth, i.e. the more rapid the growth, the softer the tumour.

Microscopically it is composed mainly of two types of cells—epithelial and connective-tissue. Cartilage, bone, and smooth or striped muscle fibres are occasional findings. The epithelial and connective-tissue cells exist side by side, but one type is usually predominant. Thus the tumour is composed of mixed cellular elements, some of which are radio-sensitive and some radio-resistant. Consequently, the radio-resistant elements show a continuance of activity in spite of radiotherapy.

Clinical Features :

Abdominal Tumour.—The symptomatology is always the same. An abdominal tumour (fig. 1453) appears and progresses rapidly, while the general health deteriorates. Examination of the abdomen reveals a mass which may be enormous; the bulk of the tumour is on one side of the abdomen. Wilms's tumour tends to grow within a capsule, pushing the rest of the kidney aside; thus the reniform shape of the kidney is lost early.



FIG. 1453.—
Wilms's tumour.

Pyrexia.—Half these patients have some elevation of temperature, which disappears if the tumour is removed.

Hæmaturia is a sign of ill omen. It denotes that the previously encapsulated tumour has burst into the renal pelvis, and all patients suffering from a Wilms's tumour with this sign die within nine months (Scott).

Pyelography shows gross deformity of some of the calyces.

Metastases.—Wilms's tumour metastasises early, mainly by the blood-stream to the lungs, less commonly to the liver, rarely to bones, and exceptionally to the brain. Lymphatic dissemination is much less common.

Differential Diagnosis.—From a practical standpoint there is but one swelling with which Wilms's tumour can be confused, and that is a retroperitoneal neuroblastoma, but as the treatment is precisely the same such differentiation is an academic

exercise. However, if radiography reveals bone metastases, this distinctly favours a neuroblastoma. Although these abdominal tumours arouse considerable diagnostic interest, for fear of accelerating dissemination, multiple examinations by, for instance, a class of students, must be forbidden.

Treatment.—*Immediate nephrectomy and post-operative radiotherapy.* The earlier nephrectomy can be carried out, the better. A few hours may be necessary to correct anæmia by transfusion. Radiotherapy should be commenced immediately after operation, and 200 rads given daily to a total of 4,000 to 4,500 rads.

Prognosis.—Under the age of one year 80 per cent. survive five years, over this age it drops to 30 per cent. Recurrences usually occur within a year, so a child surviving eighteen months is probably cured.

Bilateral Cases and Inoperable Cases.—Radiotherapy should be given to the renal areas. There is some hope that chemotherapeutic agents and particularly actinomycin D may prove of value in these cases.

IN ADULTS

1. Grawitz tumour (*syn.* adenocarcinoma; hypernephroma) is the commonest neoplasm of the kidney (75 per cent.). It arises in the cortex, possibly from a pre-existing adenoma, probably *per primam* in cells of the uriniferous tubules.

Pathology.—A tumour of moderate size is spherical in shape, and it often occupies one or other pole, the seat of election being the upper pole; less often it is in the central portion of the kidney. On section it is characteristically yellow (due to lipoid); less often it is dull white, or semi-transparent. Hæmorrhagic areas are often seen. The tumour is divided into numerous lobules by fibrous septa (fig. 1454). The larger the tumour the more extensive is central hæmorrhage and necrosis.

Microscopical Structure.—The most common appearance is solid alveoli of cubical or polyhedral clear cells, with deeply stained small rounded nuclei and abundant cytoplasm containing lipoids, cholesterol, and glycogen. The cells may also be arranged in the form of papillary cysts or tubules. In a much smaller percentage the cells are granular (dark). Clear and dark cells can co-exist in different parts of the same tumour. In all cases the stroma is scanty but rich in large blood-vessels, the walls of which often appear, in places, to consist of tumour cells.

Spread.—As the tumour enlarges, it encroaches upon a group of calyces and is prone to grow into the renal veins. Pieces of growth becoming detached are swept into the circulation, to become arrested particularly in the lungs ('cannon balls') and bones. Occasionally, when a secondary growth

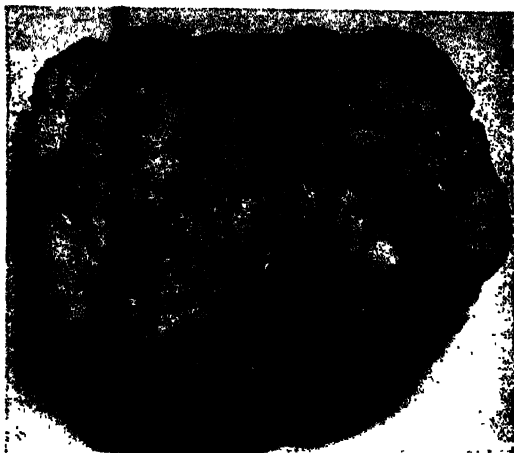


FIG. 1454.—Adenocarcinoma of the kidney.

appears in a long bone, it remains the only metastasis for perhaps a year or more.

While spread by the blood-stream predominates, spread by lymphatics occurs also. If the tumour bursts through the renal capsule into the perirenal tissues, it then metastasises to the lymph nodes in relation to the hilum of the kidney, and from there to the para-aortic and other lymph nodes.

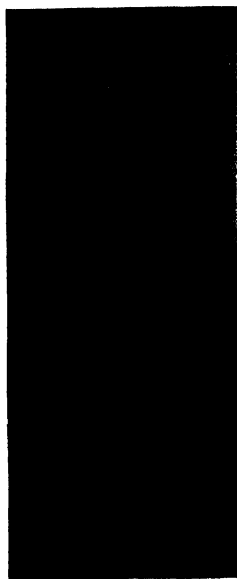


FIG. 1455.—Radiograph showing metastases from a Grawitz tumour.

Clinical Features.—Men are more often attacked than women, the ratio being 2 : 1. Typically the first sign is intermittent hæmaturia. Clot colic may follow. Dragging pain in the loin is sometimes a leading symptom. Occasionally the first manifestation is a palpable renal swelling. In the adult male a rapidly oncoming varicocele is a rare but impressive sign.¹

Atypical Cases.—(a) In 25 per cent. of cases the primary growth remains 'silent', the patient presenting because of secondary deposits such as a painful enlargement of a bone (fig. 1455), a spontaneous fracture, persistent cough, or hæmoptysis.

(b) Occasionally persistent pyrexia (100° to 102° F. (37·8° to 38·9° C.)) is the only symptom, there being no infection to account for the temperature. It is presumably due to absorption of blood and necrotic material. Such pyrexia usually disappears after nephrectomy: should it persist metastases are present.

(c) In a small group of cases the patient presents on account of lassitude and is found to be extremely anæmic, and anæmia being out of proportion to that which could be expected from the hæmaturia, if such be present.

(d) 'Polycythæmia' occurs in 4 per cent. of cases. The sedimentation rate is *always raised* above the 1 to 2 mm. found in idiopathic polycythæmia vera. The blood count will return to normal after nephrectomy, but a return of the polycythæmia has been known with development of metastases.

Early Diagnosis.—By the time a patient has the classical triad of symptoms, viz. hæmaturia, pain, and a palpable renal tumour, he nearly always has metastases. It is therefore of paramount importance to endeavour to make an early diagnosis, and any one of these symptoms calls for a thorough renal investigation. Patients with hæmaturia should be examined cystoscopically while the bleeding is in progress. If blood is seen issuing from one ureteric orifice, and there is a clear efflux from the other, the information gained is of considerable importance, but is by no means conclusive evidence of a renal neoplasm. Extremely suggestive is bleeding occurring from a palpable kidney. When blood is not seen issuing from a ureter, but an enlarged middle lobe of the prostate or a papilloma of the bladder is discovered, it must not be assumed that the source of the hæmorrhage has been found.

¹ A left-sided varicocele is more likely, as the left spermatic vein joins the left renal vein.

Investigation must proceed until each kidney has been proved to be normal or culpable.

Pyelography.—The early diagnosis rests almost entirely on pyelography.

Excretory pyelography may be inconclusive in early cases, because of lack of precise definition, and in late cases because there is poor or absent concentration of the medium, but at all times it is of great value in determining the function of the contralateral kidney. The presence of a projection in the cortical outline is important.

Retrograde pyelography is often required because of the better delineation it affords. The principal pyelographic changes are:

(a) Filling defects due to invasion of one or more of the minor or major calyces. Very characteristic is failure of the medium to enter one major calyx.

(b) Elongation and compression of one or more calyces and sometimes of the renal pelvis causing the 'spider-leg' deformity (fig. 1456). In congenital cystic kidneys the 'spider legs' are broader, more clearly cut, and involve most if not all of the calyces; also, the condition is bilateral.

(c) Displacement of the renal pelvis and the distorted calyces downwards in growths occupying the upper pole. Growths of the lower pole often displace the ureter inwards.

(d) In advanced cases (which may give no shadow on excretory pyelography) the interior of the calyces and renal pelvis are so encroached upon that they are represented by a few, irregular, widely separated shadows.

A radiograph of the thorax should always be taken before deciding the best course of treatment. It is not rare for a hitherto unsuspected metastasis or metastases in a lung to be revealed in this way.

Differential Diagnosis.—(a) Hæmorrhage into a hydronephrosis, (b) congenital cystic kidney with hæmaturia, or (c) a solitary cyst of the kidney can closely simulate a renal neoplasm, or (d) aneurysm of a renal artery.

Treatment.—In cases without metastases the treatment is nephrectomy with removal of the perinephric fat.

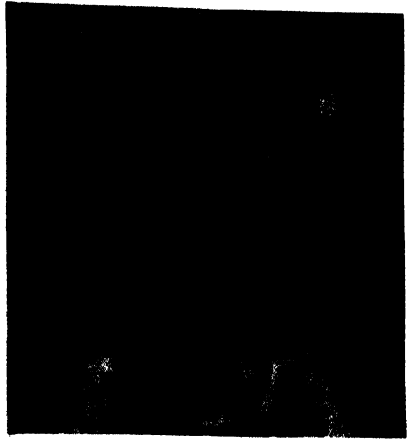


FIG. 1456.—Pyelogram in a case of hypernephroma of the left kidney. The only symptom was one attack of painless hæmaturia.

The incision is a matter of personal preference, the objective being to gain wide exposure of the enlarged kidney and especially of its pedicle. The abdominal approach (p. 1119) is best for accomplishing the following manoeuvre.

Preliminary Division of the Renal Pedicle.—It is advisable to deal with the renal pedicle before manipulating the kidney, otherwise blood-borne metastases may occur as a direct result of the operation. Before undertaking ligation and division of the renal pedicle the renal vein should be palpated delicately between the finger and thumb for a tumour-thrombus which, if present, calls for opening the vein and

extracting the thrombus. Similarly, should thrombus be seen within the severed renal vein, while the assistant compresses the inferior vena cava, the ligature should be cut in the hope that the remains of the tumour will be swept out in the hæmorrhage that ensues, or that it can be extracted before reclosing the vein (Donovan's *man-œuvre*, p. 71).

In other respects the details of the operation are those described on p. 1119.

When an extremely large tumour is encountered the incision can easily be extended upwards into the thorax.

Radiotherapy:

Pre-operative irradiation sometimes renders a large tumour smaller but, because of the uncertainty and the delay in operation entailed, pre-operative irradiation is not often advised.

Post-operative irradiation has been proved statistically to be of value.

In *inoperable cases* radiotherapy often results in dramatic regression of a metastasis, but too often the improvement is only temporary, and there follow other metastases that are less radio-sensitive.

Cytotoxic chemotherapy (e.g. methotrexate vinblastine) may have a place during surgery to kill circulating cells, and, in conjunction with super-voltage therapy, in treating metastases.

Prognosis.—Even some of the largest neoplasms have been followed by a permanent cure. In operable cases 44 per cent. of the patients are alive and well after three years, and 30 per cent. after five years. If there is macroscopic involvement of the renal vein or its branches, the prognosis is poor.

Grawitz Tumour with a Solitary Metastasis.—A number of patients with a solitary metastasis in a long bone have been treated with varying success by wide local excision and bone graft, or by amputation in addition to nephrectomy. In not a few the neoplasm of the bone has received prior treatment in the belief that it was a primary bone tumour, the true nature of the growth being revealed by the pathological examination. On several occasions a solitary metastasis in the lung has been removed by lobectomy, and in a few instances a metastasis has been excised from other situations.

2. **Papilloma of the renal pelvis** is similar in structure to papilloma of the bladder. It tends to invade the kidney proper, and to take on malignant characteristics. It also spreads down the ureter and may invade the bladder.



FIG. 1457. — Pyelograph showing a papilloma of the right renal pelvis. (Professor Carl Krebs, Aarhus, Denmark.)

Clinical Features.—Hæmaturia is often continued for months at a time. A renal swelling is absent, except in rare cases where the pelvi-ureteric junction becomes occluded and a hæmonephrosis results, in which event there is lumbar pain. Clot colic may occur.

Pyelography reveals a characteristic filling defect of the renal pelvis (fig. 1457).

Treatment.—Nephrectomy with extended ureterectomy is the correct treatment. The intramural portion of the ureter along with the ureteric orifice must be excised in continuity by sleeve resection of the bladder wall.

3. **Squamous-celled carcinoma of the renal pelvis** is the least common variety of malignant disease

of the kidney. In some instances it appears to be preceded by leukoplakia, and in most cases a stone or stones are present.

Unless the stone gives rise to symptoms, this is the most elusive of all renal neoplasms, for it causes indefinite abdominal pain which finally becomes localised in the region of the involved kidney. Hæmaturia is slight, or to be found only on microscopical examination of the urine. Excretory pyelography shows a kidney excreting little or no medium. Retrograde pyelography sometimes reveals a deformed renal pelvis. Owing to diagnostic difficulties, by the time nephrectomy is performed there are often widespread metastases that are radio-resistant. This neoplasm has the worst prognosis of any renal neoplasm.

Primary neoplasms of the ureter are rare, and similar to those of the renal pelvis. The symptoms are identical with those of a neoplasm of the renal pelvis, although hydronephrosis, hæmonephrosis, and pyonephrosis are more frequent accompaniments. Occasionally a portion of a papillomatous growth can be seen protruding from a ureteric orifice. Usually the diagnosis is made by pyelography.

For a localised tumour the treatment is wide resection of that part of the ureter containing the tumour, followed by bridging the gap by means of an isolated segment of ileum, viz. →

In the case of papillomas of the ureter, and other diffuse neoplasms, nephro-ureterectomy, including that segment of the bladder containing the intramural portion of the ureter, is required. On the whole, the prognosis of tumours of the ureter is very poor, largely on account of late diagnosis.



EXPOSURE AND REMOVAL OF THE KIDNEY

The kidney lies in a deep recess beneath the rigid bulwarks of the thoracic cage and, having regard to the possibility of a short renal pedicle, no single incision will meet all requirements.

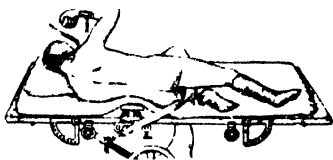


FIG. 1458.—Position on the operating table for exposure of the kidney by Morris's incision.

The Oblique Lumbar Incision (Morris).—The patient is placed in the lateral 'kidney' position (fig. 1458), and is held in this position by a broad band of adhesive strapping, which is fixed to one side of the table and passed over the pelvis to the other side. The arm-rest also steadies the patient, and, in addition, relieves the chest of the weight of the arm, which would otherwise embarrass respiration. The incision commences $\frac{1}{2}$ inch (1.25 cm.) below and

$\frac{1}{2}$ inch lateral to the angle made by the twelfth rib with the outer border of the sacrospinalis, and it passes downwards and outwards towards the anterior superior iliac spine (fig. 1459) and all muscular layers are incised until the extraperitoneal fat is reached. If the access obtained is inadequate, the twelfth rib can be dislocated upwards or resected. The renal pedicle is cleared of fat so as to display its vascular components. After isolating the ureter, it is divided between hæmostats as far from the kidney as is convenient; its distal end is ligated. Commencing inferiorly, a portion of the vascular pedicle is caught in a long hæmostat, care being taken to avoid the renal pelvis. A short hæmostat is placed in juxtaposition nearer to the kidney, and the tissue between the hæmostats is divided. The process is repeated until all the pedicle has been severed, thus freeing the kidney. At the most, three such sectionings are required. Each moiety of the renal pedicle is ligated by transfixion with an aneurism needle, which makes slipping impossible. Only when it is certain that hæmostasis is complete are the ends of the ligatures cut, and the wound closed, with drainage.

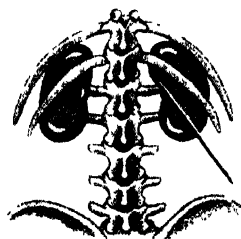


FIG. 1459.—Morris's incision.

Transabdominal nephrectomy is frequently employed for the removal of renal tumours, as it gives an unrivalled approach to the renal vessels which must be tied

before the tumour is handled, in order to prevent cells being displaced into the circulation by the manipulations. A transverse or paramedian incision is used.

Subcapsular Nephrectomy.—In order to prevent damage to densely adherent structures such as the duodenum, colon, and spleen, the kidney may be removed by incising the capsule along the convex border and dissecting it free to the hilum, where it is again incised to allow access to the renal pedicle.

Partial Nephrectomy (Semb).—A wide lumbar exposure with resection of the twelfth rib is required. Bleeding is controlled by compression of the appropriate branches of the renal vessels. The stages of the excision are shown in fig. 1460.

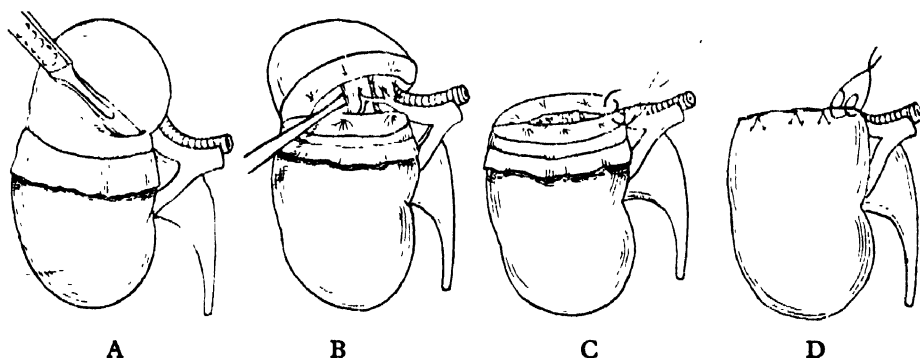


FIG. 1460.—(A) Incising the anterior wall of the kidney; cuff of capsule turned back. (B) Isolating the trunk of the affected calyx. (C) Closing the renal parenchyma. (D) Approximating sutures over the flap of capsule which has been returned to cover the stump. (After C. Semb.)

Transplantation of the Kidney.—This operation is still in its infancy. At present it is likely to be permanently successful only if a patient with chronic progressive bilateral renal disease (e.g. chronic nephritis) is fortunate enough to have a healthy identical twin, a situation which has been taken advantage of on many occasions. Cadaver kidneys and kidneys from other donors (with a similar blood group to the patient) can be made to function for many months, providing rejection by the immunological reaction of the host is suppressed by such substances as Actinomycin D and 6-mercaptopurine. The blood-vessels of the donor kidney are anastomosed to the common iliac vessels and the ureter implanted into the bladder, so that the new kidney lies in the pelvis.

CHAPTER 46

THE URINARY BLADDER

SURGICAL ANATOMY AND PHYSIOLOGY

Surgical Anatomy.—Possessing an anterior, superior and posterior surface (fig. 1461) and being lined by transitional epithelium, the average bladder is capable of holding approximately between 400 and 500 ml. of urine without over-distension.

The greater part of the thickness of the bladder wall is made up of a muscular coat of unstriated muscle, known as the **detrusor muscle**, the fibres of which are arranged in three more or less distinct strata.

The *internal layer* is thin, and its fasciculi have a reticular¹ arrangement.

The *middle layer* is thicker than the others, and consists of densely interlaced fasciculi running mainly in a circular direction. When hypertrophy of the musculature of the bladder occurs it is the fasciculi of this layer that stand out and give rise to the characteristic trabeculation of the bladder wall (fig. 1522). While this layer is virtually absent within the limits of the trigone, its fibres are so well marked below that structure as to warrant their being accorded the dignity of the term **sphincter vesicæ**. The **trigonal muscle** is a separate entity derived from a prolongation of the longitudinal layer of each ureter. In the trigone the muscular strands from each side decussate, and eventually are inserted into the wall of the posterior urethra.

The *external layer* is composed mainly of longitudinal fibres. Some of the fibres of this layer are prolonged in the pubo-prostatic ligaments (see below).

Supports of the Bladder.—Several parts of the related pelvic fascia are described as **true ligaments of the bladder**. One of these is the rectovesical portion of the pelvic fascia, which supports the bladder posteriorly. At the base of the bladder this fascia is united closely to the muscular tunic, but it thins out rapidly as it is reflected upwards. The two lateral true ligaments of the bladder are indistinct, but the anterior, or **pubo-prostatic ligaments**, are well defined, and are of great surgical importance. Each stretches from the front of the prostate and adjacent portion of the bladder to the lower part of the periosteum of the pubis.

The urachus and obliterated hypogastric arteries, together with the folds of peritoneum overlying these structures, help to moor the bladder, and are called the **false (median and lateral umbilical) ligaments of the bladder**. Condensation of fascia around the blood-vessels passing to the bladder (fig. 1462), is known as the **superior and inferior vascular pedicles**.

Arteries.—The superior and inferior vesical arteries are derived from the anterior trunk of the internal iliac artery. Branches from the obturator and inferior gluteal arteries, and in the female from the uterine and vaginal arteries, also help to nourish the bladder.

Veins form a plexus on the antero-inferior surface of the bladder; in the male the prostatic plexus (fig. 1520) is continuous with the vesical plexus, which drains into the internal iliac vein.

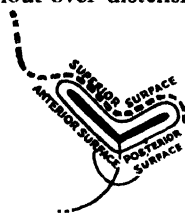


FIG. 1461.—The shape of the bladder when empty. (After F. H. Gerrish.)

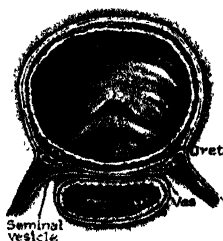


FIG. 1462.—Coronal section through the lower third of the bladder of a male, showing the inferior vascular pedicles.

¹ Reticular (Lat. *reticularis*) = resembling a net.

Lymphatics accompany the veins, and drain into the lymph nodes along the internal iliac vessels. Some lymphatics from the fundus pass to lymph nodes situated at the promontory of the sacrum.

Physiology.—Micturition is partly a reflex and partly a voluntary act. The nerves concerned in micturition are:

The sympathetic fibres, which arise in segments twelfth dorsal to third lumbar. These fibres pass via the presacral nerve and the sacral sympathetic chains to the inferior hypogastric plexus, and thence to the bladder.

The parasympathetic innervation, which is derived from the anterior primary divisions of the second, third and fourth sacral segments. These fibres pass through the pelvic splanchnic nerves to the inferior hypogastric plexus, from which they are distributed to the bladder.

The somatic innervation also comes from the second, third and fourth sacral segments as the pudendal nerves which pass, not to the bladder itself, but to the sphincter urethræ (striated muscle).

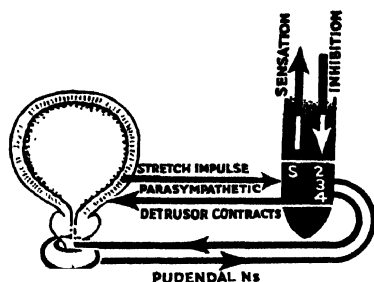


FIG. 1463.—The nervous control of the bladder. The sympathetic innervation has been omitted for the sake of simplification.

While the sympathetic nerves convey afferent painful stimuli of over-distension from the bladder to the brain, it is unlikely that either set of autonomic nerves convey to the bladder any cortical impulses, such impulses being transmitted via the pudendal nerves to the sphincter urethræ, relaxing the latter. At the same time stretching of the muscle fibres by distension of the bladder initiates a reflex which is mediated through the parasympathetic nerves, and the detrusor muscle contracts. The neurogenic theory therefore ascribes the act of micturition to a stretch reflex which, if the time or place are not propitious, can be inhibited by cerebral control (fig. 1463).

ECTOPIA VESICÆ (syn. EXSTROPHY OF THE BLADDER)

Ætiology.—Incomplete development of the infra-umbilical part of the anterior abdominal wall, associated with incomplete development of the anterior wall of the bladder, is the embryological origin of the condition.

Clinical Features.—Ectopia vesicæ occurs once in 50,000 births, and may be complete or incomplete. In the more common complete variety, because of the pressure of the viscera behind it, the deep-red posterior bladder wall protrudes through the defect (fig. 1464). If the exposed mucous membrane, which bleeds readily, is drawn gently upwards, the paler, wet trigone is displayed, and effluxes of urine from the ureteric orifices can be seen. There is a well-defined line of demarcation between the protruding mucous membrane and the adjacent skin, and, especially after reducing the extruded bladder wall beneath the mucocutaneous junction, the firm edge of the hernial ring can be felt. Usually the umbilicus is absent. There may be umbilical and inguinal hernias. The rectal sphincter is often lax. The sex ratio is about four males to one female.

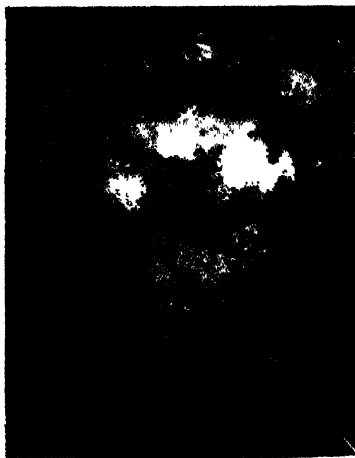


FIG. 1464.—Ectopia vesicæ in a male. The markers are in the ureteric orifices. (G. D. Adhia, F.R.C.S., Bombay.)

In the male, the completely epispadiac penis is broader and shorter than normal, and not infrequently bilateral inguinal herniæ are present; the prostate and seminal vesicles are rudimentary or absent, whereas the testes are normal and descend more often than not. *In the female*, the clitoris is cleft and the labia minora are separated anteriorly, exposing the vaginal orifice. In both sexes there is separation of the pubic bones (fig. 1465), which are connected by a strong ligament, and, except for the fact that the patient has a waddling gait, this bony defect causes no disability; indeed, in those female sufferers who become pregnant, it facilitates delivery. The linea alba is as wide as the separation of the pubic bones. In the rare incomplete form the pubes are united and the external genitalia are more normal.

The lot of a patient with ectopia vesicæ is particularly unfortunate. A portable urinal seldom keeps him dry for long, and the odour of urine always accompanies him. Frequently the extroverted vesical mucous membrane becomes ulcerated and painful. It undergoes metaplastic changes, and sometimes becomes the seat of an adenocarcinoma. Attempts to prevent recurrent attacks of ascending pyelonephritis by chemotherapeutic and antibiotic agents are only partially successful; 50 per cent. of those afflicted die of renal failure before the age of thirty.



FIG. 1465.—Showing the separation of the pubes in a case of ectopia vesicæ. (The late Professor Grey Turner, London.)

Treatment

(a) *Diversion of urine* (p. 1156), into the colon, followed some months later by cystectomy. The operation is undertaken between four and six years of age.

(b) The best long term results would seem to follow diversion into an *ileal conduit* (p. 1159), *with excision of the bladder*, at the age mentioned. Renal function is likely to be better maintained than following uretero-colic anastomosis, and there are no problems of rectal control (see clinical features).

(c) *Iliac Osteotomy and closure of abdominal wall*.—In recent years, attempts to reconstruct the bladder and sphincters by operation within the first year of life have shown that it is possible to obtain a sound mid-line abdominal scar by commencing the operation with osteotomy of both iliac bones just lateral to the sacro-iliac joints. The bladder can be closed, and the reconstructed urethra displaced behind the pubis, which now meets in the mid-line. However, the ultimate results in respect of continence, freedom from urinary infection, and stone formation are disappointing, though in males the procedure may be a useful method of keeping the patient dry with an appliance. It is better, if this is the intention, to make no attempt to produce continence at the bladder neck.

(d) *Lowsley's operation* (p. 1159) may be of value if the anal sphincter is normal, but it does result in a lower bowel devoid of normal afferent sensation.

Results.—Comparatively few who have undergone successful uretero-sigmoidal implantation for ectopia vesicæ live for twenty years. The causes of the untimely demise are (1) stricture at the site of anastomosis and bilateral hydronephroses, (2) recurrent pyelonephritis and (3) hyperchloræmic acidosis (p. 1158). Many different methods of implanting the ureters have been devised (fig. 1515); all aim at reducing the risks of stenosis and reflux.

RUPTURE OF THE BLADDER < Extraperitoneal 80 per cent.
Intraperitoneal 20 per cent.

The past fifty years have witnessed a complete reversal in the ratio of intra-peritoneal to extraperitoneal rupture of the bladder, brought about by (a) a reduction of intraperitoneal rupture due to greater national sobriety (an inebriated person is likely to become embroiled in a quarrel and receive a blow on the abdomen, or fall on some hard object, whilst his bladder is over-distended), and (b) by the increased frequency of traffic accidents with their toll of fractured pelvises.

Intraperitoneal rupture of the bladder is seventy times more common in males than in females. This remarkable discrepancy can be accounted for partly by the more commodious pelvis of the female, and less inebriety.

A rare type of accident causing rupture of the bladder, peculiar to the female, is pillion riding, or falling in a squatting position on to a projecting object, the anterior vaginal wall receiving the brunt of the violence.

Extraperitoneal Rupture.—Ten per cent. of fractured pelves are complicated by a ruptured bladder. A lateral crushing injury resulting in severe disruption of the architecture of the true pelvis and tearing of the bladder from its ligamentous moorings, is the usual cause. A bladder containing even a small quantity of urine is not immune to such violence, but a full one is extremely vulnerable.

After the initial shock has passed off, one of the first symptoms is an intense desire to micturate, but either no urine is passed or only a few drops of blood-stained urine with great effort. The spasms recur at intervals. Extravasation occurs into the prevesical space, causing a tender swelling above the pubis (fig. 1466); later, the extravasated urine passes up the anterior abdominal wall between the fascia transversalis and the peritoneum, causing

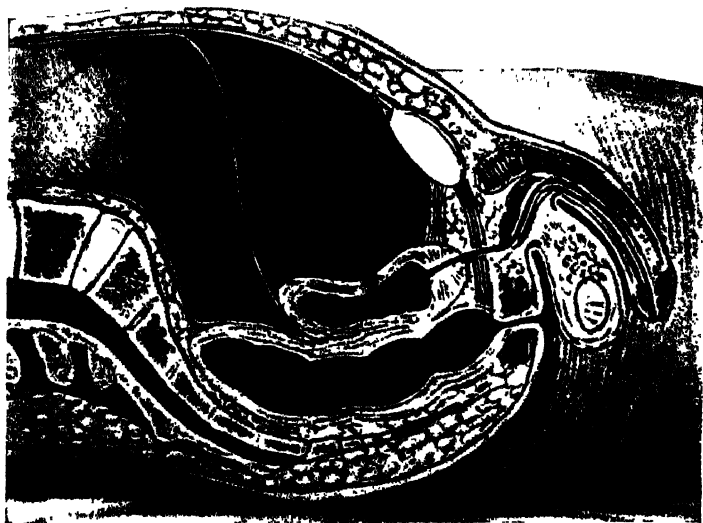


FIG. 1466.—Extraperitoneal rupture of the bladder.

necrosis of the intervening connective tissue. In the male, extraperitoneal rupture of the bladder can be distinguished from intrapelvic rupture of the urethra, for in the latter the prostate is impalpable or felt with difficulty (fig. 1558).

About 35 per cent. of patients with a fractured pelvis, *without rupture of the bladder or urethra*, suffer from retention of urine, and 30 per cent. pass urine containing blood, due to bruising of the bladder wall or to tearing of its mucous membrane.

Intraperitoneal Rupture.—There is sudden, agonising pain in the hypogastrium, often accompanied by severe shock and perhaps syncope. However, especially if the patient was 'drunk and distended', in a few minutes

the shock subsides and the pain lessens, so that the patient may continue with his orgies, but the abdomen commences to distend. Usually the patient has no desire to micturate. On examination varying degrees of abdominal rigidity and, after a few hours, abdominal distension are present. In spite of the fact that the patient has not passed urine since the accident, there is no dullness above the pubes corresponding to a distended bladder. Usually there is tenderness in the hypogastrium. If the amount of urine in the peritoneal cavity is considerable, shifting dullness can be elicited. Rectal examination often reveals a bulging in the rectovesical pouch. When the urine is sterile, symptoms and signs of peritonitis are delayed for hours.

Confirming a Suspected Diagnosis.—*Retrograde cystography* is the only reliable method of proving the integrity or otherwise of the bladder. It should be carried out at the same time that radiographs of the bony pelvis are taken. A catheter is passed with full aseptic precautions. This may recover a small amount of blood-stained urine, which supports the diagnosis. 120 ml. Sodium Chloride Injection B.P. with 40 ml. 35 per cent. Hypaque (diatrizoate) are injected with a bladder syringe. Particularly characteristic of extraperitoneal rupture is the 'tear-drop' bladder (fig. 1468) due to its elevation by extraperitoneal extravasation of blood and urine.



FIG. 1468.—The 'tear-drop' bladder of extraperitoneal rupture. (Dr. J. E. Kicklighter, Sarasota, Florida.)

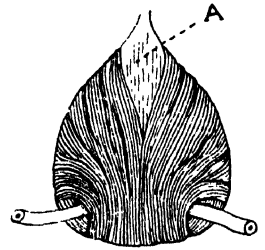


FIG. 1467.—Intraperitoneal rupture occurs within the area A. The tear is nearly always longitudinal.

Treatment.—When there are reasonable grounds for suspecting rupture of the bladder, operation should be undertaken without delay.

Intraperitoneal Rupture.—Lower laparotomy is performed. Urine is removed by suction, after which the patient is placed in Trendelenburg's position. The edges of the rent, which is usually situated in the posterior part of the dome of the bladder, are trimmed and sutured by two layers of interrupted catgut stitches, and the operation completed by stitching a large Malécot catheter into an extraperitoneal suprapubic incision in the bladder. The latter ensures intravesical tension being kept at a minimum during healing of the sutured tear. Unless obvious infection is present the peritoneum can be closed without drainage. The abdominal wall is repaired around the Malécot catheter, not omitting drainage of the prevesical space. Following the operation, antibiotic therapy is given until the urine is proved sterile after removal of the catheter in 8–10 days.

Extraperitoneal Rupture.—Suprapubic cystostomy is performed, and the prevesical space is drained. It is unnecessary to attempt to suture the tear, which from its situation at the base of the bladder may prove difficult, and which in any case will heal readily.

Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Leipzig. In 1839 Dr. Walther, a General Practitioner of Pittsburg, successfully sutured an intraperitoneal rupture of the bladder of a blacksmith.
Achille-Etienne Malécot, 1852–?. He invented his catheter while Interne des Hôpitaux de Paris.

Prognosis.—When operation is performed within twelve hours the mortality is approximately 11 per cent. ; when operation is delayed to twenty-four hours the mortality rises to 55 per cent. As in the days of ancient Greece, when the condition was regarded as inevitably fatal, without operation the mortality is 100 per cent.

WOUNDING OF THE BLADDER DURING OPERATION

Operations in which the bladder is liable to be injured are (1) inguinal or femoral herniotomy (especially during the performance of the low operation for strangulated femoral hernia); (2) hysterectomy (particularly panhysterectomy by either the abdominal or vaginal route) and (3) excision of the rectum. In all these operations, to minimise this accident the bladder must be emptied *after* the patient has been anaesthetised. If the injury is recognised at the time of its infliction, the bladder must be repaired in two layers and urethral catheter drainage maintained for seven days. If it is not so recognised, the treatment is similar to that of rupture of the bladder.

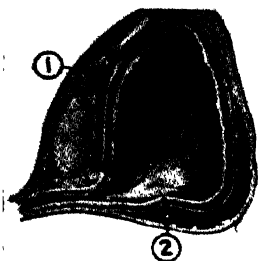


FIG. 1469.—Perforation by a diathermy cutting loop (see text).

When accidental perforation of the bladder occurs during endoscopic resection of a bladder tumour, or the prostatic capsule is perforated during transurethral prostatectomy, for practical purposes the perforation is always extraperitoneal. When the accident is recognised at the time, drainage of the bladder with a large urethral catheter, and the administration of antibiotics, usually suffice. If the accident is not recognised at the time, suprapubic

drainage of the prevesical space in the case of an anterior perforation (fig. 1469 (1)), or perineal drainage of the pararectal space for a posterior perforation (fig. 1469 (2)), is urgent and imperative.

RETENTION OF URINE

Retention of the urine is either acute or chronic, the latter leading ultimately to retention-with-overflow.

Acute retention is often acute-on-chronic retention, for, except in post-operative and traumatic cases, it seldom occurs unheralded.

Ætiology.—The condition is comparatively rare in women and children, and the most frequent causes are :

<i>In the male</i>	<i>In the female</i>	<i>In the male child</i>
Prostatic enlargement.	Retroverted gravid uterus.	Meatal ulcer with scabbing.
Urethral stricture.		
Post-operative retention.	Disseminated sclerosis.	
	Hysteria.	

Other causes .

Following spinal anæsthesia.
Blood-clot in the bladder.
Rupture of the urethra (p. 1186).
Neurogenic (injury or disease of the spinal cord).
Fæcal impaction in the rectum.

Acute urethritis or prostatitis.
Urethral calculus (p. 1199).
Ring around the penis (p. 1205).
Phimosis.
Certain drugs (p. 1129).
Muscular atony from advanced age.

Clinical Features.—The patient has not passed urine for some hours, and is unable to do so. The swelling caused by a full bladder often can be

seen in a thin person; it is somewhat tender to palpation, and dull to percussion above the symphysis pubis. Spasms of acute pain occur periodically as the muscle of the bladder contracts. An attempt is made to elicit the cause of the retention; this is most frequently due to vascular engorgement of an already enlarged prostate (p. 1163), but the floor of the urethra should be palpated for the induration characteristic of a stricture, and the reflexes in the lower limb and perianal sensation, tested.

Treatment.—It is of paramount importance never to relieve, or attempt to relieve, acute retention and forthwith send the patient home. He must always be confined to bed immediately, and kept there for at least twenty-four hours after relief has been obtained. The blood urea must be estimated. Once the patient is in suitable surroundings, the effect of administering a dose of Omnopon to relieve anxiety, and a hot bath to diminish internal congestion, is tried. In many cases the patient is able to pass urine into the bath. If this fails, catheterisation is attempted. This should always be performed with full aseptic ritual (p. 1166). If the blood-urea level is raised, the catheter will have to be left indwelling, and a pubic shave before catheterisation greatly facilitates ease and comfort in retaining the catheter in position with strapping. A small (12F) pre-sterilised plastic catheter (Jaques), is employed; if this is unsuccessful, a Tiemann's hard rubber catheter (fig. 1470) should be tried. If the catheter is to be left indwelling, a Gibbon catheter (fig. 1477), or a small Foley catheter (fig. 1471) should be used. This often succeeds in passing the obstruction. Should this catheter not be available, or fail, and



FIG. 1470.—Tiemann's catheter.

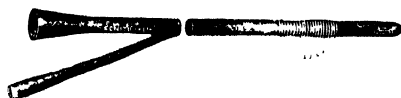


FIG. 1471.—Foley's catheter.

the case is one of a suspected enlarged prostate, a large bi-coudé¹ gum-elastic catheter (fig. 1472) is tried. On the other hand, when the case is one of urethral stricture, a gum-elastic olivary catheter (fig. 1473) is selected. If the catheter passes the obstruction, the bladder can be emptied completely.



FIG. 1472.—Bi-coudé catheter.

(The bevel indicates the direction of the bend at the opposite end of the catheter.)

FIG. 1473.—Olivary catheter.

All catheters (and gum-elastic bougies) are calibrated in the French scale (F.),² and as the narrowest part of the urethra is the external urinary meatus, unless there is reason to believe that the urethra is strictured, it is usual to choose a catheter with a diameter a little less than that of the orifice, viz. —→

● 10F. ● 16F.
● 12F. ● 18F.

If, after a reasonable attempt with catheters, the bladder has not been entered, one of four courses may be adopted, according to circumstances.

¹ coudé (adjective) = bent; coude (noun) = elbow.

² The French scale is also known as Charrière's scale, so called after Joseph Charrière (1803-1876), a Paris surgical-instrument maker.

1. **Suprapubic Puncture.**—Suprapubic puncture with a lumbar puncture needle or an 'intracath' (p. 90) is a useful method of relieving acute retention when catheterisation has failed. If the bladder is allowed to refill after it has been punctured, leakage into the prevesical space may follow.

2. **Suprapubic Catheterisation (Riches' Technique).**—A catheter is inserted into the bladder suprapubically through a $\frac{1}{4}$ -inch (1.25 cm.) incision made under local anaesthesia 1 inch (2.5 cm.) below the level at which the anterior surface of the bladder curves upwards and backwards to form the dome. The fibres of the

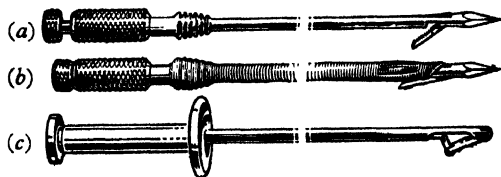


FIG. 1474.—(a) Riches' suprapubic catheter introducer; (b) the catheter stretched on the introducer; (c) the advancer.

the linea alba are divided in the length of the incision. After the special catheter has been mounted on its introducer (fig. 1474 (a), (b)) it is passed through the incision until its sharp point touches the surface of the bladder, and the instrument is passed into the bladder with a short, sharp thrust directed backwards and downwards. The introducer is removed and the advancer (fig. 1474 (c)) is

passed down the lumen of the catheter and its expander is opened. Thus the catheter is carried towards the trigone, and the advancer is removed. A stitch secures the catheter to the skin, and this is reinforced with a strip of adhesive plaster.

3. **Immediate Prostatectomy.**—Possibly immediate prostatectomy in cases of benign enlargement of the prostate in relatively fit patients.

4. **Urethral instrumentation** (p. 1194), in the case of a urethral stricture.

CHRONIC RETENTION

In such cases the distension of the bladder is painless. The blood urea must be estimated before any attempt is made to relieve the retention. If it is below 70 mg. per cent, treatment is as outlined for acute retention. Above this figure slow decompression is advisable (p. 1166).

RETENTION WITH OVERFLOW

In this condition the patient has no control of his urine, small amounts passing involuntarily from time to time, overflowing from a bladder which is distended to its maximum capacity. It may follow a neglected acute retention, or chronic retention.

Retention with overflow is referred to also under the headings of 'false incontinence' (p. 1131) and 'prostatic enlargement' (p. 1164).

The general principles which govern the treatment of this condition are similar to those of acute retention, but decompression of the bladder must be carried out very slowly (p. 1166).

THE INDWELLING CATHETER AND THE CLOSED SYSTEM OF CATHETER DRAINAGE

The incidence of ascending infection is nearly halved by connecting the catheter (urethral, suprapubic or perineal) to sterile tubing conducted to a sterile collecting bottle (fig. 1475), and employing irrigations only if clot retention occurs. When a catheter has been *in situ* for five or more days, some

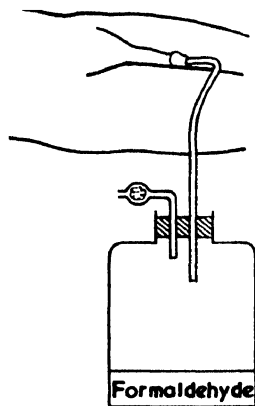


FIG. 1475.—The essentials of continuous bladder drainage. The indwelling catheter has been introduced, using aseptic and antiseptic precautions (p. 1166). The sterile connecting tubing leads to the collecting bottle. This bottle should contain a suitable disinfectant, e.g. formaldehyde to keep the contents sterile. Alternatively, a closed sterile plastic container fitted with a non-return valve may be used.

degree of urethritis is likely to supervene, though this has been much reduced by the use of catheters made from various non-irritant plastic substitutes for rubber. To change the catheter in the presence of urethritis entails a grave risk of infection from the anterior to the posterior urethra, and thence to other parts of the urogenital system (fig. 1476).

SPECIAL FORMS OF RETENTION OF URINE

Post-operative Retention of Urine.—Retention of urine can be encountered after any operation. It is common after operations on the anal canal and perineal region, and is due to reflex spasm of the urinary sphincters. After operations on the pelvic viscera, retention of urine is so common that it is usual to forestall it by inserting a catheter before or at the conclusion of the operation.

When the patient is an elderly male, prostatic obstruction, hitherto latent, should be suspected. Many patients cannot urinate while lying or sitting in bed. Another extremely common cause is sedation: when a sufficient quantity of an analgesic drug to relieve pain is administered, frequently the desire to micturate is suppressed until the sympathetic nerve fibres are stimulated from overdistension of the organ.

Treatment.—If the male patient, while supported, is permitted to sit on the edge of his bed, he is often able to empty his bladder. The sound of running water is often helpful. Parasympathetic stimulating drugs, e.g. Carbachol, may be prescribed, but they should be avoided after operations on the gastrointestinal tract. If after a reasonable trial the patient cannot pass urine, he or she must be catheterised.

Acute Retention due to Drugs other than Sedatives.—A number of the newer drugs are prone to induce or precipitate retention of urine. Propantheline bromide, used to decrease secretion and motility in peptic ulcer, antihistamine drugs, antihypertensive drugs, anticholinergic drugs and I.N.A.H. compounds (chemotherapeutic agents for tuberculosis) have all been responsible for producing acute retention of urine. Indeed, 'drug retention' has become a clinical entity.

Retention of Urine due to Lesion of the Spinal Cord.—The bladder, the nervous control of which is disrupted because of a complete or incomplete lesion of the spinal cord, passes through various stages of dysfunction (p. 408).

The Atonic Neurogenic Bladder.—Immediately following the injury the detrusor muscle becomes paralysed, the sphincter vesicæ contracted tightly and the sphincter urethræ relaxed. The only factor permitting any evacuation of the bladder is the elasticity of its walls. In lesions situated above S.2 (fig. 1463) to below D.4–6, because the sympathetic innervation of the bladder is intact, the patient can appreciate when the bladder becomes filled to capacity. If allowed to do so, the bladder becomes greatly distended and paralytic overflow incontinence ensues. This phase (due to spinal shock)

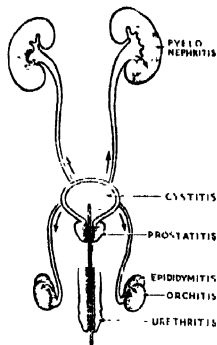


FIG. 1476.—Complications liable to follow changing a urethral catheter in the presence of urethritis. (After C. G. Scorer.)

lasts for days, weeks or even months. As recovery from spinal shock takes place, depending on the level of the lesion, *automaticity* or *autonomy* of bladder function occurs, but before it does so it passes through two intermediate phases.

(a) *Phase of Ineffectual Emptying Contractions*.—Contractions of the detrusor muscle occur, but because the sphincter vesicæ remains in spasm, small quantities only of urine are voided frequently. This phase is of a few hours' duration.

(b) *Phase of Hypertonicity*.—The detrusor passes into spasm. The sphincter vesicæ relaxes physiologically and the sphincter urethræ reflexly.

The Automatic (syn. Reflex) Bladder.—When the lesion is situated above the centre of micturition the bladder empties reflexly every one to four hours, the amount of residual urine varying from case to case. When the filling reaches a certain point the detrusor muscle contracts reflexly—a condition similar to that seen in early infancy.

The Autonomous Bladder results when a lesion is situated at a level that will destroy the centre for micturition (fig. 1463), or a lesion in the cauda equina. Emptying of the bladder is dependent upon the precarious and inefficient control by the nerve plexus situated between the muscular strata of the bladder wall. Continual dribbling is the outcome, but the bladder can be emptied by manual compression. Residual urine, however, is never absent and dilatation of the ureters and, eventually, of the renal pelves and calyces results.



FIG. 1477.—Portex Gibbon catheter in place.

Treatment.—The most practical solution of the problem of how best to relieve retention of urine during the vigil of the period of atonicity, is to pass, with the strictest aseptic precautions, a catheter of Portex tubing 1.5 mm. in diameter which is so designed that it can be retained by its wings in both the male (fig. 1477) and in the female (Gibbon). It is advisable to introduce the tubing at the earliest possible time, as there is evidence that over-distension prejudices the eventual

development of detrusor contractions (Ross). Especially important is early drainage in the female, for otherwise wet beds favour the development of bedsores. Should the tubing become blocked with mucus, it can usually be freed by suction applied with a needle and syringe to its distal end. A high fluid intake (6 pints (3 litres) in the twenty-four hours) must be insisted upon. Usually chemotherapeutic and antibiotic agents are held in reserve in case infection supervenes.

The state of automaticity is heralded by return of bulbo-cavernosus reflexes (erections) and anal reflexes, and should the catheter tubing become blocked, urine will be passed alongside the catheter.

With the return of detrusor activity *bladder training* is commenced. At first the catheter is clipped off and released at regular, but lengthening, in-

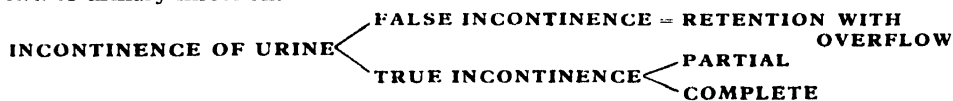
tervals. Later the catheter is removed, and reflexes are initiated by the patient lifting himself in his chair or bed, again at regular intervals by the clock. Drinks are given during the day, but none after seven p.m. (19.00 hrs.).

Enhancing or Initiating Automaticity in Refractory Cases.—Cysto-urethrography is invaluable in determining (1) spasm of the sphincter vesicæ, (2) contracture of the prostatic urethra, (3) prostatic shelf obstruction and (4) spasm of the sphincter urethræ.

Transurethral resection of narrow strips of the vesical neck and prostatic urethra between five and seven o'clock is often very successful in types of obstruction 1, 2 and 3. Pudendal neurectomy sometimes abolishes spasm of the sphincter urethræ. For this operation the patient is placed in the jack-knife position (p. 984) and the pudendal nerves are exposed as they leave Alcock's canal and resected. In the few cases that do not respond to pudendal neurectomy, endoscopic resection of a portion of the sphincter urethræ is likely to succeed.

The State of Autonomy.—Manual expression of urine by the patient, if he has the use of his arms, plus contracture of the abdominal muscles, if they are not paralysed, is the only (but often tolerably successful) method of avoiding life-long urethral or suprapubic catheter drainage.

Persistent Retention of Urine following Excision of the Rectum.—In 10 per cent. of cases retention of urine persists for three months or more. Probably, owing to the absence of cystometric substantiation of damage to the parasympathetic nerve supply to the bladder, the retention is due to mechanical factors, notably sagging of the bladder into the space formerly occupied by the rectum. In most cases the retention can be cured by transurethral resection of the prostate. Usually the prostate is not enlarged, and consequently the resection can be carried out easily and quickly. Therefore, if after two weeks the patient is unable to empty his bladder and his general condition is satisfactory, transurethral prostatic resection should not be postponed. Thus the patient is spared what would probably be a long period of invalidism and the risk of urinary infection.



False incontinence has been dealt with already (p. 1128).

Partial Incontinence.—The patient is unable to exercise full control, and loses urine without warning. There are two main varieties:

1. **Stress incontinence** is not uncommon in women. The pubococcygeal muscle plays an important part in maintaining control of urination in women. Weakness of that muscle due to overstretching or partial tearing of it during labour is the principal cause of stress incontinence, but weakness of the muscles of the pelvic floor in general, and of this muscle in particular, sometimes occurs in nulliparæ. Expulsive acts, such as coughing or sneezing, may be sufficient to cause a leakage of urine.

Treatment.—In the absence of an obvious cause requiring operative treatment on its own merits, e.g. fibromyomas, prolapse of the uterus, *non-operative treatment* can be recommended, and if carried out conscientiously for a sufficient length of time, the results are often extremely good. The treatment consists of voluntary exercises (drawing-in of the perineum) to tighten the rectal and vaginal sphincters carried out a dozen times three times a day, regardless of where the patient is, or what she is doing. Faradism to the pelvic musculature is also beneficial.

Operative Treatment.—In patients with considerable bulging of the anterior vaginal wall (cystocele), anterior colporrhaphy frequently improves continence. In the absence of a cystocele an operation to rectify sagging of the bladder neck can be undertaken in cases in which there is little or no improvement after a course of exercises.

Everard Williams' operation for stress incontinence is simple, and gives as good results as more complicated procedures. A Foley's catheter having been introduced into the bladder, the bladder neck and adjoining portion of the urethra are exposed via the retropubic route. The lower part of the bladder and the intrapelvic portion of the urethra are mobilised, but there is no necessity to separate the urethra from the vagina. Three chromic catgut sutures are introduced on either side, each traversing the wall of the urethra or that of the bladder neck, being careful not to penetrate the mucous membrane. The sutures are then passed through the fascia covering the deep surface of the pubic bones near the symphysis. When the sutures are tied they suspend the bladder neck and the proximal part of the urethra.

2. Enuresis in Children.—Nocturnal enuresis is usually a continuance of infantile bed-wetting. Occasionally it commences months or years after voluntary micturition has been established. In 4 per cent. of cases involuntary micturition is only diurnal; in 12 per cent. of cases it is both nocturnal and diurnal. In all patients who have reached the age of four or over, a cause for the involuntary micturition should be sought:

1. The urine should be examined for pus and organisms.
2. Phimosis or atresia meati should be corrected; likewise vulvitis or vaginitis.
3. Radiography and excretory pyelography may reveal a hydronephrosis, an ectopic ureteric orifice or a urinary calculus. Voiding cysto-urethrography is invaluable for detecting urethral valves (p. 1184).
4. Urethroscopy sometimes shows inflammation of the verumontanum, in which event periodic dilatation with a urethral bougie is sometimes beneficial.
5. Thread worms should be eliminated.
6. Spina bifida and epilepsy can cause enuresis (p. 411).

In the great majority (over 90 per cent.) no cause for the enuresis is discovered, in which event the condition is described as primary enuresis.

Treatment.—Many forms of treatment have been advocated, and the literature on this subject has become voluminous. The results of treatment are difficult to assess, as the majority of sufferers gain control at or before puberty. One routine is recommended here:—The importance of *not* relieving the bladder immediately during the day-time is explained to the child as soon as he or she is old enough to understand that an effort must be made to increase the time between the urge to micturate and voiding. The objective is to increase the bladder capacity by such training. Belladonna-containing medicines (e.g. Bellergal) and ephedrine in small doses are given at bed-time to prevent deep sleep; this can be continued for several weeks without harmful effect, by which time many patients have learned to arise when the bladder is full. In refractory cases the advice of a children's psychologist should be sought.

Complete Incontinence.—The urine dribbles away without fully distending the bladder. This is the result of either extensive damage to the sphincter urethræ, or a central nervous lesion affecting its nerve supply (the pudendal nerves). Rupture of the urethra, difficult labour, perineal prostatectomy, and also, but less frequently, retropubic prostatectomy and endoscopic resection, account for a certain number of cases. The remainder are due to organic spinal or cerebral disease, which include in early life some form

of spina bifida, in middle life disseminated sclerosis or tabes dorsalis, and late in life a cerebrovascular accident.

Appliances for the Relief of Incontinence.—In the female it is impossible to fit any apparatus that will keep the patient dry. An indwelling Foley catheter (fig. 1471) is the simplest solution. Diversion of urine (p. 1156) is the alternative. In

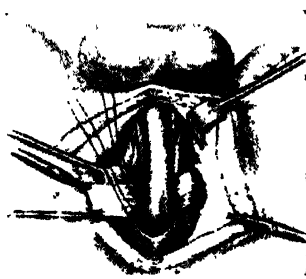


FIG. 1479.—Transplantation of the gracilis muscle to make a new sphincter urethrae.

the male the choice lies between a dribble-bag and an incontinence clamp (fig. 1478). Should these prove unacceptable, an attempt may be made to plect the bulbo-cavernosus muscle with silk sutures (Millin), or to pass a sling of gracilis muscle around the bulb (fig. 1479).

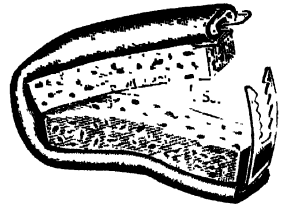


FIG. 1478.—Cunningham's penile clamp.

Ileo-ureterostomy (p. 1159).—The collection of urine from an artificial bladder into an ileostomy bag (especially if a plastic operation has been performed on the ileostomy opening so as to render it spout-like and covered with skin) is so much more water-tight than a portable urinal worn over the genitalia, and the incidence of pyelonephritis is so much less, that the substitution of an artificial for a normal non-functioning bladder is a change for the better, not only for the patient but for those with whom he or she lives (the uriniferous odour is reduced, sometimes to vanishing point). The operation is particularly successful in cases of incontinence due to spina bifida (Wells).

VESICAL CALCULUS

Definition.—A *primary vesical calculus* is one that develops in sterile urine; it often, but not necessarily, originates in a kidney and passes down the ureter to the bladder, where it enlarges.

A *secondary vesical calculus* occurs in the presence of infection.

A vesical calculus can occur also by the deposition of urinary salts upon a foreign body in the bladder.

Incidence.—Until the twentieth century vesical calculus was one of the most prevalent disorders among the working classes, and the incidence was especially high in childhood and adolescence. Owing to improved diet, especially an increased protein-carbohydrate ratio, primary vesical calculus is comparatively rarely encountered. The most remarkable fall in incidence is among children.



FIG. 1480.—'Jack-stone' ¹ calculus. This type of vesical calculus is typically found in a post-prostatic pouch.

Composition and Cystoscopic Appearance.—Most vesical calculi are composite, but have one component in excess, and assume the appearance of that variety (see also p. 1089).

1. *Oxalate calculus* is a primary calculus that grows slowly. Usually it is of moderate size, and is solitary. Its surface is uneven (mulberry type fig. 1482); sometimes it bristles with spines (fig. 1480). Although calcium

¹ 'Jack-stone' = the kernel of the fruit of the jack tree. These fruit stones are used for a game played by children in the U.S.A.

Terence John Millin, *Contemporary*. Honorary Consulting Surgeon, All Saints' Hospital, London.
Charles Alexander Wells, *Contemporary*. Professor Emeritus of Surgery, Liverpool.

oxalate is white, the stone is usually dark brown or black because of deposits of blood pigment upon it.

2. *Uric acid and urate calculi* are round or oval, fairly smooth, and vary in colour from pale yellow to light brown: they may be single or multiple. These stones also are primary: they are not opaque to X-rays.

3. *Cystine calculus* occurs only in the presence of cystinuria (p. 1090), and is radio-opaque due to its high sulphur content.

4. *Phosphatic calculus* is composed of triple phosphates and occurs in urine infected with urea-splitting organisms. It tends to grow rapidly. In some instances it occurs on a nucleus of one of the foregoing types of calculus; much more rarely on a foreign body. In others the nucleus is composed of desquamated epithelium and bacteria. It is dirty white in colour, and of chalky consistency.

A vesical calculus is usually free to move in the bladder. It gravitates to the lowest part of the organ, which, when the patient is erect or sitting, is the bladder outlet. In the recumbent position (as also at cystoscopy) the stone occupies a position behind the inter-ureteric ridge. Less commonly the stone is wholly or partially in a diverticulum or a post-prostatic pouch, and in either case the stone may be partially or completely hidden from view.

Clinical Features.—Males are eight times more often affected than females.

(a) *Latent.*—When a stone is situated in a post-prostatic pouch or a diverticulum of the bladder, it is usually discovered unexpectedly at cystoscopy or on X-ray examination.

(b) *Typical.*—*Frequency* is the earliest symptom. Unlike other forms of frequent micturition, it is not much in evidence during the night. After micturition the patient does not feel satisfied that the bladder is empty.

Pain is most in evidence in cases of spiculated oxalate calculus. It occurs at the end of micturition, and is usually referred to the tip of the penis or to the labia majora; more rarely to the perineum or suprapubic region. Pain and discomfort are much in evidence during exertion, and are aggravated by jolting movements (e.g. riding in a vehicle). If the patient lies down, the symptoms tend to pass off because the stone falls away from the sensitive portion of the bladder, the trigone. Thus he usually sleeps peacefully through the night. In young boys, screaming and pulling at the penis with the hand at the end of micturition are indicative of vesical calculus.

Hæmaturia is characterised by the passage of a few drops of bright red blood at the end of micturition, and is due to the stone abrading the vascular trigone—a fact that also accounts for the pain.

Interruption of the urinary stream by the stone blocking the internal meatus, occurs occasionally, and may be remedied by a change of posture.

Acute retention of urine from a vesical calculus is extremely uncommon in adults, but not so in children.

(c) *Masked.*—The symptoms of a concomitant persistent cystitis may overshadow those that might be occasioned by the stone.

Rectal or vaginal examination, when accompanied by abdominal palpation, occasionally enables a vesical calculus to be felt. Unless the stone is large, rectal examination is negative in the adult male, but in a female or child a calculus of moderate size may be palpable.

Examination of the urine will reveal blood, and possibly pus or crystals typical of the calculus, e.g. envelope-like in the case of an oxalate stone, or hexagonal plates with cystine calculi.



FIG. 1481. — Radiograph showing a vesical calculus.

Radiography.—In at least 92 per cent. of cases vesical calculus can be demonstrated on an X-ray film (fig. 1481). If the stone is not opaque, a filling defect may be visualised. Radiographs of the whole of the urinary tract should be taken, after which excretory pyelography is carried out; the former will reveal or disprove the presence of opaque renal or ureteric calculi, while the latter will help to determine the normality or otherwise of the kidneys.

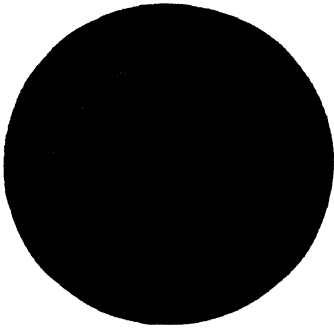


FIG. 1482. —A 'mulberry' oxalate stone in the bladder as viewed by a cystoscope.

Cystoscopy is essential for evaluation of the case. Frequently, on introducing the sheath of the cystoscope, a significant 'click' will be felt when a free-lying stone comes in contact with the instrument. As described already, cystoscopy usually determines the composition of the calculus (fig. 1482). Stones non-opaque to X-rays, e.g. uric acid, can be seen.

In all cases the whole of the bladder wall is inspected: in primary calculus aseptic cystitis is basal; in secondary calculus bacterial cystitis is universal. In appropriate cases the exit of the bladder is examined for prostatic enlargement or contracture of the bladder neck.

Treatment:

Litholapaxy.—In most cases crushing the stone with a lithotrite is highly satisfactory. However, the contraindications to the method are as follows:

- | | | |
|--|---------|---|
| Contra-indications to Litholapaxy | Urethra | Prostatic obstruction. |
| | | A urethral stricture that cannot be dilated sufficiently. |
| | | When the patient is below ten years of age. |
| | Bladder | Vesical diverticulum. |
| | | Cystitis that fails to respond to treatment. |
| | | Contracted bladder. |
| | | Other conditions requiring operation, e.g. enlarged prostate, new growth. |
| | | |
| | 'Stone | A very large stone. |
| | | A very small stone, which should pass naturally. |
| Too hard to crush—an oxalate calculus. | | |
| Too soft to crush—a cystine calculus. | | |
| A stone encrusting a foreign body. | | |

Technique.—For several days before the operation a suitable drug is administered to reduce infection. While blind litholapaxy is preferred by those who have had much experience with the solid lithotrite, its only advantage is that, by reason of its solidity and greater strength, harder stones can be crushed than is the case with an instrument that contains a light and telescope incorporated in its shaft. Canny



FIG. 1483.—A cystoscopic lithotrite.

Ryall's cystoscopic lithotrite enables the stone, and such fragments as are necessary, to be seized under vision. The instrument, with its obturator in place, is introduced into the bladder, and rotated so that its closed jaws point downwards. The obturator is removed and the bladder is irrigated with boric lotion by means of two 6-ounce (200-ml.) syringes until the lotion is returned clear. The bladder is then filled with not more than 10 ounces (300 ml.) of the solution, and after inserting the telescope, the stone is seen. The screw on the handle of the instrument is turned, and the jaws thereby opened. The distal blade is hooked over the centre of the stone (fig. 1483) and by rotating the screw

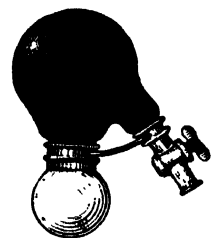


FIG. 1485.—Modified Bigelow's evacuator.



FIG. 1484.—An evacuating cannula.

handle the proximal movable blade is advanced so that the stone is grasped firmly. The ocular end of the lithotrite is depressed, thereby lifting the calculus away from the bladder wall. After withdrawing the telescope slightly, to prevent damage to the light bulb, the screw is turned slowly, breaking the stone. Large fragments are crushed into small ones by repeating the manœuvre. With the jaws closed, the lithotrite is rotated so that the jaws point upwards, and after removing the telescope and allowing the lotion to escape, the instrument is withdrawn.

Evacuation of the Fragments.—An evacuating cannula (fig. 1484), the largest that the urethra will take, is passed, and 6 ounces (200 ml.) of lotion are introduced into the bladder. The evacuator (fig. 1485), filled with lotion and with its tap closed, is fitted on to the cannula. The tap is opened and the bulb is elevated so as to depress the beak of the cannula towards the base of the bladder, after which the bulb is compressed slowly. The bulb is then permitted to expand, and the returning lotion carries with it sand and fragments of the stone which, being heavier than the lotion, drop into the glass receptacle. Compression of the bulb and aspiration is continued until no further fragments fall. The beak of the cannula is turned to the left and to the right, and suction is applied in these situations. To save time in emptying and refilling two evacuators can be used. If at any time the bulb fails to expand, this may be due to blocking of the eye by bladder mucosa, in which event release can be effected by further compression of the bulb and slight rotation of the instrument. Blockage of the cannula by fragments of the stone can sometimes be remedied in the same way, but more often they must be dislodged by detaching the evacuator and passing the obturator of the cannula. When no more fragments can be aspirated, the evacuator is detached and the bladder is irrigated until the lotion is returned clear. The cystoscopic lithotrite is reintroduced, and if no fragments remain, a Foley's catheter is passed. After the patient has been returned to bed the catheter is connected to a sterile bottle. Usually the catheter can be removed in twenty-four hours. As soon as it is assured that the patient is passing urine without difficulty, he can return home. Suitable drugs should be given until the urine is sterile.

Suprapubic Lithotomy.—The alternative to litholapaxy is removal of the stone through a suprapubic incision, after which the bladder is closed and drained by a urethral catheter.

Lithotomy in Special Circumstances.—A stone associated with an enlarged prostate can be removed in the course of a suprapubic or retropubic prostatectomy. When the stone is associated with contracture of the bladder neck, it can be removed in the course of an open operation for that condition.

Frère Jacques de Beaulieu, 1651–1714, was the most famous Journeyman Lithotomist; he mainly practised the perineal operation using a bread-knife.

Edward Canny Ryall, 1865–1934. Senior Surgeon, All Saints' Hospital for Genito-Urinary Diseases, London.
Henry Jacob Bigelow, 1818–1890. Surgeon, Massachusetts General Hospital, U.S.A.

A very small stone sometimes can be removed by means of an evacuator after passing the largest-sized cannula commensurate with the calibre of the urethra. For stones too large to pass through the cannula but small enough to pass through the urethra, removal by seizing the stone in the jaws of a cystoscopic rongeur (fig. 1486) is ideal.

FOREIGN BODIES IN THE BLADDER

A piece of catheter or bougie may become broken and remain in the bladder. The variety of foreign bodies which have been removed from the bladder is astonishing, e.g. manicure sticks, hair-clasps (fig. 1487), hairpins and candle-grease. The presence of such objects in the bladder is usually accounted for by urethral masturbation, or attempts to produce a miscarriage. Occasionally a foreign body enters through the wall of the bladder, e.g.



FIG. 1487.—A hair-clasp in the bladder.

a piece of rubber tubing after cystostomy; unabsorbable sutures used in an extravesical pelvic operation. The diagnosis rests on cystoscopy, and in the case of radio-opaque foreign bodies on radiography.

Complications of a Foreign Body in the Bladder:

1. Cystitis, which is the most common complication.
2. Perforation of the bladder wall.
3. Vesical calculus.

Treatment.—A small foreign body can be removed per urethram by means of an operating cystoscope or Young's cystoscopic rongeur. When the foreign body is heavily encrusted, penetrating the bladder wall, or accompanied by severe cystitis, the suprapubic route should be chosen. Paraffin wax can be dissolved by the introduction of equal parts of xylol and water into the bladder for half an hour.



FIG. 1486.—Young's cystoscopic rongeur.

DIVERTICULUM OF THE BLADDER

Definition.—It is most important to distinguish a saccule from a diverticulum of the bladder. The normal intravesical pressure at the commencement of micturition is about 35 cm. of water. Pressures as great as 100 cm. are reached by a hypertrophied (trabeculated) bladder (fig. 1522) endeavouring to force urine past an obstruction. This pressure causes the mucous lining between the inner layer of hypertrophied muscle bundles to protrude, so forming multiple saccules. If one or more, but usually one, saccule is forced through the whole thickness of the musculature of the bladder wall it becomes a diverticulum.

Ætiology

(a) **Congenital diverticulum** is rare and unimportant. It is situated in the middle line anterosuperiorly, and represents the unobliterated vesical end of the urachus. It empties with the bladder, and is symptomless.

(b) **Pulsion Diverticulum.**—The causative obstructive lesion, in order of frequency, is contracture of the bladder neck, benign enlargement of the prostate, fibrous prostate, urethral stricture and congenital valves of the posterior urethra.

Pathology.—Usually the mouth of the diverticulum is situated above and to the outer side of one ureteric orifice. Exceptionally, it is near

Hugh Hampton Young, 1870-1945. Emeritus Professor of Urology, Johns Hopkins School of Medicine, Baltimore, U.S.A.

the middle line behind the interureteric ridge. The size varies from 2–5 cm., but may be larger. It is lined by bladder mucosa and the wall is composed of fibrous tissue only (compare traction diverticulum p. 1140). A diverticulum enlarges in a downward direction and sometimes may obstruct a ureter.

Complications

1. **Recurrent Cystitis.**—As the pouch cannot empty itself there remains a stagnant pool of urine within it. Once infected, the infection persists and continues to reinfect the bladder. In long-standing cases peridiverticulitis causes dense adhesions between the diverticulum and surrounding structures.

2. **Vesical calculus** due to stagnation and infection is present in 20 per cent. of cases, most often in the bladder, sometimes in the diverticulum as well, less often in the diverticulum only. On rare occasions a dumb-bell calculus fills the diverticulum and projects into the bladder.

3. **Hydronephrosis and hydroureter**, consequent upon compression of the lower end of the corresponding ureter, are liable to be followed by pyelonephritis and pyonephrosis.

4. **Neoplasm** arising in a diverticulum is an uncommon complication. Unless the diagnosis is made at a very early stage, the prognosis is poor, because extravescical invasion through the thin wall of the sac occurs so readily.



Clinical Features.—An uninfected diverticulum of the bladder may cause no symptoms whatsoever. The patient is nearly always a male (95 per cent. of cases), usually over fifty years of age.

There are no pathognomonic symptoms of a vesical diverticulum; they are those of lower urinary tract obstruction, recurrent cystitis, and pyelonephritis. Hæmaturia (due to cystitis, vesical calculus, or, rarely, a neoplasm) is a leading symptom in one-third of cases. In a few cases micturition occurs twice in rapid succession (the second act may follow a change of posture), and when the first specimen is clear and the second cloudy, diverticulum of the bladder should be strongly suspected. In cases of chronic retention of urine, two swellings may be recognised rising out of the pelvis, one being the bladder and the other the diverticulum.

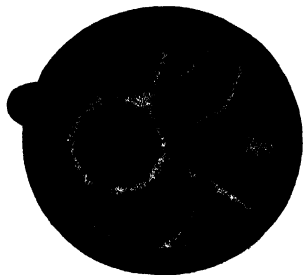


FIG. 1488.—Cystoscopic appearance of the orifice of a diverticulum and trabeculation of the bladder.

Cystoscopy is the usual means of discovering the diverticulum. Most often its orifice is seen as a clear-cut hole about the diameter of a lead pencil, the depths of which are black and unilluminated (fig. 1488). With inadequate distension of the bladder, sometimes the mouth of the diverticulum is seen closed, when the mucous membrane around the potential orifice is thrown into radiating pleats (fig. 1489); therefore, when

searching for a diverticulum, it is important to have the bladder fully distended. In heavily infected cases much irrigation is necessary before a clear view of the bladder wall can be obtained. The differential diagnosis of a diverticulum from sacculation of a trabeculated bladder is not difficult, for the orifice of the sacculi is relatively large, and the shallow interior

can be seen. It is sometimes possible to pass an endoscope into a diverticulum and examine its interior.

Excretory pyelography will not only exclude or reveal implication of the upper urinary tract, but in many instances the accompanying cystogram will give information regarding the size of the diverticulum.

Retrograde cystography is employed only when the former fails to show the pouch clearly. The bladder is emptied of urine, and filled with a non-irritating radio-opaque medium. Radio-

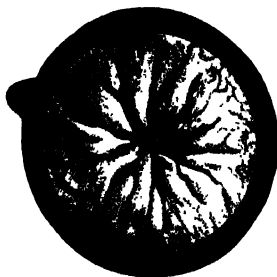


FIG. 1489.—Occasional appearance with inadequate distension of the bladder.

graphs taken before and after micturition show the dimensions and position of the diverticulum clearly (fig. 1490).



FIG. 1490.—Diverticulum of the bladder shown by cystography.

Indications for Operation.—A vesical diverticulum gradually increases in size, and once infected the infection cannot be permanently eradicated. Therefore, unless the pouch is small and uninfected, it should be removed, usually when the associated lower urinary tract obstructive lesion is treated.

Pre-operative Treatment.—When, as is usual, the urine is infected, suitable pre-operative antibiotic treatment is given. In the presence of gross sepsis and retention of urine, it is necessary to resort to an indwelling urethral catheter for forty-eight hours at a time, and give bladder washes. Only in exceptional cases, when the infection cannot be controlled by these means, is suprapubic cystostomy indicated, and then a second tube should be passed into the diverticulum and stitched to the orifice so that both cavities can be irrigated frequently. Suprapubic cystostomy renders subsequent diverticulectomy more difficult.

Combined intravesical and extravesical diverticulectomy is the operation that is practised most frequently. Cystoscopy is performed, and a large ureteric catheter is passed up the ureter on the affected side, and left in place, 8 ounces (240 ml.) of lotion remaining in the bladder. With the patient in the Trendelenburg position, the anterior bladder wall is exposed through a suprapubic incision. The peritoneum is dissected upwards, and that side of the bladder bearing the diverticulum is cleared from surrounding structures with the fingers until some part of the pouch is brought into view. The bladder is then incised in the middle line near its dome, and emptied by suction. The interior of the diverticulum is packed with a strip of gauze, and with the fingers of one hand in the bladder and the recti muscles retracted widely, the diverticulum is freed from surrounding structures by gauze and sharp dissection. Usually the neck of the diverticulum can be separated from the ureter, and when the pouch is free it is severed from its attachment to the bladder with a diathermy knife. The resulting defect is closed in two layers. The cystostomy incision is sutured around a Malécot catheter, and the abdominal wall is closed, leaving a drainage tube in the extravesical space which housed the sac.

An alternative method, if the sac is densely adherent, is to carry the incision in the bladder down to the rim of the diverticular orifice, then to detach the diverticulum, together with its fibrous rim. The incision in the bladder is closed and the diverticulum left in position with a corrugated drain into it for two to three days. The track fibroses rapidly after removal of the drain.

TRACTION DIVERTICULUM OF THE BLADDER (*syn.* **HERNIA OF THE BLADDER**)

A portion of the bladder protruding through the inguinal or femoral hernial orifice occurs in 1.5 per cent. of such herniæ treated by operation (Sir Cecil Wakeley). The condition is relatively frequent in femoral and direct inguinal herniæ. The disposition of the diverticulum in the hernia varies, and in order of frequency is:

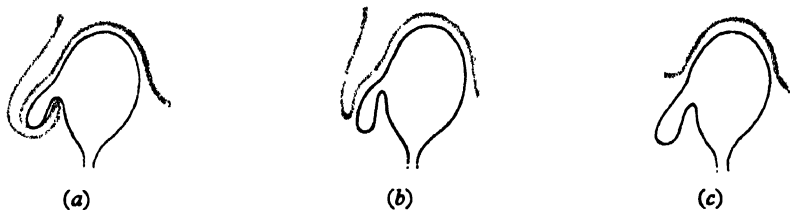


FIG. 1491.—(a) Intraperitoneal, (b) paraperitoneal, (c) extraperitoneal hernia of the bladder.

Clinical Features.—In a few cases, in order to empty the bladder completely, the patient finds it necessary to exert pressure on the hernia. Suspicion of a bladder hernia can be confirmed by cystoscopy or cystography.

Accidental Wounding of the Bladder during an Operation for Hernia.—When recognised at the time, the wound in the bladder should be closed by a double layer of catgut and an indwelling catheter is retained for a week or so. If the accident is not recognised at the time, unless early operation is undertaken to repair the rent and drain the prevesical space or peritoneal cavity, as the case may be, together with suprapubic cystostomy, the outlook is grave.

BLADDER FISTULÆ

Vesico-vaginal fistula “is the greatest misfortune that can happen to a woman, the more so because she is condemned to live with it, and cannot hope to die from it” (Dieffenbach).

Ætiology:

1. *Obstetrical Causes.*—From time immemorial the usual cause has been protracted or neglected labour.
2. *Gynæcological Causes.*—The operations chiefly concerned with this complication are total hysterectomy and anterior colporrhaphy.
3. *Radiotherapeutic Causes.*—First and foremost is the radium treatment of carcinoma of the cervix uteri; to a lesser extent irradiation of the pelvic viscera for other reasons is responsible.
4. *Direct Neoplastic Infiltration.*—Exceptionally carcinoma of the cervix uteri ulcerates through the anterior fornix to implicate the bladder.

When a wound of the bladder is recognised and repaired at once, leakage is uncommon, but escape of urine will quickly follow if such damage is passed unnoticed. However, most vesico-vaginal fistulæ are the result of ischæmic necrosis of the bladder wall due to prolonged pressure of the foetal head in obstetric cases. In gynæcological cases the ischæmia is brought about by grasping the bladder wall in a hæmostat, including the bladder wall in a suture, or perhaps even by local œdema or hæmatoma. Leakage due to necrosis of tissue seldom manifests itself before seven days after the operation.

An intractable fistula following radium treatment of carcinoma of the cervix uteri may arise from avascular necrosis years after the apparent cure of the original lesion.

Clinical Features.—There is leakage of urine from the vagina, and, as a consequence, excoriation of the vulva. Digital examination of the vagina reveals a localised thickening on its anterior wall. On inserting a bivalved speculum, urine will be seen escaping from an opening in the anterior vaginal wall. It is usually possible to pass a bent probe from the vagina into the bladder. Cystoscopy is often difficult, owing to the contraction of the bladder from cystitis and the escape of urine from the fistula; however, usually the tip of the probe that has been passed can be seen emerging through an area of granulation tissue.

Differential Diagnosis between a Uretero-vaginal and Vesico-vaginal Fistula.—If a swab is placed in the vagina and a solution of methylene blue is injected through the urethra, the vaginal swab becomes coloured blue. In the case of a uretero-vaginal fistula the vaginal swab is not so coloured. Uretero-vaginal fistula is discussed on p. 1083.

Treatment.—Operation must be postponed until four to six months after the development of the fistula, so as to enable viable tissue in the neighbourhood to recover fully, and for scar tissue to consolidate. The fistula is exposed via the vagina; the edges are freshened and a longitudinal repair of tantalum wire used to traverse the bladder wall (not mucosa) and the whole thickness of the vagina. Parallel vaginal incisions may be used to relieve tension on the suture lines. The bladder is drained by No. 4 polythene tubing (Chassar Moir) for fourteen days, unless the urethra is primarily affected, when a suprapubic cystostomy is performed.

Vesico-intestinal Fistula

Congenital (p. 979).

Inflammatory.—Colonic diverticulitis is the most common inflammatory cause. Other inflammatory causes are Crohn's disease, pelvic appendix abscess, tuberculous peritonitis and suppurative parametritis.

Neoplastic.—Carcinoma of the pelvic colon ulcerating into the bladder or, more rarely, carcinoma of the bladder ulcerating into the colon or rectum.

Clinical Features.—The condition is more common in men than in women, owing to the interposition of the uterus. In cases due to colonic diverticulitis sometimes there are inaugural bladder symptoms due to the inflamed diverticulum impinging upon the bladder (p. 901). Once a vesico-intestinal fistula has penetrated the full thickness of the bladder wall, in addition to intractable cystitis, the patient passes gas per urethram (pneumaturia). Occasionally faecal matter is passed with the urine, and more rarely still urine is passed via the bowel. There is often an inflammatory or neoplastic mass to be felt in the recto-vesical or utero-vesical pouch.

Cystoscopy.—That a vesico-intestinal fistula is present may be obvious (fig. 1492). If the fistula is small and its edges smooth, it is likely to be inflammatory. Other methods of investigation which may help in determining accurately the situation of the fistula are retrograde cystography and a barium enema.

Treatment.—A defunctioning colostomy is made above the fistula as the first step. Inflammation is allowed to subside over two to three months. At laparotomy the communication is separated, the hole in the bladder being closed and patched with omentum, and the segment of diseased bowel resected. A temporary suprapubic cystostomy completes the operation. The colostomy is closed two weeks later.

Cases due to Carcinoma.—By the time a fistula between the bowel and the bladder has developed, as a rule, the growth is inoperable or requires pelvic evisceration for its attempted eradication. As so often these patients are unfitted for this major procedure, it is best to leave the patient with the defunctioning colostomy performed in the first instance. Exceptions do occur, and occasionally it is warranted to undertake segmental resection of the colon and a partial or complete cystectomy.

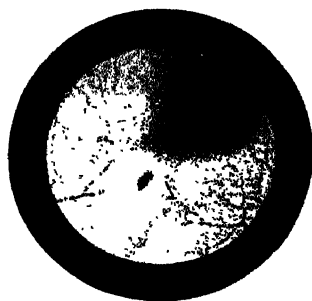


FIG. 1492.—Cystoscopic view of a vesico-intestinal fistula. Bubbles of gas can be seen issuing from the orifice of the fistula.

James Marion Sims, 1813–1883, while a country Practitioner at Montgomery, Alabama, was the first to succeed in closing a vesico-vaginal fistula.

John Chassar Moir, Contemporary. Nuffield Professor of Obstetrics and Gynaecology, Oxford.

Burrill B. Crohn, Contemporary. Gastroenterologist, Mount Sinai Hospital, New York, U.S.A.

CYSTITIS

Both acute and chronic cystitis occur at all times of life, and in both sexes, but they are especially common in women.

Predisposing Causes

1. Incomplete emptying of the bladder, such as occurs in prostatic obstruction, urethral stricture, stenosis of the external urinary meatus, diverticulum of the bladder, pregnancy (and more particularly the puerperium), cystocele, also injuries and diseases of the spinal cord.
2. The presence in the bladder of a calculus, foreign body, or neoplasm.
3. Lowered general resistance from intercurrent disease and avitaminosis.

Avenues of Infection

1. *Descending* from the kidney along the lumen of the ureter.
2. *Ascending* from the urethra is the usual cause. The shortness of the urethra is held responsible for the comparatively common occurrence of cystitis in females. The passage of urethral instruments is a source of cystitis in both sexes, more especially when residual urine is present in the bladder. This does not necessarily imply that the instrument was unsterile, but rather that instrumentation awakens a latent infection in the posterior urethra.
3. *Lymphogenous*.—The rich lymphatic network at the base of the bladder which communicates with the prostate, the seminal vesicles or the cervix uteri, and the rectum, provides an avenue by which organisms can reach the bladder from these commonly infected structures.
4. *From Adjacent Structures*.—Fistulous communications with the intestine, the vagina, and the Fallopian tube, or by suprapubic cystostomy.
5. *Hæmatogenous*.—Metastasis from a distant focus is exceptional. Interstitial cystitis is regarded as a probable example.

When cystitis has been brought under control, every endeavour should be made to search for, and if present, treat a primary focus of infection or remove a predisposing cause. In many cases none can be found.

Bacteriology.—*Esch. coli* is the commonest infecting organism. Less frequently *Streptococcus faecalis*, *Staphylococcus aureus*, *Staphylococcus albus*, *B. proteus* or *Ps. æruginosa* is responsible. *B. proteus* and *Staphylococcus albus* are urea-splitting organisms that cause persistent alkalinity of the urine. Tuberculous cystitis is considered under 'Special Forms of Cystitis', p. 1143.

The presence of pus cells without organisms calls for repeated examinations for the *M. tuberculosis* and the gonococcus (an exceedingly rare cause of cystitis). Having eliminated these possibilities, the condition may be one of abacterial cystitis (p. 1143).

Clinical Features.—The severity of the symptoms varies greatly; those of acute cystitis are usually the more distressing, but some chronic cases vie with them in intensity.

Frequency is the outstanding symptom, both during the day and at night. The desire to urinate occurs from every hour to every few minutes, and often it is so urgent that if the bladder cannot be emptied forthwith, incontinence results. In severe cases, because of infection and loss of sleep, the patient looks drawn and haggard.

Pain varies from mild to agonising. When the inflammation is situated in the dome of the bladder, pain is referred to the suprapubic region, while

when, as is often the case, the inflammation involves the trigone, pain is referred to the tip of the penis, the labia majora, and the perineum.

Hæmaturia.—The passage of a few drops of blood-stained urine or blood-stained debris at the end of micturition is a frequent accompaniment. Less often the whole specimen is blood-stained, but more so at the end.

Pyuria is always present, except in some cases of trigonitis and interstitial cystitis (p. 1145). If urine is passed into two glasses, the second is the more cloudy.

On examination there is tenderness over the bladder suprapubically, per rectum, or per vaginam. In the acute stage it is necessary to differentiate between cystitis secondary to pyelonephritis and primary cystitis. Absence of tenderness over the renal angles, and a normal or slightly elevated temperature also point to a non-renal origin. The absence of a urethral discharge or a tender swelling of the prostate eliminates a primary focus in the urethra or the prostate. A mid-stream specimen of urine (in the male and in the female) should be sent for bacteriological examination with a request that the organisms responsible be tested regarding their sensitivity to antibiotics. The only other investigation permissible at this stage is excretory pyelography, and normal renal shadows substantiate a non-renal origin of the infection.

Treatment should be commenced forthwith, and modified if necessary when the bacteriological report is to hand. The patient is urged to drink plentifully, and in severe infections it is a good practice to commence treatment with nitrofurantoin (100 mg. q.d.s. p.c). This drug has a broad spectrum of antibacterial activity and is particularly effective against *Esch. coli* and pathogenic cocci. Failure to respond, or early recurrence of infection, indicates the necessity for further investigation to exclude predisposing factors.

Cystoscopy to confirm the diagnosis may be performed in women during the acute phase. The appearances may vary from hyperæmia, sometimes with mucosal hæmorrhages, to severe sloughing and ulceration of the mucosa. In men, cystoscopy to exclude predisposing causes should be delayed until the infection has been controlled.

SPECIAL FORMS OF CYSTITIS

Acute Abacterial Cystitis (*syn.* Acute Hæmorrhagic Cystitis).—The patient, a young man or woman, presents with symptoms of severe cystitis. Pus is present in the urine, but no organism can be cultured therefrom. The condition, which sometimes is associated with abacterial urethritis, commonly follows recent venereal exposure. While tuberculous cystitis always must be ruled out by cultural or biological tests, the fact that excretory pyelography shows normal excretion on both sides strongly favours acute abacterial cystitis. In 60 per cent. of cases, by special cultural methods the pleuro-pneumonia-like organism, which frequently accompanies acute abacterial urethritis, is found.

Although this is a self-limiting disease its course can be shortened by the antibiotic therapy recommended for abacterial urethritis (p. 1191). When cystoscopy is performed after the acute symptoms have abated, abundant mucosal hæmorrhages are seen, and presumably at its height the infection causes a hæmorrhagic cystitis.

Uretero-trigonitis is a common form of recurrent lower urinary tract infection in women. Cystoscopy shows increased vascularity of the trigone,

œdema of its mucous membrane (most marked towards the apex) and, in severe cases, there is a pseudo-membrane limited to the trigone. Inflammatory polypi may be present at the bladder neck (fig. 1582). Later the urethra becomes narrowed.

A primary focus of infection must be sought; the most common being (a) the cervix uteri (chronic cervicitis or a cervical erosion) or (b) the intestinal tract, notably chronic colonic diverticulitis. No examination is complete without examining the cervix with a speculum, and if the cervix is healthy a barium enema is necessary for evidence of diverticulitis.

Treatment.—In addition to the usual measures, urethral polypi should be destroyed by light fulguration and the urethra dilated, if necessary. Cervicitis or a cervical erosion is treated by excising the glandular tissue of the cervix with a diathermy loop. Diverticulitis is discussed on p. 901.

Tuberculous cystitis is always secondary to renal tuberculosis, and unless treated early is a particularly relentless form of chronic cystitis.

Cystoscopy shows that early tuberculosis of the bladder commences around one ureteric orifice or in the neighbourhood of the trigone, the earliest evidence being pallor of the mucosa, due to sub-mucous œdema. Subsequently tubercles may be seen, and in long-standing cases there is much fibrosis, and the capacity of the bladder is so much reduced that it has earned the name of 'thimble' bladder. This feature can be well shown by cystography (fig. 1493).

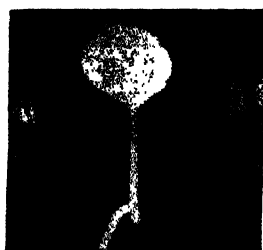


FIG. 1493.—Retrograde cystograph showing exceedingly contracted ('thimble') bladder in a case of tuberculous cystitis.

Treatment.—Tuberculous cystitis usually responds rapidly to anti-tuberculous drugs (p. 23), but occasionally, in cases with advanced renal changes, may not subside until the involved kidney and ureter have been removed. Local treatment of persistent tuberculous cystitis consists of instillations of a substance inimical to *M. tuberculosis*. Among the best is B53, a soap derived from a branched fatty acid, having a high inhibitory index for the *M. tuberculosis*. A correctly buffered 0.5 per cent. solution containing local anæsthetic is instilled and retained for one hour. A fortnight's course can be repeated after an interval of a week.

When the bladder is considerably contracted, but free from ulceration, remarkable relief from the distressing frequency can be achieved by ileo-cystoplasty (fig. 1494), which is an extremely efficacious method of increasing the capacity of a contracted bladder. When, in addition, tuberculous contracture threatens to implicate the remaining kidney by back pressure, as shown by dilatation of the ureter on excretory pyelography, the ureter is implanted into the newly constructed pouch of intestine (p. 1159) with every hope of conserving renal function as well as rendering the patient symptom-free.

Ileocystoplasty.—After pre-operative sterilisation of the intestines (p. 921) a 6-inch (15-cm.) segment of ileum with an ample blood supply as demonstrated by transillumination is disconnected, leaving its mesentery intact, and the continuity of the intestine is restored by anastomosis. After closing the ends of the donor segment and opening it longitudinally it is anastomosed to the dome of the contracted bladder (fig. 1494).

Interstitial Cystitis (syn. Hunner's Ulcer; Elusive Ulcer).—For practical purposes Hunner's ulcer can be said to be a condition peculiar to women. The symptoms commence in the early forties. It can cause more pain, mental anguish and associated neurosis than does carcinoma of the bladder; as a consequence, the incessant painful micturition it occasions sometimes leads to drug addiction and even suicide.

Ætiology is still as obscure as it was when Guy Hunner first described the condition in 1914. Certainly it does not commence as an ordinary pyogenic infection of the mucous membrane, but rather as an infection of the paravesical tissues secondary, it is suggested, to infection of the adnexae, or even to infection from a more distant focus such as the nasopharynx. Some believe the condition is due to an attenuated *M. tuberculosis* which so far has defied isolation. In a number of instances the symptoms have commenced three or four months after a pelvic operation.

Pathology.—As a result of the paracystitis, fibrosis of the vesical musculature ensues, leading to contracture of the bladder and areas of avascular atrophy of the mucous membrane. Finally, ulceration of the mucosa occurs in the least vascular portion of the bladder, namely the fundus. Often the capacity of the bladder is reduced to 1 to 2 ounces (30 to 60 ml.). The characteristic linear bleeding ulcer is a crack due to splitting of the mucous membrane when the bladder is distended under anæsthesia for cystoscopy.

Microscopically, inflammation of all coats of the bladder is present with granulation tissue in the submucosa underlying the ulcer. The muscularis is hypertrophied and the peritoneum in proximity to the area of maximum disease is thickened.

Clinical Features.—Bladder capacity becoming much reduced, increased frequency, eventually every hour both day and night, is the leading symptom. Pain, relieved by micturition and aggravated by jarring and overdistension of the bladder is the second and most characteristic symptom. In early cases the urine is crystal clear and sterile; in later cases it contains ordinary pyogenic bacteria in under half the specimens examined; in the remainder the urine remains sterile. In 60 per cent. of cases hæmaturia is a leading symptom.

Cystoscopy.—The lesion appears in the roof of the bladder as a star-shaped area of intense congestion in which a fissure (fig. 1495) can be seen when the bladder is distended.

The ulcer, if ulcer it can be called, bleeds readily.

Treatment is difficult and unsatisfactory. Local hydrocortisone and irrigation of the bladder with various sedatives have proved valuable. Hydrostatic dilatation under anæsthesia may give relief for some months. Light diathermy fulguration of the ulcer may help. Complete relief may be obtained by urinary diversion, while ileo-cystoplasty after excision of the affected bladder wall, though frequently very satisfactory, is not always permanently successful in relieving all symptoms.

Alkaline encrusting cystitis is due to urea-splitting organisms causing phosphatic encrustations on the bladder mucosa of elderly women. There are symptoms of chronic cystitis and a plain X-ray shows the

bladder outline. The encrustations can be curetted and removed via a cystoscope or a suprapubic cystotomy, or controlled by tidal lavage using a dissolvent solution or solution G (p. 1094), or $\frac{1}{2}$ per cent. acetic acid.

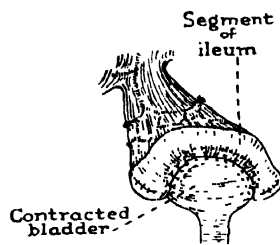


FIG. 1494. — Ileocystoplasty using a 6-inch segment of ileum. (After D. O. Ferris.)



FIG. 1495. — Hunner's ulcer, cystoscopic appearance.

Cystitis Cystica.—Glands are not found in the normal vesical mucosa. Under the influence of chronic inflammation, the surface epithelium sends down buds, resulting in minute cysts filled with clear fluid, most abundant on the trigone. Most of the cases are seen in women, and the urine usually contains pus, and commonly *E. coli*. An adenocarcinoma may originate in one of these cysts.

BILHARZIASIS OF THE BLADDER

Geographical Distribution.—The disease is endemic in the greater part of Africa, throughout Israël, Syria, Arabia, Iran and Iraq. It is met with frequently in Portugal and in Greece, and in the islands of Madagascar, Mauritius and Cyprus. Dwellers along the valley of the Nile have suffered from time immemorial. The condition also occurs along the shores of some of China's great lakes. Marshes or slow-running fresh water provide a favourable habitat for the particular fresh-water snail (*Bullinus contortus*), which is the intermediate host.

Mode of Infestation.—The disease is acquired while bathing or standing in infected water. The free-swimming bifid-tailed embryos (cercariæ) of the trematode *Schistosoma hæmatobium* penetrate the skin by their motile and erosive secretory powers. Shedding their tails, which enabled them to swim, they enter blood-vessels and are swept by the blood-stream into all parts of the body. All perish save those

that are carried to the liver. Once within the liver the survivors flourish by sustaining themselves on erythrocytes, and they develop into male and female worms. The female is long, smooth and slender and is furnished with two weak suckers anteriorly. The male is broader, shorter (11 mm. in length), bosselated and provided with a strong sucker at either end. Sexual maturity having been attained, the nematodes leave the hepatic nursery and enter the portal vein. Here the male bends into the shape of a canoe and in so doing a gutter (the gynæcophoric canal) is formed along its length. Into the groove a female nestles and, thus conjugating, the pair (fig. 1496) paddle their way against the stream towards the inferior mesenteric vein. The long journey is accomplished in short stages, the male's suckers serving as anchors to the vein wall during periods of rest. *Schistosoma hæmatobium* worms have an affinity for the vesical venous plexus, and to reach it they must pass along the portal-systemic



FIG. 1496.—Conjugating male and female *Schistosoma hæmatobium* trematodes. (After A. Looss.)

anastomotic channels, the most important in this respect being the venules in relation to the lumbar lymph nodes (Makar); other communications being venules in relation to the lower part of the ureters.

Having reached one of the smaller radicles of the vesical plexus the pair can go no further coupled, so the female, now pregnant but still comparatively slender, parts from the male and moves forward until she enters a submucous venule so small that she completely blocks it. She now proceeds to lay about twenty ova in a chain, and after each is deposited, by slightly withdrawing herself, the venule contracts upon the ovum. Each ovum is provided with a terminal spine (fig. 1497) which penetrates the vessel wall. Aided by muscular contraction of the bladder, and perhaps by secreting lytic fluid, some of the ova reach the lumen of the bladder; others die incarcerated in the mucous membrane. A heavily infected subject passes many hundreds of ova a day. If the ova reach fresh water, the low osmotic pressure of the new environment causes their envelope to burst, and thus emerges the miracidium, which is ciliated. To survive, the miracidium must reach and penetrate the intermediate host within thirty-six hours. Within the snail's liver the miracidium enlarges and gives rise to myriads of daughter cysts, broods of which are set free on the death of the snail. A single miracidium begets thousands of cercariæ to complete the life-cycle.

Clinical Features.—After penetration of the skin there may arise urticaria, which lasts for about five days and sometimes recurs (swimmer's itch). After an incubation period ranging from four to twelve weeks, high evening temperature, sweating, and asthma, together with leucocytosis and eosinophilia of over 10 per cent., sometimes



FIG. 1497.—A bilharzia *hæmatobium* ovum.

last several weeks. Usually an asymptomatic period of several months supervenes before the ova laid in the bladder wall find egress and occasion the typical early sign and symptom of intermittent, painless, terminal hæmaturia. Men are three times more often affected than women. Native patients of the peasant class rarely consult a doctor for this hæmaturia *per se*, so that many late cases are encountered.

Examination of the Urine.—The last few millilitres of an early-morning specimen of urine are collected and centrifuged. It is essential that all receptacles be absolutely dry. The ovum is recognised without staining under the low-powered microscope. Examination on several consecutive days may be required. Even so, a negative result does not exclude bilharziasis, especially in patients no longer resident in bilharzial districts.

Cystoscopy.—Dependent on the length of time the disease has remained untreated, cystoscopy will reveal one or more of the following :

1. *Bilharzial pseudo-tubercles* are the earliest specific appearance. The pseudo-tubercles are larger, more prominent, more numerous, more yellow and more distinctly grouped (fig. 1498) than those of tuberculosis.
2. *Bilharzial nodules* (fig. 1499) are due to the fusion of tubercles in the presence of secondary infection. They are larger and greyer than the foregoing.



FIGS. 1498.—Bilharzial tubercles ; 1499 Bilharzial nodules ; 1500 'Sandy patches'.
(After N. Makar.)

3. 'Sandy patches' are the result of calcified dead ova with degeneration of the overlying epithelium. They occur in the first instance around one or both ureteric orifices (fig. 1500). Considerable calcification of this nature is visible on the radiograph.

4. *Ulceration* is the result of sloughing of mucous membrane containing dead ova, or what is even more common, sloughing of a bilharzial papilloma. The ulcer is shallow (fig. 1501), bleeds readily, and its common position is the posterior wall of the bladder.

5. *Fibrosis* is mainly the result of secondary infection. The capacity of the bladder becomes much reduced. Contracture of the bladder neck is also a common result of bilharzial fibrosis.

6. *Granulomas.* — Bilharzial masses due to an aggregation of nodules form. They are sessile, soft and bleed readily when touched.

7. *Papillomas* are distinguished from the foregoing by being more pedunculated (fig. 1502). They vary in size from that of a pea to that of a walnut, and they may be single or multiple.

8. *Carcinoma* is a common end-result in grossly infected bilharziasis of the bladder which has been neglected for years. It usually commences, not in a papilloma, but in an ulcer, and is therefore a squamous-celled carcinoma.

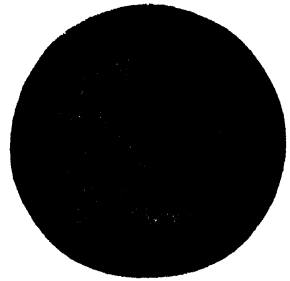


FIG. 1501.—Bilharzial ulcer. (After N. Makar.)



FIG. 1502.—Bilharzial papilloma. (After N. Makar.)

Napoleon's troops who served in his march to the Pyramids suffered from hæmaturia.

Treatment.—Lesions 1 to 6 inclusive can be expected to heal under general treatment by antimony preparations as described on p. 1003. It takes many months for dead ova to be expelled, and even after repeated courses and healing of the bladder lesion, living bilharzial worms have been found at necropsy in the portal system. In addition to general treatment, healing of bilharzial ulcers and granulomas is expedited by light diathermy coagulation. Bilharzial papillomas and, of course, carcinoma do not respond to general treatment: these lesions require the same surgical measures as non-bilharzial papillomas and carcinomas, in addition to general treatment.

Other Complications and their Treatment:

(a) *Secondary bacterial cystitis* is commonly present in cases of some standing and the treatment of it must be thorough and prolonged.

(b) *Urinary calculi*, especially vesical and ureteric, occur more frequently when bilharzial lesions of the bladder are present. Litholapaxy is contraindicated in cases where there is ulceration of the bladder wall.

(c) *Stricture of the ureters* affects the last inch of the ureters. These strictures often respond to dilatation, but sometimes transplantation of the affected ureter into another part of the bladder is necessary.

(d) *Prostato-seminal vesiculitis*, like tuberculosis of these structures, is made worse by prostatic massage, and general treatment alone must be employed.

(e) *Contracture of the bladder, and contracture of the bladder neck*, must be treated in the same ways as similar contractures of non-bilharzial origin.

(f) *Bilharzial urethral strictures* are often accompanied by fistulæ, and can be cured only by excision of the fistulous tracks and urethroplasty (p. 1196).

NEOPLASMS OF THE BLADDER

More than 95 per cent. of primary tumours of the bladder originate in its mucous membrane; the remainder are connective-tissue growths—angioma (the least uncommon), myoma, fibroma, and sarcoma. These are too rare to merit further description, as also is a phæochromocytoma arising in the bladder wall from accessory adrenal tissue.

Another uncommon benign bladder tumour is an **endometrioma**, characterised by a localised, smooth, vascular projection on the bladder wall, sometimes containing chocolate-coloured cysts; and at other times translucent cysts of a bluish hue. The tumour enlarges and bleeds during menstruation. Treatment is partial cystectomy.

Secondary involvement of the bladder can occur from extension of a malignant neoplasm of a neighbouring organ, particularly the large intestine (sigmoid and rectum), the uterus or an ovary.

On rare occasions the first symptoms of a carcinoma of the pelvic colon are those of cystitis. Cystoscopy reveals a circumscribed area of intense inflammation, usually on the left side of the fundus. If the diagnosis is not made and treatment carried out at this stage, a vesico-intestinal fistula results (p. 1141).

PRIMARY EPITHELIAL TUMOURS OF THE BLADDER

Ætiology.—Apart from the facts that epithelial tumours occur in aniline-dye workers, and workers in other industries using similar chemicals more frequently than in other persons; that carcinoma of the bladder not infrequently arises in cases of long-standing vesical bilharziasis, especially when it is chronically infected and encrusted; and that, as with epithelial

John Christopherson, 1868–1955, Director of Civil Hospitals, Khartoum, introduced the treatment of bilharziasis by the intravenous injection of tartar emetic.

tumours of the tongue, leukoplakia must be regarded as a precarcinomatous condition, nothing is known as to the cause of tumours of the bladder.

Some believe that these tumours are all due to a carcinogen (as yet unidentified) excreted in the urine, and that so-called recurrences are new neoplasms brought into being by the self-same carcinogen. Others maintain that some papillary neoplasms of the bladder are due to a filtrable virus and quote *papillomata acuminata* (p. 1209) as a pertinent analogy.

Whatever the cause, it is an established fact that tumours of the bladder are increasing in frequency at a rate that cannot be accounted for either by improved methods of diagnosis or the ageing of the population. Also these tumours of the bladder are occurring at an earlier age than was the case twenty years ago.

Tumours of the Bladder as an Industrial Disease.¹—Papillomas, and especially carcinomas of the bladder, occur with considerable frequency among aniline-dye workers and in workers in the rubber and several other industries where certain chemicals, notably aromatic amines and coal-tar products are used. Although in Britain the manufacture of β -naphthylamine was banned in 1952, as the bladder tumours take up to twenty years to develop, and as the exposure need not have been long, new cases due to absorption of β -naphthylamine continue to occur and are to be expected for a number of years to come (Kennedy). Means have not been found for doing without other, though less potent, carcinogens, such as benzidine, α -naphthylamine and auramine, and new cases from absorption of these chemicals are bound to occur.

Pathology

Benign Villous Tumours.—A papilloma of the bladder commences as a single frond with a central vascular core—later it becomes tufted. From these tufts spring villi—long, finger-like projections composed of three or four layers of transitional epithelium surrounding a capillary vessel with a minimum amount of supporting fibrous tissue. Thus the fully developed papilloma appears like a red sea anemone with delicate tentacles eddying to and fro with each and every movement of the bladder contents.

Diffuse papillomatosis is a term used to signify a condition in which multiple small growths are disseminated over a relatively wide area of the bladder. Pedicle formation is not so pronounced as in the solitary variety. To justify a diagnosis of diffuse papillomatosis a stage must have been reached when multiplicity has outstripped the ability to keep the bladder clear by endoscopic destruction of the neoplasms.

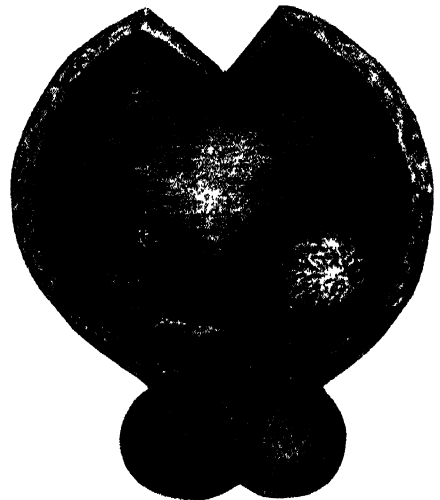


FIG. 1503.—Papillary tumour with daughter implantations ('kiss cancer').

¹ Workers exposed to this hazard should have a sample of urine centrifuged and examined for red cells and neoplastic cells at three-monthly intervals.

Malignant Villous Tumours.—Low-grade transitional cell carcinomas have exactly the same macroscopic appearance as the benign villous growths. As the degree of differentiation decreases, the appearance of the growth alters: (a) the villi are stunted, closely packed, and swollen, resembling a cauliflower; (b) the growth is sessile (90 per cent. of villous tumours are pedunculated); (c) the bladder wall immediately adjacent is more vascular and œdematous; (d) the surface of the growth may ulcerate, show areas of necrosis, or become encrusted with urinary salts; (e) a bladder tumour accompanied by cystitis is nearly always malignant; (f) submucosal lymphatic

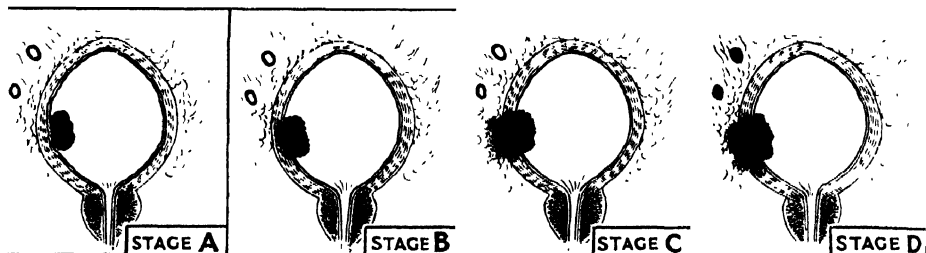


FIG. 1504.—The five stages of progression of a malignant papilloma of the bladder. Stage A, mucosal. Stage B, muscular. Stage C, peri-vesical. Stage D, fixity, with deposits in lymph nodes. Stage E (not shown), distant metastases occur. (After C. Dukes and F. Masina.)

nodules appear around the growth; (g) the depth of invasion of the bladder wall increases, and ureteric obstruction may occur.

Multiplicity of the growth is not necessarily a sign of malignancy, provided the daughter growths are limited to two or three and the rest of the bladder mucous membrane is normal. When this number is exceeded and/or a patch of œdematous vascular mucosa is seen the growths must be deemed malignant. Unablated, this neoplasm, like other forms of vesical carcinoma, invades the bladder wall (fig. 1504) and gives rise to regional and distant metastases.

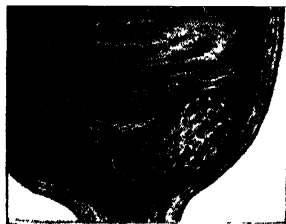


FIG. 1505.—Nodular carcinoma of the bladder, sometimes known as the 'bun-shaped' tumour.

Solid tumours are always malignant and grow rapidly through the bladder wall. The surface, instead of being tufted, is bald from the outset. The tumour, which is sessile, becomes lobulated (fig. 1505), is deep red, and bleeds on being touched. It is liable to become covered with a powder of phosphates. Later the surface becomes ulcerated in places. Lymphatic metastases occur earlier than is the case in malignant villous tumours. Solid carcinoma is the second most

common form of carcinoma of the bladder.

A **carcinomatous ulcer** is similar in appearance to a carcinomatous ulcer elsewhere, and occasionally it arises in a patch of leukoplakia. This type of growth occurs in the base of the bladder and on the trigone. This is the most malignant carcinoma of the bladder. The frequency of carcinoma at various sites is shown (fig. 1506).

Primary adenocarcinoma of the bladder is rare (1 per cent. of cases). It is a highly malignant tumour commencing in the fundus of the bladder of relatively young patients, and is believed to arise in the remnants of the urachus. Secondary adenocarcinoma of the bladder from a primary neoplasm in the stomach, colon, lung, rectum, or prostate is more common than one originating in the bladder.

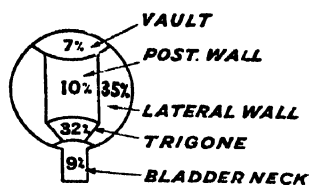


FIG. 1506.—Sites of carcinoma of the bladder. (American Urological Association Statistics.)

Metastases.—When a carcinoma has invaded the muscle coat (but no further) it is doubtful if metastasis occurs, though local recurrence is common. On the whole, vesical neoplasms are slow to metastasise and the incidence of distant metastases is relatively low, irrespective of the histological characters of the tumour. On the other hand, once the neoplasm has penetrated the musculature and has reached the prevesical fat, it disseminates by the lymphatics, to reach the lymph nodes along the external iliac and common iliac vessels, thence to the para-aortic and mediastinal lymph nodes. By the blood-stream they can be swept to the lungs, liver, bones or the brain.

Clinical Features.—*Papilloma.*—Painless, profuse, paroxysmal hæmaturia is the only symptom for a long period. The hæmaturia may last for a few hours or a few days, and then it ceases. After an interval of weeks or months, bleeding recurs; as time progresses the intervals become shorter and the hæmaturia more severe and of longer duration. Eventually the resulting anæmia may be so severe as to necessitate blood transfusion. Clot retention sometimes occurs, as also dysuria if the growth obstructs the internal urinary meatus. A slight ache in one loin is not uncommon, and usually signifies a commencing hydronephrosis. Occasionally, when papillomas are multiple or large, there is increased frequency of micturition from a feeling of incomplete emptying of the bladder. Bimanual rectal or vaginal examination is entirely negative. In 80 per cent. of cases painless hæmaturia remains the only symptom and sign.

Malignant Neoplasm.—In 95 per cent. of cases the initial symptom is intermittent hæmaturia, which eventually becomes continuous and associated with dysuria. It is a sad fact that in spite of this ominous sign more often than not twelve or more months elapse before the patient presents for treatment, and this is not always the patient's fault. Occasionally (in carcinomatous ulcer) the first symptoms are those of severe cystitis. In all cases sooner or later cystitis supervenes, and painful, frequent, blood-stained micturitions are the dominating symptoms in established cases. As time goes on, strangury occurs at the end of each act of micturition. A late manifestation, due to nerve involvement, is pain referred to the suprapubic region, the groins, perineum, the anus, or down the thighs.

Investigation:

Examination of the Urine.—If necessary the urine is examined for microscopical evidence of blood. A midstream specimen should always be sent for culture.

Excretory Pyelography.—Occasionally the preliminary plain film shows a faint shadow of an encrusted neoplasm of the bladder. As a rule in cases of papilloma, excretory pyelography displays normal kidney function. When the neoplasm is obstructing a ureteric orifice, dilatation of that ureter will be apparent. In the case of a large neoplasm a filling defect of the bladder (fig. 1507) is likely to be revealed.

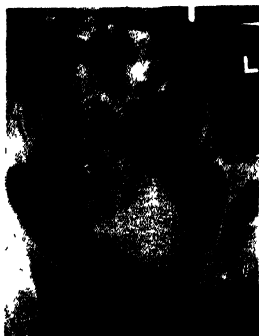


FIG. 1507.—Cystograph in a case of malignant tumour of the right side of the bladder.

Cystoscopy is the mainstay of diagnosis. It should be performed in every case of hæmaturia. The main cystoscopic appearances of bladder tumours have been described under 'Pathology' (p. 1149).

Following cystoscopy, but before any trans-urethral operation or biopsy is undertaken, the following important examination is made.

Bimanual palpation under general anaesthesia (recto-abdominally in the male and vagino-abdominally in the female), with the patient's abdominal musculature completely relaxed and the bladder absolutely empty, is of salient importance in determining the stage of progression of the neoplasm and the potentialities of a cure by any given method. When a tumour is impalpable, it is most unlikely that it has invaded the deeper layers of the bladder wall. While a very large papilloma sometimes can be felt, the tumour is mobile within the bladder (fig. 1508 (A)). When a localised but definite thickening can be felt, but which is as movable as one would expect the bladder to be, it is

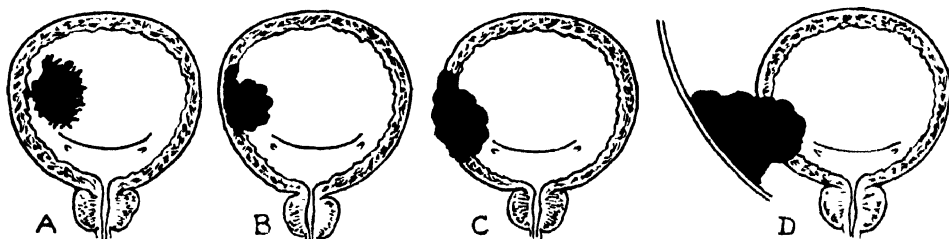


FIG. 1508.—Clinical assessment of the stage of progression of a bladder tumour by bimanual palpation. (After D. M. Wallace.)

probable that the bladder wall is infiltrated, but not penetrated (fig. 1508 (B)). On the other hand, if in addition the mass is larger than would be expected from the cystoscopic appearance but is not fixed, extravesical spread (fig. 1508 (C)) has certainly occurred. When a mass is easily palpable, fixed to the bony pelvis (fig. 1508 (D)) and firm to stony-hard in consistency, palliative measures alone are advisable (see *Mist. Euphoria*, p. 61). The degree of infiltration, as judged by bimanual palpation under anaesthesia, is a more valuable criterion of potential curability than the degree of malignancy revealed by histological studies.

Biopsy.—The removal of a tiny piece of tissue is useless in most instances—the biopsy must be adequate, should include the underlying vesical wall itself, and is best

taken with a resectoscope. Alternatively, special biopsy forceps (Riches or Lowsleys), or cystoscopic rongeur forceps (fig. 1486) may be used.

Treatment:

While the treatment of a solitary pedunculated papilloma is standardised except for comparatively minor details in technique, the same being true if a few seedlings are present, as we proceed from the probably malignant to the undoubtedly malignant bladder tumour, so the gulf of controversy as how best to treat the neoplasm widens. At the present time vacillation between ultra-radical operations and wholly conservative measures is to some extent influenced, on the one hand, by the appearance of new isotopes or new methods of applying them, and, on the other, by better methods of diverting the urinary stream after removal of the bladder.

Cystodiathermy.—The widely employed method of destroying benign papillomas is coagulation diathermy applied by an electrode through an irrigating cystoscope. A small solitary papilloma can be treated satisfactorily employing local anaesthesia of the urethra, the patient being ambulatory. For larger growths a general or spinal anaesthetic is given which permits more massive fulguration, but necessitates in-patient treatment. If daughter growths are present, they are fulgurated first. It is of great importance to attempt eradication of the growth at the first treatment.

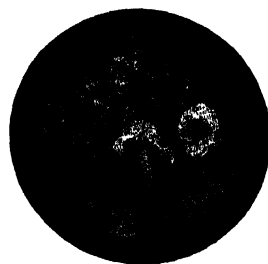


FIG. 1509.—Cystodiathermy of a papilloma of the bladder.

The patient is cystoscoped again in twelve weeks by which time necrotic tissue should have sloughed off, and the area epithelialised. Further treatment is carried out if necessary. Larger papillomas can be destroyed more quickly by Kidd's diathermy cystoscope, which has a relatively large ball electrode incorporated on the beak of the instrument, viz.

Excision with a resectoscope is indicated when the tumour is too large to be destroyed at one session of cystoscopic electro-coagulation, and especially for small papillary carcinomas with little or no infiltration. The instrument used is the McCarthy resectoscope (p. 1168). After the main part of the tumour has been sheared away, electro-excision of the base flush with the bladder wall is accomplished. Cautiously a slight gutter is then made beneath the base of the tumour, into the muscle. Avoidance of downward pressure minimises the risk of perforation of the bladder—the leading danger of this excellent method.

Transvesical Excision of a Large Villous Growth.—The bladder is distended with 1 : 2,000 silver nitrate solution and the viscus is exposed

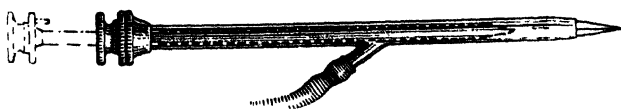


FIG. 1510.

by a suprapubic incision. The contents of the bladder having been evacuated by a trocar and cannula (fig. 1510), which permits suction of the fluid contents so that none is spilled, the edges of the abdominal wall are covered with packs soaked in silver nitrate solution or 60 per cent. alcohol, as a precaution against contamination of the wound by detached tumour cells. The bladder is opened, and the surface of the tumour is lightly coagulated (with the object of preventing detachment of cells). It is then picked up with toothed dissecting forceps as near the bladder wall as possible.

Francis Seymour Kidd, 1878-1931. Surgeon-in-Charge of the Genito-Urinary Department, The London Hospital, Joseph Francis McCarthy, Contemporary. Professor of Urology, New York Polyclinic Medical School, New York, U.S.A.

An innocent tumour can be elevated so that there is a substantial pedicle of normal mucous membrane. This is transected with the diathermy needle. After coagulating bleeding-points, the area from which the papilloma sprang is electro-coagulated. In the case of a sessile neoplasm, a diathermy loop electrode (fig. 1511) allows excision of the tumour including a fraction of the muscle layer. This accomplished, the cut surface is lightly fulgurated. The bladder can then be closed with indwelling urethral catheter drainage. Drainage of the prevesical space is necessary.



FIG. 1511.—Transvesical excision of a bladder tumour with diathermy loop electrode. (After A. R. C. Higham.)

Follow-up.—After the papilloma has been destroyed, endoscopically or by open operation, it is essential that the patient be examined cystoscopically, commencing at three-monthly intervals and, if there is no recurrence, gradually lengthening the interval to one year. In 30 per cent. of cases there is no recurrence. In the remainder there are recurrences, either at the site of the original lesion or in another part of the bladder, but as a rule they can be kept under control with regular cystodiathermy.

Local Radiotherapy.—When the lesion is 4 cm. or less in diameter, and when there is evidence of local invasion either on the pyelogram (hold-up or dilatation of one ureter), or on bimanual examination, it is wise to add local irradiation to the open diathermy excision described above. This may be achieved with radioactive gold grains or tantalum wire, inserted after the initial breach in the mucosa has been closed with 3/0 chromic catgut sutures.

(a) **Implantation of Radioactive Gold Grains (^{198}Au).**—The gold grains are inserted by means of a repeater gun, which is loaded with a cartridge containing fourteen grains. This isotope has a half life of two and a half days. The grains are implanted according to physical rules depending on the area to be irradiated. Stereoscopic films taken subsequently enable the physicist to determine the dosage the tumour will receive.

(b) **Radioactive tantalum wire (^{182}Ta) (Wallace) (fig. 1512)** has a long half-life (four months), and it therefore can be stored in the hospital ready for use when required, instead of having to be obtained from an atomic pile for each patient. The wire is bent hair-pin fashion with a loop provided at the blunt end. Through the opened bladder the sharp ends of the wire are drawn beneath the base of the tumour by means of twin, hollow needles, mounted on a boomerang needle introducer which enters and emerges some millimetres from the periphery of the neoplasm. The wire having been inserted, a plastic or rubber catheter is passed from the external urinary meatus and its tip is attached to the loop of wire by a stitch. The bladder is then closed. Again, stereo-radiographs enable the physicist to determine how long the wire should be allowed to remain in position. They are removed by withdrawing the catheter.

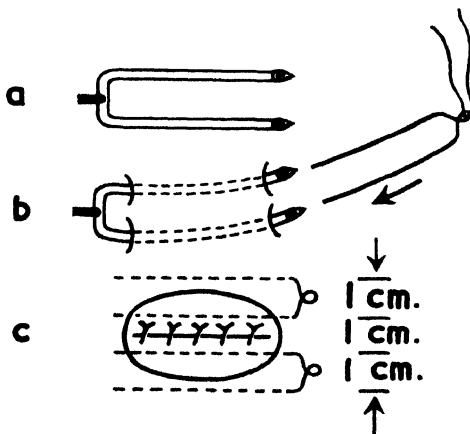


FIG. 1512.—Insertion of radioactive tantalum wire. (a) The curved twin hollow needles. (b) Insertion of the wire loop via the curved needles. (c) Position of wires relative to site of tumour pedicle.

Deep X-ray Therapy.—The advent of penetrating radiation such as Cobalt 60, and the linear accelerator which enables a high depth dose to be obtained easily, has greatly improved this method of treatment. It is now possible to irradiate a limited part of the pelvis to a high dose level and largely spare the rest.

Undifferentiated carcinomas are best treated by this method and the local results are now good. It is desirable, however, that the tumour should be accurately localised, employing cystography.

Excision of a Part or the Whole of the Bladder:

Partial Cystectomy (*syn.* Segmental Resection).—Its disadvantage is that many malignant tumours of the bladder are situated near the trigone, where the operation is inapplicable. That part of the bladder containing the growth, together with a surrounding inch of the whole thickness of healthy bladder wall, is excised. When the neoplasm is situated near a ureteric orifice, the ureter should be divided before the excision is carried out, and after the excision has been completed, transplantation of the ureter into the bladder is performed. The operation is of most value in small lesions involving muscle in the vault of the bladder.

Subtotal Cystectomy.—Like total cystectomy, the operation is conducted intraperitoneally. The whole of the bladder is mobilised to the entrance of the ureters into the bladder wall posteriorly and to within a finger's-breadth of the internal urinary meatus anteriorly. A new bladder is then constructed, utilising a segment of ileum with its blood-supply intact, the graft being split along its length. This operation has a very limited application; usually a growth extensive enough to necessitate it has encroached on the trigone.

Total Cystectomy.—The indications for total cystectomy are somewhat controversial. Provided the patient is fit to withstand this very severe operation it would be justifiable to undertake it in the following circumstances: (1) Profuse papillomatosis uncontrolled by other methods; (2) Multiple tumours of high-grade malignancy, believed to be confined to the bladder; (3) Carcinoma involving the bladder neck in either sex; (4) Recurrences in a small contracted bladder that has resulted from oft-repeated conservative operations; (5) Recurrence after partial cystectomy; (6) Recurrence or residual growth after adequate radiotherapy.

Operation.—With the bladder empty, left lower laparotomy is performed, and the incision is carried down far enough to expose the symphysis pubis. The peritoneum having been opened, the iliac and para-aortic lymph nodes and the liver and the kidneys are palpated. Having decided to proceed, the operating table is tilted and the ureters are isolated and divided as low as possible in the same way as described on p. 1159. A suitable catheter is passed along the lumen of each ureter, and the urine is allowed to drip into a bottle placed away from the operation area. Hæmorrhage is reduced by ligating the internal iliac arteries.

The bladder and its overlying peritoneum, together with the prostate and all the fascia and fat surrounding these structures (including the uterus in the female), and the greater part of the urethra, are removed *en bloc*.

One of the most important steps of the operation is to find the correct plane of cleavage between the prostate and the rectum. This is accomplished by following the vas deferens proximally. *En route* this will lead to the superior and inferior vesical vascular pedicles, which are ligated and divided.

For total cystectomy to be truly radical, the lymph vessels and nodes of the pelvis at least up to the bifurcation of the common iliac artery should be included, but of necessity this increases the operating time.

Diversion of Urine.—For methods of diversion see below.

URINARY DIVERSION

This chapter closes with an account of the principles of this important subject, and includes indications, the methods employed and their attendant problems, and some operative details.

Indications.—Diversion of the urine may be either (a) a temporary expedient to relieve distal obstruction, or (b) a permanent procedure when (1) the bladder has been removed, (2) the sphincters of the bladder have been

damaged or have lost their normal neurological control, (3) there is an incurable vesico-vaginal fistula, (4) there is an irremovable obstruction, and (5) in cases of ectopia vesicæ (p. 1122).

Methods of Urinary Diversion (fig. 1513).

The diversion may be achieved by any of the following methods, but the choice in each case will be decided largely by the primary disease.

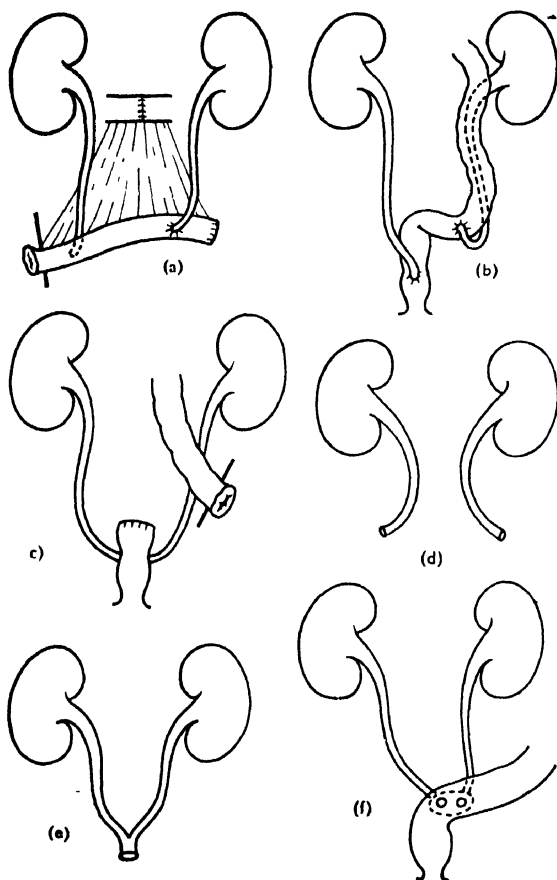


FIG. 1513.—Diversion of urine. Favoured methods: (a) Ileoconduit, (b) Uretero-sigmoidostomy, (c) Rectal bladder with terminal colostomy, (d) Bilateral cutaneous ureterostomies, (e) Joined ureters—cutaneous opening, (f) Trigono-colostomy.

Pyelostomy or nephrostomy, or urethrostomy (with indwelling catheters).

Cutaneous ureterostomy.

Suprapubic cystostomy (with in-dwelling catheter).

Cutaneous vesicostomy (cystostomy).

Suprapubic displacement of the urethra.

Uretero-sigmoidostomy (a) in continuity, (b) making a rectal bladder and colostomy.

Trigono-colostomy (a) in continuity, (b) into an isolated conduit.

Ureteric transplantation into an isolated ileal or colonic conduit.

Diversion of urine immediately raises the following problems:—1. the collection of the urine, 2. stricture-formation at any anastomosis, and 3. reflux and reabsorption of urinary solutes. The problems of infection are intimately related to all three.

Collection of the Urine

(a) **Catheters.**—In the past, indwelling catheters

have been used for permanent diversion. They invariably result in infection and they often become blocked by phosphatic encrustation. There is now *no place for permanent drainage* of the kidneys or ureters. For temporary drainage the tubes should be of latex or a polyethylene plastic (polyvinyl plastics are irritant and predispose to a stricture).

Bladder Drainage.—In elderly patients unfit for prostatectomy and in some terminal cases of carcinoma of the prostate, an indwelling Foley catheter (fig. 1471), changed every three weeks, is a satisfactory method of drainage.

Alternatively, a St. Peter's Hospital suprapubic drainage apparatus (fig. 1514) incorporating a straight catheter which the patient or nurse changes daily is acceptable.

(b) **Cutaneous Stomas: Suprapubic Vesicostomy and Urethrostomy.**—Collection from a formal suprapubic vesicostomy (cystostomy), or from the urethra displaced on to the abdominal wall (e.g. for cases of multiple sclerosis in a woman), may be unsatisfactory because the local incisions result in creases which make it difficult to apply a water-tight collecting appliance.

Cutaneous ureterostomies are very liable to stricture formation. In addition two openings (fig. 1513(d)) and appliances add to the patient's burden. Mobilisation of the ureters and the making of a central abdominal stoma (fig. 1513(e)) can be successful, but such a degree of mobilisation is attended by the high risk of sloughing of the distal ureters due to impairment of blood-supply.

Ileal or Colonic Conduit.—At present the most generally useful form of diversion is to implant each ureter with as little mobilisation as possible into an isolated segment of gut (ileum or colon), which conducts the urine onwards to a cutaneous stoma (fig. 1513(a)). Urine is then collected in an ileostomy bag. This form of diversion limits infection and avoids the problems of reabsorption of urine as contact-time with the mucosa is minimal.

Siting of Stoma.—The site for the stoma must be chosen before operation. The ileostomy ring and bag are applied to the abdominal wall, and by trial and error the point at which the belt can support the ring and bag at all times and in all positions is found. Having checked this by sticking on a bag full of water and asking the patient to perform the type of movements which his work will entail, the site of the future stoma is marked indelibly on the skin.

‡(c) **Colon and Rectum.**—The advantage of diverting urine into the colon is that no collecting apparatus is necessary (fig. 1513(b) and (f)). Clearly, however, the rectal sphincter must be competent. Before any uretero-sigmoidostomy is undertaken, the patient must prove that he can control at least six ounces (175 ml.) of fluid in the rectum. The disadvantage of the operation is that the renal tract is exposed continuously to infection from the fæces. This can be avoided by establishing a terminal left iliac colostomy, and closing the upper rectum to make a rectal bladder (fig. 1513(c)). It prevents the urine refluxing retrogradely round the colon to the caecum, diminishes reabsorption (see below), and protects renal function.

Stricture Formation

Uretero-sigmoidostomy was first used by Chaput (1894). Subsequent modifications included those made by Coffey and Grey-Turner. In these methods the ureters were cut obliquely and pulled into the gut by a stitch—the ends were not stitched to the gut wall. Stenosis was not uncommon. Nesbit, Cordonnier, and

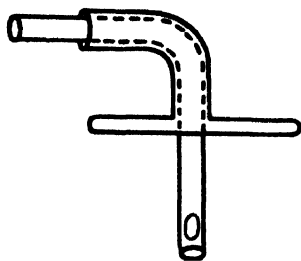


FIG. 1514.—St. Peter's Hospital suprapubic drainage apparatus.

Leadbetter all recognised that these strictures could be prevented by anastomosing mucosa to mucosa (fig. 1515).

Reflux of Urine and Reabsorption of Urinary Solutes

Reflux of Urine.—Pressure within the colon can interfere with drainage, and so force air, faeces, and organisms up to the renal pelvis. Daniel has demonstrated that a high intra-colonic pressure (over 24 cm. water) results in all these complications being more likely. For many years it had been noted that the left kidney was much more likely to cease functioning after uretero-sigmoidostomy than the right kidney. As the left kidney is implanted into the sigmoid and the right often in the upper rectum, the former is more likely to be subjected to higher intra-colonic pressures. A sigmoid myotomy performed from the upper rectum to the splenic flexure may help to overcome this problem.

Reabsorption of Urinary solutes depends upon two factors, (1) reflux of urine round the colon and even into the terminal ileum (fig. 1513 (b) and (f)), (2) because the colon is acting as a reservoir, the urine is in contact with gut wall for a longer period. Biochemical disturbances are rare if a rectal bladder is made (see above).

The biochemical changes associated with transplantation into the colon are due to a combination of reabsorption of chlorides and urea, and progressively diminishing tubular function as a result of chronic pyelo-nephritis. Diarrhoea with loss of potassium-containing mucus may exacerbate the loss of potassium.

The typical changes are a hyperchloræmic acidosis with potassium depletion, and they occur in every patient with a uretero-sigmoid diversion. When severe, the patient develops loss of appetite, weakness, thirst, and diarrhoea. He becomes listless, and respirations are rapid, deep, and unremitting. Coma may ensue. Mild acidosis, unrecognised over a long period, produces osteomalacia from calcium reabsorption. Bone pain and pathological fractures are common.

Renal impairment from pyelonephritis and reabsorption from the mucosa are infrequent complications if the diversion is into an ileal or colonic conduit.

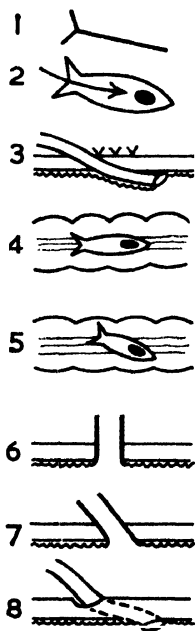


FIG. 1515.—Methods of uretero-colic anastomosis. 1. Incision in the sero-muscular layer, which, on dissection, exposes an area of submucosa and mucosa which looks rather like a fish. 2. The ureter is laid along the 'fish' and inserted through the opening into the lumen made in the 'fish's head'. 3. Lateral view of completed anastomosis. 4. Coffey type. 5. Grey Turner type. 6. Nesbit, 7. Cordonnier, and 8. Leadbetter types entail a careful mucosa to mucosa anastomosis. The last named includes a submucosal tunnel, and is probably the best anastomosis for preventing stricture formation and reflux.

Treatment.—(a) *Prevention.*—Patients should be instructed to empty the rectum two-hourly by day, and in cases where acidosis is present, a rectal tube should be inserted at night to drain the urine continuously. The patient should avoid added salt in the diet, and he should take a mixture of potassium citrate and sodium bicarbonate t.d.s. (two grams of each either as crystals or tablets). Regular biochemical analyses, including calcium, are required. The patient should be told to report if he loses his appetite or develops weakness.

(b) *In established hyperchloræmic acidosis*, the patient should be given suitable doses of sodium bicarbonate (8.4 per cent. = 1 meq./ml.) intravenously to correct the pH (p. 96), with suitable amounts of potassium, also intravenously, if necessary. Furadantin, ampicillin, or the appropriate antibiotic should be

given to correct the inevitable pyelonephritis which will be present. Later, conversion to an ileal conduit should be considered.

Operative Details

Uretero-colic Anastomosis.—The preparation of the colon is the same as that described on p. 921. The abdomen is opened by a right lower paramedian incision.

The patient is then placed in the Trendelenburg position and the right ureter is sought as it crosses above the bifurcation of the common iliac artery. An incision is made through the peritoneum overlying the medial side of the ureter, thus avoiding damage to the ureteric vessels which always approach the duct from the outer side (Daniel), and the ureter is identified and dissected from its bed towards its entry into the bladder. The ureter is then divided and its distal stump ligated. The proximal end is trimmed obliquely and split upwards for one centimetre. An incision 3 cm. long is made in the anterior wall of the bowel and the peritoneal and muscular coats are divided, but not the mucous membrane (fig. 1515(3)). An incision is made into the extreme lower end of the exposed mucous membrane and the full thickness of the ureteric wall is joined by interrupted 4/0 chromic catgut sutures to the mucosal opening. The incision in the outer coats of the bowel is approximated over the ureter (Leadbetter anastomosis). Suturing the peritoneal incision around the ureteric implant extraperitonealises the site, and a drain is left down to the area. The left ureter is implanted into the colon above in a similar manner. A full-sized Foley's catheter (fig. 1471) is inserted through the anus no farther than the rectal ampulla, the balloon then being inflated. This permits all urine to be measured until the third day, when the catheter is removed. There must be a full fluid intake. If oliguria or anuria supervene, treatment is as described on p. 1067.

Ileo-ureterostomy (Ileal Loop Conduit).—A coil of ileum, approximately 6–9 inches (15–25 cm.) long, and one foot from the ileo-cæcal valve, with its blood supply intact, is isolated. The left ureter is brought through the pelvic mesocolon, and after the ureter has been cut obliquely, end-to-side anastomosis is performed between the ureter and the intestine. The right ureter is also cut obliquely, and it is joined to an elliptical opening made in the side of the coil. In each case the anastomosis may be effected by the method of Nesbit (fig. 1515(6)). The coil of intestine is tacked lightly to the peritoneum of the anterior abdominal wall at the level of the pelvic brim. The distal end of the coil is brought out through a stab incision in the right lower abdomen, and, because of the anchoring of the intestine to the peritoneum, the opening is made a little lower than is the case when ileostomy is performed for ulcerative colitis. The method of anchoring the ileostomy opening to the abdominal wall, and the after-care of the orifice so formed, in no way differs from that described on p. 907.

Lowsley's Operation.—The rectosigmoid junction is divided completely and its lower end closed. The ureters or the trigone are implanted into the excluded lower segment, so that the rectum functions as a bladder. The sigmoid colon is mobilised sufficiently for it to be drawn through the perineum immediately anterior to the anus. Thus both the original anal canal and the new anal canal are encompassed by the external sphincter ani muscle (fig. 1516).

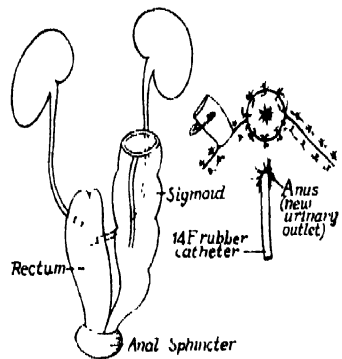


FIG. 1516.—Lowsley's operation.

CHAPTER 47

THE PROSTATE AND SEMINAL VESICLES

Embryology.—The prostate arises from the primitive urethra as a series of solid buds (fig. 1517); in a matter of weeks these become canalised. Budding takes place into the surrounding mesenchyme, which become the muscular and connective tissue of the gland. The buds are arranged into five groups—anterior, middle, posterior, and two lateral. These are the forerunners of the lobes of the prostate.

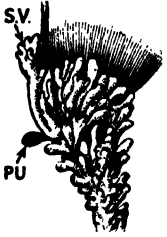


FIG. 1517.—The prostate towards the end of the fourth month of intra-uterine life. SV = seminal vesicle, PU = prostatic utricle. (After E. J. Boatt.)

Skene's tubules opening on either side of the female urethral orifice are the homologues of the prostate.

Surgical Anatomy.—The prostatic glands (fig. 1518) lie in a fibromuscular stroma and their ducts open into the posterolateral grooves on either side of the verumontanum. The epithelium is columnar. Commencing peripherally, and passing centrally, beneath the anatomical capsule lie the long branched **prostatic glands proper**. This region is named the **carcinomatous zone**. Beneath this envelope,

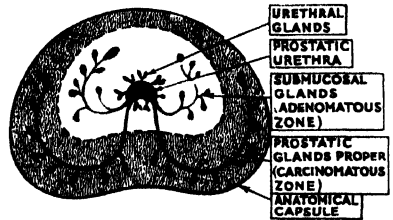


FIG. 1518.—Distribution of the normal glands of the prostate, transverse section. (After J. C. B. Grant.)

and separated from it by an indefinite capsule, lies another mass of secreting elements, also branched. These are the **submucosal glands**—and the zone that they occupy is known as the **adenomatous zone**. Still nearer the urethra are the unbranched

urethral glands whose mouths open directly into the urethra.

Into the prostatic urethra, therefore, open the prostatic ducts proper, the ducts of the submucosal and mucosal glands, as well as the common ejaculatory ducts and the prostatic utricle. No wonder that chronic infection of the prostatic urethra is difficult to eradicate!

Certain relationships and divisions of the prostate as seen on sagittal section are set out in fig. 1519. The middle lobe is that part of the prostate included between the common ejaculatory ducts and the prostatic urethra: it contains more secretory glandular element (as opposed to muscle and fibrous tissue) than the lateral lobes.

An enlarged prostate is invested with three capsules: (1) The compressed outer zone = *the false capsule*; (2) The anatomical capsule = *the*

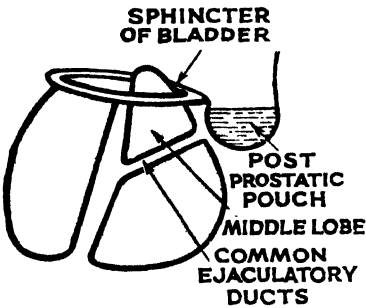


FIG. 1519.—The surgical subdivisions and relationships of the prostate.

true capsule; and external to this (3) *The prostatic sheath of pelvic fascia*. Between the anatomical capsule and the prostatic sheath lies the prostatic venous plexus (fig. 1520). The prostatic sheath is contiguous with the strong fascia of Denonvilliers that separates the prostate and its coverings from the rectum.

Physiology.—That the prostate is purely a genital organ is evinced by the fact that in such animals as manifest a seasonal sexual life, the organ is rudimentary except during the rutting season. That normal adult prostatic epithelium undergoes atrophy after castration was known to John Hunter.

Hormonal Influences.—The prostate is governed by two testicular hormones, one male (androgenic) and the other female (œstrogenic). Normally the preponderant testicular hormone is androgen, which is supplemented by androgens secreted by the adrenal glands (fig. 1521).

œEstrogens cause retrogressive changes in the testes and prostate gland. For example, most capons are produced by implanting œstrogen pellets into the neck of the bird rather than by castration.

Elaboration and Secretion of Acid Phosphatases.—Enzymes that split organic phosphates and are most active about pH5, are present in many human tissues, but their concentration in the adult prostate is several hundred times greater than in any other organ or tissue. This high level is not achieved until after puberty.

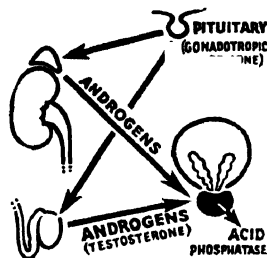


FIG. 1521.—Preponderant hormonal control of the prostate. (After L. N. Pyrah, F.R.C.S., Leeds.)

acid phosphatase level. The normal range of findings is 0.5 King-Armstrong units per 100 ml. of serum.

The serum acid phosphatase is usually raised in carcinoma of the prostate *with metastases*, but is seldom above normal if the growth is confined to the gland, and almost never in benign prostatic hyperplasia. On the other hand, slightly increased values are not uncommon (1) in acute prostatitis, (2) after prostatic massage, (3) Paget's disease of bone; (4) hepatic cirrhosis and (5) retention of urine.

BENIGN ENLARGEMENT OF THE PROSTATE

"When hair becomes grey and thin, when atheromatous deposits invade the arterial walls, when there has formed a white zone about the cornea, at the same time, ordinarily—I dare say invariably—the prostate increases in volume." (SIR BENJAMIN BRODIE.)

Benign enlargement of the prostate usually occurs in men¹ over fifty years of age; most often between sixty and seventy. In Indians prostatic enlargement is less frequent, and occurs more often in a younger age-group; in Negroes it is rare, while in Asiatics it is exceptional. The reason for these discrepancies is unknown.

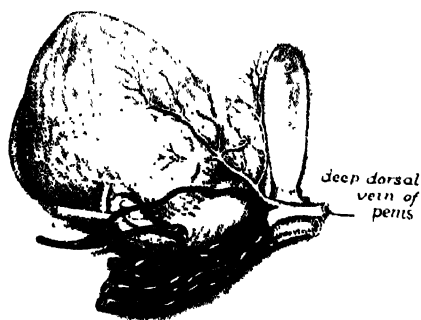


FIG. 1520.—The prostatic venous plexus.

While the function of this enzyme is speculative, it is highly suggestive that the acidity which is most favourable for its action corresponds to that of the vagina at the time of ovulation. Most of the acid phosphatase secreted by the prostate drains along the prostatic ducts into the urethra, so that the blood level of this enzyme remains low. When the cells producing this enzyme cannot discharge their products externally, the serum acid phosphatase rises.

Acid phosphatase estimations are made on plasma or serum, free from hæmolysis. The blood sample must be taken in the morning before breakfast, and on no account after a heavy meal, since lipoid-rich serum gives inaccurate estimations. For forty-eight hours prior to taking the sample of blood for phosphatase determination, rectal examination must be avoided since digital palpation of the prostate is liable to produce a transient rise in the serum

¹ It is interesting to note that the ageing dog is prone to prostatic hyperplasia but, unlike his master, does not often develop retention of urine. Due, however, to the bulging upwards of the enlarged prostate the dog's rectum is compressed. This engenders a feeling of continuous fullness in the rectum, and induces painful straining; symptoms are relieved by stilboestrol. The prostates of eunuchs are small and completely under-developed, and benign enlargement of the prostate is unknown.

Theories of causation of benign enlargement of the prostate :

The Hormonic Theory.—As age advances the male hormone diminishes while the quantity of the œstrogenic hormone is not decreased equally. According to this theory the prostate enlarges because of predominance of the œstrogenic hormone. Prostatic enlargement can be regarded as involutionary hyperplasia akin to fibro-adenosis of the breast, due to a disturbance of the ratio and quantity of the circulating androgens and œstrogens.

The Neoplastic Theory postulates that the enlargement is a benign neoplasm. As the prostate is composed essentially of fibrous tissue, muscle tissue, and glandular tissue, the neoplasm is a *fibro-myo-adenoma*. It follows that should the glandular element (adenoma) predominate, that part of the gland which is richest in secretory glandular tissue, to wit the middle lobe, will be maximally affected. When the fibrous element is most in evidence, the small, hard fibrous prostate is produced.

Pathology.—Hyperplasia affects the glandular elements and connective tissue, but in variable degrees. In this respect the changes are very similar to those occurring in the breast in 'chronic mastitis', where adenosis, epitheliosis, and stromal proliferation are seen in differing proportions. Benign adenomatous hyperplasia affects the submucous group of glands, forming a nodular enlargement—the typical 'lateral' lobes—which compresses the external group of glands into a false capsule (fig. 1518).

As the prostate enlarges extravasically, it tends to displace the seminal vesicles, so that instead of lying on the base of the bladder, these structures become a direct posterior relation of the upper limit of the prostate. When the hyperplasia affects the subcervical glands a 'middle' lobe develops which projects up into the bladder within the internal sphincter (fig. 1519). Sometimes both lateral lobes also project into the bladder, so that when viewed from within, the sides and back of the internal urinary meatus are surrounded by an intravesical prostatic collar.

SECONDARY EFFECTS OF PROSTATIC ENLARGEMENT

Urethra.—That portion of the urethra lying above the verumontanum becomes elongated, sometimes to as much as twice its normal length. The canal

is compressed laterally, so that it tends to become an antero-posterior slit. The normal posterior curve may be so exaggerated that it requires a coudé, bi-coudé, or even a fully curved metal catheter to negotiate it. When one lateral lobe is enlarged predominantly, lateral distortion of the prostatic urethra occurs.

Bladder.—The musculature of the bladder hypertrophies to overcome the obstruction. When viewed from within, bands of muscle fibres can be seen—*trabeculation* (fig. 1522). Between these hypertrophied bundles there are shallow depressions, i.e. *sacculations*. Some-



FIG. 1522. — Trabeculation of the bladder from prostatic obstruction. (The late Professor K. A. L. Aschoff, Freiburg.)

times one of the saccules (rarely two or more) continues to enlarge, and forms a *diverticulum*.

When the middle lobe projects upwards into the bladder it acts as a dam to the last ounce or more of urine, which remains in the post-prostatic pouch (figs. 1519/1523). Calculi (fig. 1523) are prone to form in this stagnant pool of urine.

The enlarged prostate may compress the prostatic venous plexus; the resulting congested veins (vesical 'piles') at the base of the bladder are apt to cause hæmaturia.

Unless the obstruction is relieved, a time is reached when bladder hypertrophy gives place to atony, the tired muscle making no attempt to overcome the obstruction.

Ureters and Kidneys.—Increasing intravesical pressure, or in some cases direct pressure of the intravesical portion of the prostate on the ureteric orifices, causes gradual dilatation of the ureters, followed by some degree of bilateral hydronephrosis. When bladder hypertrophy wanes, the sphincter mechanism around the ureteric orifices ceases to function, permitting reflux of urine from the bladder into the dilated ureters, with increasing damage to the renal parenchyma. As a result of ascending infection, or more rarely from the blood-stream, acute or chronic pyelonephritis supervenes.

Sexual Organs.—In the early stages of prostatic enlargement there is increased libido. Later, impotence is the rule.

Clinical features are variable and depend upon the lobes affected. *Frequency* is the earliest symptom. At first it is nocturnal, the patient being obliged to get up to micturate twice or more during the night, usually commencing at 2 or 3 a.m. Frequent micturition at this stage is probably due to vesical introversion of the sensitive prostatic mucous membrane by the intravesical enlargement of the prostate. The frequency becomes progressive, and is then present both by night and by day. When the vesical sphincter becomes stretched, a little urine escapes into the normally empty prostatic urethra, causing an intense reflex desire to void, and *urgency* is added to the frequency. Later, as residual urine increases, frequency becomes more and more in evidence, and there is terminal dribbling. Finally, in neglected cases, frequency is further encouraged by cystitis, and polyuria due to renal inefficiency.

Difficulty in Micturition.—The patient notices that he must wait patiently for urination to start; it is useless to strain (cf. urethral stricture and fibrous prostate—pp. 1193 and 1170).

The stream is variable, often weak, tending to stop and start, and dribble towards the end of micturition.

Pain occurs with cystitis or acute retention of urine. When hydronephro-



FIG. 1523.—Calculi in a post-prostatic pouch behind the hypertrophied middle lobe of the prostate as viewed through the cystoscope. (After H. H. Young.)

sis commences there may be a dull pain in the loins. A feeling of weight in the perineum, or a fullness in the rectum, are occasional complaints.

Acute retention of urine is sometimes the first symptom to compel the patient to seek relief because of the intense pain it produces. The postponement of micturition is a common precipitating cause (e.g. a non-corridor train), as also is indulgence in alcoholic liquors, particularly when he goes out of doors on a cold night, as congestion of internal organs then tends to occur. Confinement to bed on account of some intercurrent illness or operation is another cause of acute retention of urine (p. 1126).

Retention with Overflow.—The patient complains that urine constantly dribbles away. It is exceptional for him to have noticed the swelling caused by the distended bladder and he experiences no pain (fig. 1524).



FIG. 1524.—Retention with overflow. The patient's only complaint was that he 'wet his trousers'. Note the distended bladder.

Hæmaturia.—A drop of blood at the beginning or end of micturition is not unusual. Occasionally alarming hæmaturia occurs from a ruptured prostatic vein (p. 1163), or from an erosion on the enlarged prostate itself. Incrimination of an enlarged prostate as the source of hæmaturia before excluding other causes has resulted in the term 'decoy' prostate.

Renal Insufficiency.—The patient presents himself with signs of renal failure (p. 1067).

Examination.—The patient lies on a couch and the abdomen is examined. In patients with a long history, varying degrees of chronic retention of urine will be found on palpation, percussion, and sometimes on inspection. The renal areas are palpated for tenderness and possible enlargement of the kidneys. The state of the tongue is noted; a dry brown tongue and a urine of low specific gravity indicate a considerable degree of renal insufficiency. The external urinary meatus is ex-

amined to exclude stenosis, and the epididymes are palpated for signs of inflammation.

Rectal examination is carried out and, in the absence of a full bladder, bimanually in the dorsal position. In benign enlargement of the lateral lobes, increase in their size is evident. They are smooth, convex, and typically elastic, but because all grades of fibroadenomatosis occur, the fibrous element may give the prostate a firm consistency. The rectal mucosa can be made to move over the prostate. On bimanual palpation an intravesical lobe can sometimes be felt. By exerting pressure on the apex of the prostate by the finger in the rectum, it will be found that a gland which is the seat of benign enlargement possesses a definite degree of mobility. Residual urine in a post-prostatic pouch may be felt as a fluctuating swelling above the prostate. It should be noted that if there is a considerable amount of residual

urine present, it pushes the prostate downwards, making it appear larger than it is.

When possible, the act of micturition should be watched. Loss of projectile power is significant. The urine is passed into two glasses; mere inspection of it is often helpful. It is later examined chemically, and a mid-stream specimen may be sent for bacteriological examination.

The nervous system is examined by testing the reflexes and the reactions of the pupils, to eliminate a neurogenic lesion. *Tubes* and disseminated sclerosis may give symptoms similar to those of prostatic obstruction.

Examination of the Blood.—A blood urea estimation, a blood count, and a Wassermann reaction are all important, the first two being essential.

Excretory pyelography is omitted only if the patient shows clinical signs of renal failure, or if the blood urea estimation is high. It affords a great deal of information without resorting to instrumentation and it is an excellent test of renal function. The preliminary radiograph may show a renal calculus, or a stone in the bladder, or in a diverticulum thereof. Stones in a post-prostatic pouch are sometimes not evident until cystoscopy has been performed.

The pyclogram will exclude or confirm the presence of hydrourters and hydronephroses; a vesical diverticulum may be shown and if a film is taken after micturition, residual urine will be revealed. Sometimes an intravesical enlargement of the prostate is outlined by the medium. Nevertheless, complete reliance must not be placed on radiological evidence of residual urine as the patient may have difficulty in passing urine in the radiological department, especially if a female radiographer is nearby!

Cysto-urethroscopy.—Cysto-urethroscopy is an essential preliminary to prostatectomy, especially when the operation is not transvesical, to exclude the presence of a growth, a diverticulum, or non-opaque stones. It may also be necessary in order to make a diagnosis when the gland is small, and sometimes to distinguish between a urethral stricture and a bladder-neck obstruction. Cysto-urethroscopy should be delayed in the presence of infection, a distended bladder, or when there is mild renal impairment (blood urea between 40 and 100 mg. per cent.) until immediately before operation.

The patient must empty his bladder just prior to the examination, so that the amount of residual urine present in the bladder can be measured. After irrigation, if necessary, the bladder is distended and is first examined. Trabeculation and sacculation are the significant findings. The presence of cystitis will have been anticipated by the examination of the urine. Normally the posterior edge of the internal urinary meatus and a ureteric orifice cannot both be seen in the same cystoscopic field. In cases of moderate enlargement of the middle lobe this becomes possible (Marion's sign, fig. 1525). Should the intravesical projection be considerable, the ureteric orifices and the inter-ureteric bar are hidden completely beneath the prostatic shelf.

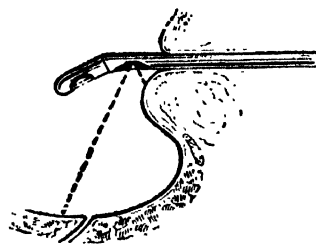


FIG. 1525.—Marion's sign.

THE MANAGEMENT OF RETENTION

Acute retention initially is very painful. Chronic retention is painless. It is safe to decompress an acute retention rapidly. It may not be safe to decompress a chronic retention rapidly. If the kidneys are hydronephrotic, reactionary hyperæmia may lead to bleeding from the kidney, impaired blood flow to the tubules, and an acute tubular necrosis. In chronic retention an urgent blood urea estimation is essential, and if above 100 mg. per cent. slow decompression should be the rule. Below this level it is better to avoid instrumentation until the cystoscopy immediately prior to prostatectomy.

Catheterisation.—Where there is a possibility that the catheter may be left indwelling, wash and shave the pubic, perineal, and scrotal regions. Anæsthetise the urethra with 1 per cent. Xylocaine combined with 0.5 per cent. chlorhexidine in a methyl-cellulose base. Allow three minutes for the anæsthetic and the antiseptic effects to take place. With strict aseptic precautions pass a size 8, 10, or 12 F. plastic catheter by a non-touch technique (Gibbon-type if it is to be left indwelling, p. 1130). Collect a specimen of urine for culture.

Slow decompression can be obtained by joining such a catheter to a bottle and arranging the end of the inlet into the bottle at such a height, 7 to 10 inches (18 to 25 cm.), above the pubis so that urine flows over only when the patient coughs or moves, thus raising his intra-abdominal pressure. The bottle can be lowered 1 inch (2.5 cm.) every four hours.

In cases of acute retention, especially where there is a history of overholding the urine, as on a long journey or after drinking alcohol, the bladder should be emptied and the catheter removed. Micturition may then be re-established. Failure to do so confirms the need for prostatectomy.

Suprapubic Puncture.—If there is a long history of prostatism, and early prostatectomy is decided upon, relief of pain can be achieved by suprapubic needle puncture, running in a length of polythene tube with multiple side holes and connecting this to a sterile drainage bottle or bag.

All catheters should be connected by straight connections to sterile tubes and drainage bottles, which should contain 4 ounces (120 ml.) of 40 per cent. formalin solution. Air vents should be protected by formalin tablets (fig. 1475).

INDICATIONS FOR OPERATION

(1) *Prostatism.*—Increasing difficulty in micturition, with considerable frequency day and night, delay in starting, and a poor stream—such are the usual symptoms for which prostatectomy is advised. It must be stressed that *frequency alone* is never an indication for prostatectomy, and also that the natural progression of benign prostatic enlargement is variable and rarely gets worse after ten years. A patient with symptoms for nine years is unlikely to get much more severe symptoms, while a similar degree of symptoms reached in three years is a greater indication for surgery. This fact may be helpful when considering the correct advice to give to elderly patients possibly suffering from other disease.

(2) *Acute retention which is unrelieved* by passing a catheter, emptying the bladder, and immediately removing the catheter.¹

(3) *Chronic Retention*.—A residual urine of 6 ounces (200 ml.) or more. A raised blood urea, hydroureter, or hydronephrosis demonstrated on urography.

(4) *Complications*.—Stone, infection, and diverticulum formation, which are often present in varying combinations.

(5) *Hæmorrhage*.—Occasionally venous bleeding from a ruptured vein overlying the prostate will not stop with catheter drainage, so prostatectomy must be performed as an emergency measure.

Operative Treatment

Until the commencement of the present century the accepted treatment of prostatic obstruction was a catheter-life. The patient passed the instrument himself, often lubricating it with saliva. The more affluent sufferers carried their catheters in their top hats. Nearly 20 per cent. of all sufferers died within three months. Some of the remainder acquired partial immunity to recurring bouts of infection.

Nowadays a permanent catheter, either urethral or suprapubic, is a rarity. Prostatectomy, or more correctly the removal of the adenomatous hyperplasia, by one of the four routes, is practicable in the great majority of cases.

The prostate can be approached (1) through the bladder (transvesical), (2) retropubically, (3) from the perineum, or (4) perurethrally (fig. 1526).

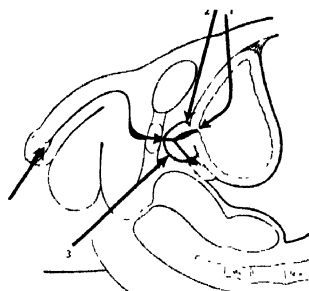


FIG. 1526.—The surgical approaches to the prostate.

Preliminary Vasectomy.—In all the operations about to be described, bilateral vasectomy (dividing the vasa deferentia) is advisable. It prevents infection from the prostatic bed spreading to the epididymes. This minor procedure can be undertaken as a preliminary step, or at the conclusion of prostatectomy. *Technique.*—Through a small incision over each superficial abdominal ring the vas deferens is isolated from the spermatic cord and a small piece resected, the ends being ligated with catgut.

Transvesical Prostatectomy.—The bladder is opened, the prostate enucleated by putting a finger into the urethra, pushing forwards towards the pubes to separate the lateral lobes, and then working the finger between the adenoma and the false capsule. In **Freyer's operation** (1901) the bladder was left open widely and drained



FIG. 1527.—A boomerang needle.

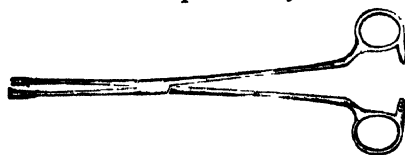


FIG. 1528.—Harris's ligature carrier.

by a suprapubic tube with a 16-mm. lumen, in order to allow free drainage of blood and urine. **Harris** (1934) advocated control of the prostatic arteries by lateral stitches inserted with his boomerang needle (fig. 1527 and 1528), and closure of the raw areas by stitching the mucosa of the posterior lip of the internal meatus to the urethral

¹ Wilson Hey recommended immediate prostatectomy as the treatment of choice in such patients who are usually fit, thus avoiding the dangers of infection by instrumentation and the increased liability to venous thrombosis if catheter drainage and bed-rest are instituted.

mucosa, and narrowing the cavity with stitches in front of the urethral catheter which was left in to drain the bladder. The bladder wall was left completely closed and the wound drained. **Wilson Hey** (1944) also advocated a transvesical prostatectomy, stressing the importance of careful asepsis in this as in any other surgery. The catheter for drainage was passed retrogradely to avoid carrying urethral organisms up into the raw prostatic cavity. Mass ligatures of tissue for hæmostasis was replaced by careful diathermy of bleeding points with insulated hæmostats (fig. 1529). Careful closure of the bladder enabled the urethral catheter to be removed on the fourth day.

Retropubic Prostatectomy (**Millin**, 1945).—Using a low, curved transverse suprapubic incision, which includes the rectus sheath, the recti are retracted to expose the bladder with its typical appearance of pale brown muscle bundles with a loose covering of fatty tissue and veins. With the patient in the Trendelenberg position the surgeon separates the bladder and the prostate from the posterior aspect of the pubis. In the space thus obtained the anterior capsule of the prostate is incised with



FIG. 1529.—Insulated hæmostat (Riches' pattern).



FIG. 1530.—McCarthy's endoscopic prostatic resector.

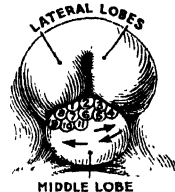


FIG. 1531.—Transvesical view showing the order in which strips are removed (middle lobe). (After R. W. Barnes.)

diathermy below the bladder neck, care being taken to obtain complete control of bleeding from divided prostatic veins. The prostatic adenoma is exposed and enucleated with a finger. A wedge is taken out of the posterior lip of the bladder neck to prevent secondary stricture in this region. The exposure of the inside of the prostatic cavity is good, and control of hæmorrhage is achieved with diathermy and stitching before closure of the capsule over a Porges catheter (inserted per urethram) draining the bladder.

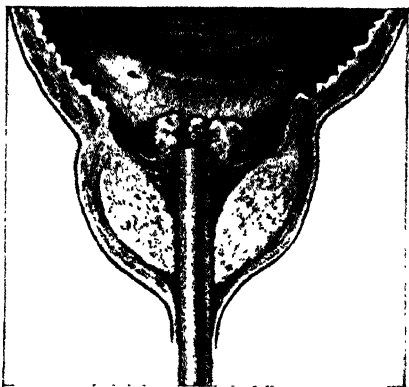


FIG. 1532.—Transurethral resection showing the loop commencing to remove a strip from the middle lobe of the prostate. (After R. M. Nesbit.)

scope or a cold punch (figs. 1530, 1531, 1532, 1533).

Strips of tissue are cut from the posterior lip of the bladder neck down to the verumontanum. Damage to the external sphincter will not occur if this is the lowest point of the resection. With the McCarthy resectoscope cutting

Perineal Prostatectomy (**Young**).—A A-shaped incision in the perineum in front of the anus. The rectum is retracted backwards to expose the posterior aspect of the prostate above the triangular ligament. This is incised and the adenoma enucleated. This operation is not popular in Britain because of the incidence of incontinence.

Perurethral prostate resection, (P.U.R. or T.U.R.) is indicated when obstruction is due to a fibrous prostate, a median bar, or a carcinoma, and occasionally when a middle lobe is associated with slight lateral lobe enlargement. It can be achieved with a McCarthy resecto-



FIG. 1533.—Punch prostatectomy. The fenestrum engaging the tissue of the middle lobe.

Terence Millin, Contemporary. Honorary Consulting Surgeon, All Saints' Hospital, London.

Porges. Surgical Instrument Manufacturers, Paris, France.

Hugh Hampton Young, 1870-1945. Professor of Urology, Johns Hopkins School of Medicine, Baltimore, U.S.A.

Sir Eric Riches, Contemporary. Honorary Consulting Surgeon and Urologist, The Middlesex Hospital, London.

Joseph Francis McCarthy, Contemporary. Professor of Urology, New York Polyclinic Medical School, U.S.A.

is accomplished by a high-frequency diathermy current applied to the loop. In the case of the Thompson punch, a tubular knife moves across a fenestrum at the distal end of the sheath of a direct-vision cysto-urethroscope. Magnification is less than with the resectoscope, but as less necrotic tissue is left behind, secondary hæmorrhage is less frequent.

AFTER TREATMENT

(1) Four ounces (120 ml.) of sterile normal saline are left in the bladder for one hour to dilute initial bleeding before continuous drainage is established.

(2) After one hour the catheter is joined via a sterile (non-tapered) connection to a sterile tube and a bottle containing 4 ounces (120 ml.) of formalin (fig. 1475).

(3) Bladder wash-outs with full aseptic technique are only given if drainage is unsatisfactory or bleeding excessive.

(4) A high urinary output to 'wash out' the bladder is essential.

(5) If the urine is infected, a suitable drug or antibiotic should be given pre-operatively to control bacteræmia and to reduce the danger of secondary hæmorrhage.

(6) Early ambulation has diminished the incidence of venous thrombosis and pulmonary embolism.

Complications.—*Hæmorrhage* may continue; transfusion is necessary if the blood pressure falls. *Clot retention* can occur if bleeding is heavy and it is not observed that drainage is poor. Suction applied with a Riches's bladder syringe and irrigation with sterile normal saline will clear this. (No fluid must be put into the distended bladder until some has been sucked out. The use of a Bigelow's evacuator in such instances is dangerous and may lead to rupture of the bladder.)

Infection.—The routine employment of sulphonamide and appropriate antibiotic therapy before operation and during the post-operative treatment has considerably reduced complications consequent upon infection of the prostatic bed.

Epididymitis.—Division of the vasa deferentia at prostatectomy almost eliminates post-operative epididymitis, but a localised inflammation of the spermatic cord or a small abscess at the site of the division sometimes occurs.

Acute Pyelonephritis.—Ascending pyelonephritis can often be combated by antibiotics, but bilateral infection occurring in kidneys, already the seat of some degree of hydronephrosis, is always serious.

Renal Failure.—*Oliguria* or *anuria* which does not respond to treatment detailed on page 1069, is the most lethal complication of prostatectomy.

Extraperitoneal perforation of the bladder is an occasional accident during transurethral prostatectomy. When the patient is under low spinal anaesthesia, immediately the perforation occurs he complains of severe suprapubic pain, often radiating towards the epigastrium. Such symptoms appearing after the patient has regained consciousness from a general anaesthetic are more difficult to interpret. When perforation of the bladder is suspected, immediate suprapubic cystostomy and drainage of the pre-vesical space should be carried out. If this operation is delayed more than twelve hours, the prognosis becomes grave, except in a few instances when extravasation occurs slowly with the formation of an abscess in one or other inguinal region.

Incontinence of urine occurs in retropubic prostatectomy when the membranous urethra is drawn up and divided. Damage to the external sphincter can occur in transurethral resections.

Later Complications

Osteitis pubis is rare. It is probably due to pricking of the periosteum during operation, or to infection in the prevesical space. The symptoms do not appear

Gershon Joseph Thompson, Contemporary. Senior Consultant, Mayo Clinic, Rochester, U.S.A.
Henry J. Bigelow, 1816-1890. Surgeon, Massachusetts General Hospital, U.S.A.

until two or more weeks after the operation. There is pain and tenderness over the pubes, with inability to walk more than a few steps owing to painful spasms of the adductor muscles. An X-ray shows irregular rarefaction of the ischio-pubic rami (fig. 1534) and widening of the symphysis.



FIG. 1534.—Radiograph showing osteitis pubis following retropubic prostatectomy. (N. M. Matheson, F.R.C.S., Ashford, Middlesex.)

This exceedingly painful state continues for many weeks. Spontaneous cure with recalcification of the rarefied bone occurs after several months. Earlier recovery can be achieved by incising the posterior aspect of the symphyses and curetting out the necrotic cartilage and bone. Penicillin and streptomycin are placed in the area and the wound is closed with a small corrugated drain which is removed after forty-eight hours.

Post-operative stricture of the prostatic bed is a troublesome complication. Dilatation must be carried out regularly. More common now are strictures of the anterior urethra following the passage of a wide-bore resectoscope or punch.

CONTRACTURE OF THE BLADDER NECK

Ætiology.—The condition occurs in children of both sexes and in women as well as men. It may be due to a congenital muscular hypertrophy, or fibrosis of the tissues at the bladder neck and is then an aftermath of chronic prostatitis in men, or urethro-trigonitis in women. The former cause may give rise to symptoms at any age, usually in young children or early adult life, the latter after the age of forty-five.

Clinical Syndromes:

(a) *Due to Muscle Hypertrophy.*—Marion described a series of cases in which muscular hypertrophy of the internal sphincter in a young person had resulted in the development of a vesical diverticulum or hydronephrosis (Marion's Disease, or *prostatism sans prostate*) (fig. 1535). Milder degrees of obstruction can occur without the development of these secondary effects (p. 1162). These patients have difficulty in micturition or may present with recurrent urinary infections.

A severe degree of obstruction produces symptoms at birth or in early childhood. A distended bladder may be visible or easily felt. Many cases develop urinary infection. Ureteric reflux of urine associated with high intravesical pressure during micturition leads to loin pain and severe pyelonephritis with gross scarring and contraction of the renal cortex.

(b) *Due to Fibrosis.*—The symptoms are similar to those of prostatic enlargement, but straining helps to expel the urine, and as a consequence an inguinal hernia may develop. There is no prostatic enlargement. Secondary effects of the



FIG. 1535.—Contracture of the bladder neck causing 'prostatic' obstruction. (After H. H. Young.)

obstruction are in all respects similar to those seen in prostatic enlargement. Contracture of the bladder neck is the most common cause of a diverticulum of the bladder.

Treatment:

Dilatation.—Minor degrees of the condition sometimes respond satisfactorily to intermittent dilatation by bougies and treatment of the chronic prostatitis, if it is still active. Otherwise operation is always needed.

Transurethral resection is practised widely for this condition. The immediate results are brilliant, but early relapses so often follow, especially in women, that opinion is veering to open operation.

Open Operation.—The principle of open operative treatment, like that of Johanson's operation for urethral stricture (p. 1196), is to establish in the strictured portion of the canal one part of the circumference that is non-fibrotic and pliant.

Sphincteroplasty is conducted through a retropubic incision.

Bonnin's operation is suitable for adult male patients.

A V-shaped flap in the bladder (muscle and mucosa) is introduced into the anterior aspect of the vesical outlet, and the steps of the operation are shown in fig. 1536. When severe vesical atony is present, which is usual in cases of some standing, it is desirable to resect a large portion of the bladder. This is accomplished by mobilising the antero-superior aspect of the bladder before incising it. To have the viscus well distended is most important.

In women the shortness of the urethra makes the above operation very difficult. A vertical incision over the neck of the bladder extending into the urethra, which is sewn up transversely (fig. 1537), gives excellent results.

In children of both sexes the same operation as in women gives good results, and allows excision of obstructing valves, if such be present.

Congenital Valves of the Prostatic Urethra (p. 1184).

PROSTATIC CALCULI

Prostatic calculi are of two varieties—endogenous, which are common, and exogenous, which are comparatively rare.

An *exogenous* prostatic calculus is a urinary (commonly ureteric) calculus that becomes arrested in the prostatic urethra. This is considered on p. 1199.

Endogenous prostatic calculi are usually composed of calcium phosphate combined with about 20 per cent. of organic material.

Clinical Features.—Quite often prostatic calculi are symptomless, being discovered on radiography of the pelvis, during prostatectomy, or associated with carcinoma of the prostate or chronic prostatitis. The symptoms vary in severity and are at first those of chronic prostatitis, or of prostatic obstruction. On rectal examination it is difficult to differentiate from carcinoma. On X-ray these stones often form a horse-shoe (fig. 1538) or a circle.

Noel James Bonnin, *Contemporary*. Surgeon, Queen Elizabeth Hospital, Adelaide, Australia.

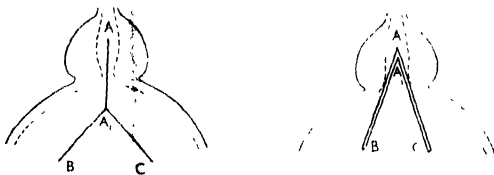


FIG. 1536.—Bonnín's operation for contracture of the bladder neck in the male.

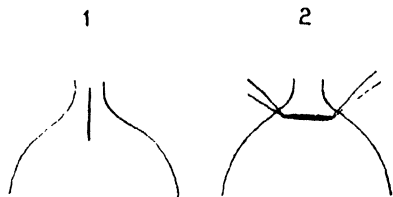


FIG. 1537.—Vesical sphincteroplasty in the female.



FIG. 1538. — Radiograph showing endogenous prostatic calculi.

Treatment of Prostatic Calculi:

Conservative Measures.—When the calculi are small and the symptoms are mild, the treatment of chronic prostatitis by prostatic massage and a course of sulphonamide often keeps the patient free from symptoms for months at a time.

Transurethral resection of the fibrous prostate will release small calculi as the strips of prostatic tissue are excised, and many more are removed by the irrigation inseparable from transurethral resection. Others are passed per urethram at a later date.

Retropubic Prostate-lithotomy.—After incising the capsule of the prostate longitudinally, the incision is deepened until the prostatic urethra is opened. Using a sharp curette, all the calculi and as much as possible of the infected prostatic tissue is removed. The bladder neck is then exposed, and a generous wedge of the posterior lip is resected with curved scissors. The operation is concluded in a manner similar to that of retropubic prostatectomy. Total prostatectomy may be necessary in cases where there are frequent acute exacerbations of a chronic prostatitis.

Corpora amylaceæ

Corpora amylaceæ are found in the prostates of elderly men and apes, but not in the prostates of animals lower in the phylogenetic scale than anthropoids. Corpora amylaceæ occur also in the homologue of the prostate—the tubules of the bladder neck of women (Skene's tubules). These bodies consist of amorphous débris and desquamated epithelium. Often an intact epithelial wall of a prostatic duct forms their outer coat. They are always deeply pigmented, and are variously described as like poppy seeds, black pepper, or coal dust. Neither the composition nor the origin of the pigment is known. Probably corpora amylaceæ are the forerunners of endogenous prostatic calculi.

CARCINOMA OF THE PROSTATE

Carcinoma of the prostate is the commonest malignant condition in men over the age of sixty-five years. About 20 per cent. of cases of prostatic obstruction prove to be due to carcinoma, to which must be added a substantial number in whom the first and main symptoms are due to metastases. The incidence of carcinoma of the prostate has been increasing, due to an ageing population as well as to improved methods of diagnosis. Carcinoma of the prostate commences in one of the following ways:

- (a) In the posterior zone of the normal gland (fig. 1518).
- (b) Diffusely in one or other normal lateral lobes.
- (c) In association with benign enlargement.

Prostatectomy for benign enlargement of the gland confers little protection to the subsequent development of carcinoma.

Latent Carcinoma of the Prostate.—Serial sections of prostates obtained at routine necropsy has revealed carcinoma in that organ in no less than 15 per cent. of men over fifty years of age. True, many of these neoplasms are tiny and (if life had continued) might have remained latent for years. In men over ninety years of age, over three-quarters of the prostates are involved (Franks). It seems, therefore, that a seedling carcinoma is often present in the prostate of an elderly man, awaiting favourable conditions to become active.

Non-latent Carcinoma of the Prostate.—Usually the carcinoma is spheroidal celled, with a varying degree of tubule formation, in which case

the tumour is a slowly growing one. The anaplastic type is more malignant and manifests itself more aggressively than an adenocarcinoma.

Local Spread.—A growth *commencing in the posterior zone of the gland* is prevented (at least temporarily) from extending backwards by the strong fascia of Denonvilliers. Consequently it tends to grow upwards to involve the seminal vesicles. Further upward extension obstructs the lower end of one or both ureters, the latter terminating in anuria. Carcinoma *commencing in a lateral lobe* involves the prostatic urethra early: in advanced cases the base of the bladder is invaded. The rectum is involved occasionally by infiltration, and stricture of the rectal wall occurs. The mucosa does not ulcerate unless traumatised, e.g. by transrectal biopsy.

Spread by the blood-stream occurs particularly to bones; indeed, the prostate is the most common site of origin for skeletal metastases, being followed in turn by the breasts, the kidneys, the bronchial tree, and the thyroid gland. The bones involved most frequently by metastases of carcinoma of the prostate are shown in fig. 1539. The pelvic bones and the lower lumbar vertebræ are particularly common sites. The frequent proximity of skeletal metastases to the primary growth has been attributed to reversed flow from the vesical venous plexus to the emissary veins of the pelvic bones during coughing, sneezing, etc.

Lymphatic Spread.—(a) Via lymphatic vessels passing along the sides of the rectum to the lymph nodes along the internal iliac vein and in the hollow of the sacrum. (b) Via lymphatics which pass over the seminal vesicles and follow the vas deferens for a short distance to drain into the external iliac lymph nodes. From both these situations the retroperitoneal lymph nodes, and later the mediastinal nodes, and occasionally the supraclavicular lymph nodes, become implicated.

Clinical Features.—Carcinoma of the prostate usually occurs at an earlier age than benign hypertrophy. More than half the patients present with acute or chronic retention of urine.

Type 1. The Pathological.—The symptoms and signs are identical with those of benign enlargement. At histological examination 2 to 5 per cent. of prostates removed in the belief that they were benign prove to contain one or more areas of carcinoma.

Type 2. The Clinically Doubtful.—The symptoms and signs are similar to those of benign enlargement or fibrous prostate, but as a rule the

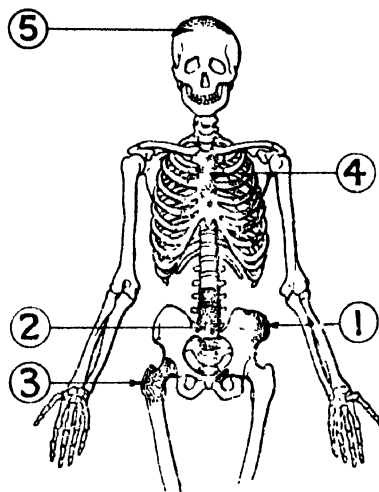


FIG. 1539.—Sites of location, in order of frequency, of metastases in bones from carcinoma of the prostate. (After Swift Joly.

history is comparatively short (weeks, not months). A hard nodule or increased fixation of the gland favours the diagnosis of carcinoma.

Type 3. The Clinically Certain.—The patient complains of pain in the perineum or suprapubic region, in addition to symptoms of prostatic obstruction. On rectal examination (see below) the findings leave no doubt as to the diagnosis.

Type 4. The Occult.—Urinary symptoms are absent or slight. Pain in the back, or sciatica, is the main symptom. Bilateral sciatica in an elderly man is most often due to metastases in the spine from carcinoma of the prostate. Œdema of one or both legs, paraplegia, or a spontaneous fracture are occasionally due to metastases from a carcinoma of the prostate. Anæmia may be the presenting symptom.

On account of destruction of bone marrow, bone metastases from carcinoma of the prostate can give rise to a hæmorrhagic diathesis, and the patient suffers hæmorrhage, often severe, not necessarily from the urinary tract.

Rectal Examination.—Irregular induration, characteristically stony hard, in a part or the whole of the gland, with decreased mobility suggests carcinoma. Sometimes the rectal mucosa may be felt to be 'tethered' to the back of the prostate by infiltration of the carcinoma (Lett). Obliteration of the notch between the seminal vesicles, or of the groove between the lateral lobes, adds to the suspicion. If, in addition, the induration extends beyond the lateral limits of the gland, causing obliteration of the lateral sulci, or to the membranous urethra, the diagnosis is certain. Prostatic calculi and calcareous changes secondary to tuberculosis of the seminal vesicles, present the greatest difficulties in the diagnosis of prostatic carcinoma. In the doubtful case an examination under anæsthesia may be helpful.

Radiological examination to exclude or confirm the presence of prostatic calculi or pelvic and lumbar skeletal metastases is always necessary. Whereas osseous metastases from carcinomas of other organs are usually osteolytic, producing a rarefied 'moth-eaten' appearance, those in the lower lumbar vertebræ and pelvic bones from a carcinoma of the prostate are typically osteoblastic, resulting in increased density of the bone (fig. 1540), and must be distinguished from Paget's disease of bone (p. 246).

Additional Aids to Diagnosis in Doubtful Cases.—One or more of the following procedures are often required to confirm or disprove a doubtful lesion :



FIG. 1540.—Osseous metastases of the pelvic bones in carcinoma of the prostate. (L. N. Pyrah, F.R.C.S., Leeds.)

Serum Acid Phosphatase.—In about 40 per cent. of patients with carcinoma of the prostate the acid phosphatase raised above normal (1 to 3 King-Armstrong units). A reading between 3 and 5 units is suspicious of carcinoma of the prostate, and above 5 units practically diagnostic. In patients with metastases, 10 K-A units per 100 ml. of serum is not uncommon. See also p. 1161.

Exfoliate Cytology.—Prostatic massage is conducted in a systematic manner. The patient

should stand with the legs apart, leaning forward and steadying himself with one hand resting upon the examination couch; with the other hand he retracts his prepuce. Four slides, duly numbered, are placed in readiness on the couch.

Prostatic massage is conducted in such a way as to avoid compressing the seminal vesicles, viz. —————→

4 drops being obtained and collected separately. In this way contamination with seminal fluid is likely to be avoided (Fergusson). The massage concluded, each slide is covered with another, similarly numbered. Each pair is slid apart, leaving a film of secretion upon it, and they are placed in fixative fluid. Procuring a prostatic smear for cytological examination should not be conducted within three days of a diagnostic rectal examination of the prostate. Pathologists with special experience can state whether or not the specimen contains carcinoma cells.

Biopsy.—Needle, open, and transurethral biopsy are practised.

Needle Biopsy.—Turkel's or a similar instrument is passed as shown in fig. 1541. The cannula is removed and the trephine is passed through the needle and directed to a suspicious nodule.

Open Biopsy.—Many believe that a formal biopsy through a Π -shaped perineal incision is the best method. Obviously a wedge of tissue obtained in this way gives the pathologist more scope in doubtful cases.

Transurethral Biopsy (see below).

Cysto-urethroscopy.—When there is a history of hæmaturia, this examination is essential. There is often a grating sensation as the prostatic urethra is traversed. With the instrument in place, deep induration in the prostate is more readily appreciated by a finger in the rectum, and fixation of the gland becomes more evident. Puckering of the apex of the trigone, submucous nodes in the base of the bladder, or ulceration of an intravesical projection are late manifestations. In earlier cases there are no pathognomonic visible signs.

Transurethral Biopsy.—Transurethral resection of the prostate has the advantage of removing the obstruction and providing large pieces of tissue, but it may not reach the posterior zone of the prostate, which is commonly the seat of early carcinoma.

Vesiculography.—In comparatively early cases distortion of the topography of the ampullæ of the seminal vesicles is extremely suggestive of carcinomatous infiltration.

Bone marrow aspiration (sternum or ilium) reveals metastatic carcinoma cells in a surprisingly high percentage of those cases where radiological examination revealed no evidence of secondary deposits. This investigation should be carried out in every case before radical prostatectomy is even considered.

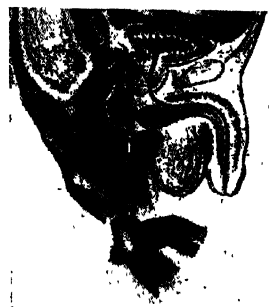
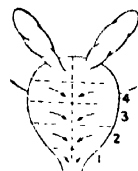


FIG. 1541.—Obtaining a specimen of prostatic tissue by means of Turkel's needle.

TREATMENT OF CARCINOMA OF THE PROSTATE

In spite of every endeavour, the diagnosis of carcinoma of the prostate usually is made late in the course of the disease, as the following statistics of the condition at the time of diagnosis testify:

Neoplasm confined within the anatomical capsule of the prostate	5 per cent.
Local spread	55 per cent.
Metastases present	40 per cent.

Radical cure by surgery plays a very small part in the management of the condition. However, a great deal can be done to alleviate the symptoms. Huggins first showed the beneficial effects of castration or stilbæstrol.

Stilbæstrol causes improvement in 85 per cent. or more cases. Histological changes in many of the malignant cells may be apparent within forty-eight hours of commencing treatment. The cells become distended with glycogen, the nucleus becomes pyknotic, and moves to the edge of the cell, being extruded when the cell breaks up.



FIG. 1542.—Gynæcomasia following stilbæstrol therapy in a man 80 years of age with carcinoma of the prostate.

Symptomatic changes occur in many cases within two weeks—there is less dysuria, a better stream, less frequency, and aches and pains may disappear. The prostate becomes smaller and softer, and these changes may be detected rectally in three to four weeks. Bone deposits may remain static and occasionally disappear. Dosage—up to 25 mg. four times daily.

Side-effects of Stilbæstrol.—(1) Gynæcomasia and pigmentation of the nipples is marked with large doses and impotence is invariable. (2) Nausea and gastric upsets are occasionally present, and these are unrelated to dosage. (3) Oedema of the ankles from salt retention may occur. (4) Excessive nasal secretion is complained of by some patients and disturbance of vision by others. (5) Adrenal stimulation elevating the level of androgen secretion may occur.

In all such cases subcapsular orchiectomy (figs. 1543 and 1544), and cessation of œstrogen therapy can be tried, especially valuable in types of side-effects 2, 3, and 4 previously described. Alternatively T.A.C.E. (tripara-anisil-chlor-ethylene), which is said to stimulate the breast and adrenal less, may be tried. Dose—12 to 24 mg. per day.

Dienæstrol is considered by some to be an improvement on stilbæstrol. It is a stronger preparation, and the dose is 45 mg. daily given by mouth.

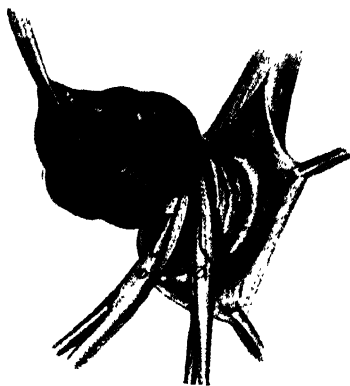


FIG. 1543.—Subcapsular orchiectomy in progress.

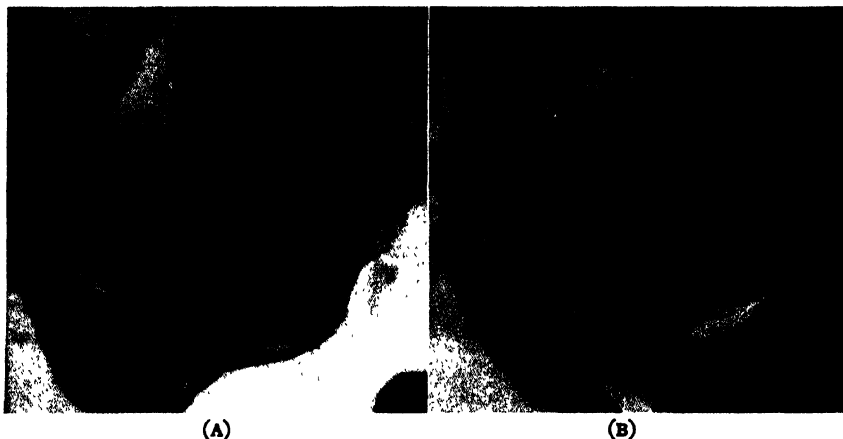


FIG. 1544.—(A) Metastases of prostatic carcinoma in the scalp. (B) Thirteen months after orchiectomy. (Professor C. Huggins, Chicago.)

Charles Huggins, Professor of Surgery, Chicago, and his co-workers introduced the treatment of carcinoma of the prostate by orchiectomy and stilbæstrol in 1941. Nobel prize-winner 1966.

Honvan (phosphorylated diethylstilbœstrol) is given intravenously. It is claimed that the acid phosphatase of the prostatic carcinoma cells splits the phosphate element of the preparation, thus releasing the œstrogenic hormone locally and favouring its direct action on the carcinoma cells, and the carcinoma cells only. At first 100 mg. are injected (it must be injected very slowly) three times a day for ten days. The dose is then increased to 200 mg., and eventually (enfeebled patients excepted) to as much as 500 to 1,000 mg., until the twentieth day. The dosage is then gradually decreased, until one 200-mg. injection is given weekly, and finally monthly. The intravenous injections, like all intravenous injections, require skilled administration. Liver damage may occur.

TREATMENT OF RETENTION IN CARCINOMA OF THE PROSTATE

Acute retention due to a carcinoma of the prostate should be treated with an indwelling Gibbon catheter (p. 1130) and 25 mg. stilbœstrol four times daily for two weeks. Usually micturition will then be resumed. If after three weeks the patient cannot pass urine normally, then the obstruction should be relieved. This is usually done by a **trans-** (per) urethral resection (fig. 1545), but if the gland is also the seat of adenomas, a retropubic or **transvesical** removal of most of the tissue may be considered.



FIG. 1546.—Approximating the base of the bladder to the membranous urethra. (After T. Millin, F.R.C.S.)

Chronic Retention.—Such cases should not be catheterised, especially if the urine is sterile when first seen. Infection once established is impossible to eradicate in the presence of a growth. The residual urine may decrease gradually with treatment. Perurethral resection may be necessary if this does not occur.



FIG. 1545.—A passage is made through the carcinomatous prostate by a resectoscope. (Dr. J. K. Lattimer, New York.)

Radical Surgery for Carcinoma of the Prostate.—Total prostatectomy is performed either by a retropubic approach (Millin) or through the perineum (Young). After the prostate is removed the membranous urethra is anastomosed to the base of the bladder (fig. 1546). The advantage of the perineal over the retropubic approach is the ability to perform a biopsy as a preliminary part of the operation.

Other Measures and Prognosis

Treatment of Pain and Enlarged Lymph Nodes.—Severe pain from bone secondaries, but more usually from perineural lymphatic infiltration, can be difficult to control, as is œdema of a leg from enlarged lymph nodes. Deep X-ray therapy combined with intra-arterial nitrogen mustard given into the pelvic vessels via a retrograde catheter up the femoral artery (two doses of 10 to 15 mg. at weekly intervals) is most useful.

Adrenalectomy is of little value. Pituitary ablation has occasionally been found helpful.

Prognosis.—Patients who fail to respond to stilbœstrol usually die within one year. On stilbœstrol five-year survival amounts to 25 to 30 per cent.

Occasional survivals up to ten years are recorded, but ultimately nearly all patients die of the disease.

PROSTATITIS

In both acute and chronic prostatitis the seminal vesicles are usually infected, and when, as is often the case, the prostatic urethra is involved also, there is present a triad of pathological conditions, to wit: posterior urethritis, prostatitis, and seminal vesiculitis. Symptoms due to infection of any one of these structures may predominate.

Acute Prostatitis

Ætiology.—Acute prostatitis is common.—The usual organism responsible is *Esch. coli*, but *Staphylococcus aureus* and *albus*, *Streptococcus faecalis*, and the *gonococcus* may be responsible. As a rule the infection is hæmatogenous from a distant focus, notably furunculosis, infected tonsils, carious teeth, or diverticulitis. In a minority of cases the infection ascends from the urethra or descends from the bladder or kidneys.

Clinical Features.—Infection in most cases seems to be blood-borne, as general manifestations overshadow the local: the patient feels ill, shivers, may have a rigor, has 'aches' all over, especially the back, and may easily be diagnosed as having influenza. The temperature may be up to 102° F. (39°C.). Dysuria is usual, but not invariable. The urine always contains threads in the first glass. Perineal heaviness, rectal irritation and pain on defæcation can occur, and sitting may be uncomfortable. A urethral discharge is rare. Frequency occurs when the infection spreads up to the bladder. Rectal examination reveals a tender prostate, one lobe may be swollen more than the other, and the seminal vesicles may be involved. A frankly fluctuant abscess is uncommon.

Treatment.—Must be rigorous and prolonged or the infection will not be eradicated and recurrent attacks will ensue. Unless treated early, spread of the infection to the epididymes and testes is common. While awaiting culture, the patient is put to bed, given copious fluids, and started on a broad spectrum drug such as tetracycline. As soon as the culture result is available the most suitable drug can be chosen and continued for ten days. It should be followed by a month's course of a long-acting sulphonamide such as sulfamethoxypyridazine (Midicel) to prevent recurrence. Bed-rest for ten to fourteen days, avoidance of alcohol and intercourse for six weeks is essential.

Prostatic Abscess.—In addition to the foregoing symptoms and signs, the advent of a prostatic abscess is heralded by the temperature rising steeply, rigors being not unusual. However, antibiotics disguise these leading features. Severe, unremitting perineal and rectal pain with occasional tenesmus often cause the condition to be confused with an ano-rectal abscess. Nevertheless, if a rectal examination is performed, the prostate will be felt to be enlarged, hot, extremely tender, and perhaps softened in one place. Retention of urine is likely to occur.

Treatment.—The abscess should be drained without delay. It is true that if a catheter is passed to relieve acute retention of urine, sometimes the abscess is ruptured into the prostatic urethra. However, in most cases such drainage is insufficient, and is followed by intractable chronic prostatitis or a residual abscess.

(a) The abscess can be drained by perurethral resection—unroofing the whole cavity.

(b) The perineal route is preferable when there is marked peri-prostatic spread.

CHRONIC PROSTATITIS

Ætiology.—This is a sequel of inadequately treated acute prostatitis. While pus is present in the prostatic secretion, often the responsible organism is difficult to find. Smears show bacteria in about 40 per cent. and cultures are positive in 70 per cent. of cases. The predominant organisms are *E. coli*, staphylococci, streptococci, and diphtheroids, in that order. Recently trichomonas has been found to be a cause of chronic prostatitis and may be common to both husband and wife.

Pathology.—The lumina of the ducts become blocked with epithelial debris and pus. This causes a soft enlargement of the organ. Later fibrosis occurs, and the prostate becomes smaller and harder.

Clinical features are extremely varied.

1. *Causing Chronic Posterior Urethritis.*—Prostatitis should not be diagnosed unless, after irrigating the urethra and massaging the prostate, the resultant specimen shows fifteen or more pus cells per high power field.

2. *Causing Epididymitis.*—Acute or subacute non-tuberculous epididymitis rarely occurs unless prostatitis is present.

3. *Pain:* (a) *Local pain* is a dull ache in the perineum or rectum. It is increased by sitting on a hard chair. (b) *Referred Pain.*—Particularly common is low back pain, sometimes extending down the legs (fig. 1547); such pain is usually attributed to lumbago, and many sufferers from prostatitis receive orthopædic treatment and physiotherapy without benefit.

4. *'Silent' Prostatitis.*—Arthritis, myositis, neuritis, and sometimes iritis and conjunctivitis are on occasions explained only when pus has been obtained from the prostate (see diagnosis).

5. *Recurring attacks of mild pyrexia*, lasting about three days and accompanied by malaise.

6. *Sexual Dysfunction.*—Premature ejaculations, prosta-torrhœa, and impotence are sometimes due to prostatitis.

Many patients with sexual dysfunction due to chronic prostatitis become hypochondriacal.

Diagnosis: (a) *A three-glass urine test* is valuable. If the first glass shows urine containing prostatic threads, prostatitis is present.

(b) *Rectal examination* of the prostate may or may not confirm the diagnosis. If the organ is soft and boggy, it is obviously abnormal, as also it is if the prostate is smaller and harder than it should be. In mild chronic cases no change can be detected, and reliance must be placed on pathological examinations of the prostatic fluid.

(c) *Examination of the prostatic fluid* obtained by prostatic



FIG. 1547.—
Sites of pain
in chronic
prostatitis.

massage. Normal prostatic fluid is slightly opalescent and viscid. A stained specimen shows many pus cells and sometimes bacteria. Non-stained films, in addition to revealing normal epithelial cells, may show trichomonads, if present.

(d) *Urethroscopy* reveals inflammation of the prostatic urethra, and pus may be seen exuding from the prostatic ducts. The verumontanum is likely to be enlarged and œdematous.

Treatment.—*Antibiotic therapy* must be administered in accordance with bacteriological sensitivity tests. The blunderbuss administration of antibiotics in this condition is to be deprecated. Probably 0.5 G. t.d.s. of a suitable sulphonamide, e.g. Sulphatriad, for six weeks will effect as much as an expensive course of antibiotics. Where trichomonas is the responsible agent, a rapid response is obtained from administration of Flagyl (metronidazole) 200 mg. by mouth three times daily, after meals, for seven days to both partners.

Massage of the prostate per rectum is a most important measure, the prostatic secretion and the contents of the seminal vesicles being emptied thereby. Usually eight strokes on each side are given daily for a week, and then at lengthening intervals as improvement sets in. In the later stage of treatment, which usually must be prolonged over months, urethral dilatation serves to open the prostatic ducts and permits better drainage. In most cases the infection will be eradicated by these measures. After apparent cure, prostatic massage is performed on at least two occasions, at monthly intervals, and the fluid examined for re-infection.

TUBERCULOSIS OF THE PROSTATE AND SEMINAL VESICLES

Tuberculosis of the prostate and seminal vesicles is associated with renal tuberculosis in at least 60 per cent. of cases. In 30 per cent. of cases there is a history of pulmonary tuberculosis within five years of the onset of genital tuberculosis.

Tuberculosis of one or both seminal vesicles is more common than tuberculosis of the prostate, and is often discovered when examining a patient with chronic tuberculous epididymitis, there being no symptoms referable to the internal genitalia. On rectal examination the affected vesicle is found to be nodular and tender. In process of time tuberculous seminal vesiculitis may lead to congestion and œdema of the base of the bladder, and later to basal cystitis.

When the Prostate is Involved.—Rectal examination reveals characteristically one or more well-defined nodules, most often near the upper or lower border of one or both lateral lobes. Less frequently a larger solitary mass is felt occupying a more central position. Nodules in the prostate lack the stony hardness of carcinoma.

A patient with tuberculous prostatitis usually presents with one or more of the following symptoms:

A urethral discharge is occasionally the first symptom. Then the diagnosis has to be made from gonorrhœa and abacterial urethritis entirely on bacteriological findings, for the prostate at this early stage is likely to feel normal.

Painful, sometimes blood-stained, ejaculations occur in 20 per cent. of cases.

A mild ache in the perineum is not uncommon.

Infertility.—In patients with tuberculous prostatitis and/or bilateral seminal vesiculitis, fertility is very much reduced; it is safe to say that 80 per cent. of them are sterile. In this connection, owing to the considerable interest in male fertility at the present time, a number of very early cases of tuberculous prostatic-vesiculitis are being discovered by culture of the semen.

Urinary Symptoms.—In later cases, when the posterior urethra becomes involved from extension of tubercles from the prostate or by the discharge of a prostatic abscess, there is painful, frequent micturition and sometimes terminal hæmaturia.

Abscess Formation.—If a cold abscess forms in the prostate, it is felt as a slightly tender, soft swelling. Like other prostatic abscesses, it usually ruptures into the urethra, more rarely through the perineum or into the rectum. Occasionally an abscess of the prostate or seminal vesicle bursts into the bladder, and at cystoscopy a ragged, deep ulcer is seen alongside the trigone.

Special Forms of Investigation:

Even in cases without urinary symptoms a complete urological examination is necessary.

Radiography sometimes displays areas of calcification in the prostate and/or the seminal vesicles. Large scattered areas of calcification in the prostate suggest tuberculosis rather than endogenous prostatic calculi.

Bacteriological examination of the seminal fluid yields positive cultures for tubercle bacilli in most cases of tuberculous prostatitis.

Posterior urethrography often reveals one or more dilated prostatic ducts. Typically they are multiple and gaping. Dilated prostatic ducts are not specific for tuberculosis (they can occur in other forms of chronic prostatitis), but dilated ducts plus the finding of tubercle bacilli in the ejaculate establishes an absolute diagnosis.

Transmission of genital tuberculosis to the female partner is exceptional, but it does occasionally occur in the form of tuberculous cervicitis.

Treatment.—The general treatment given is as for renal tuberculosis (p. 1111). On no account must a tuberculous prostate or seminal vesicle be subjected to massage. Urethral instrumentation should be avoided, or reduced to a minimum only to confirm the diagnosis.

If a prostatic abscess forms, it is better to evacuate it by the perineal route than to permit it to rupture spontaneously.

Considerable involvement of the prostate, which goes on to suppuration, is a comparatively unfavourable form of genito-urinary tuberculosis. On the other hand, tuberculous seminal vesiculitis and non-suppurative tuberculosis of the prostate usually heal after an associated lesion of the kidney or epididymis has been eradicated, but stricture of the prostatic urethra may occur.

AFFECTIONS OF THE SEMINAL VESICLES

Acute seminal vesiculitis always occurs in association with prostatitis. Prior to the antibiotic treatment of gonorrhœa, gonococcal vesiculitis was common. When a seminal vesicle is distended it can be palpated per rectum as an acutely tender swelling above and lateral to the prostate. The treatment is discussed on p. 33.

Abscess of a Seminal Vesicle.—In addition to the usual signs of acute prostatitis, pain is frequently referred to the suprapubic region. If on rectal examination the seminal vesicle is found to be greatly enlarged and tender, the abscess should be drained through an incision in the perineum, viz. → The incision is deepened until the swelling is palpated, and a hæmostat is then thrust into the abscess cavity.



Chronic seminal vesiculitis is less frequently recognised than it should be, because the associated chronic prostatitis overshadows the symptoms of pain on coitus, hæmospermia, aching in the sacral region, recurrent epididymitis, and, at times, associated disturbances of micturition and defæcation. The treatment is the same as for chronic prostatitis, except that in rebellious cases vasotomy and irrigation of the vesicle is often rapidly curative.

Tuberculous Seminal Vesiculitis.—The clinical features and treatment are discussed on p. 1180 and above.

Diverticulum of the seminal vesicle occurs occasionally. In such cases the kidney of that side is often absent, and the diverticulum represents an abortive ureteric bud. It is a cause of persistent infection of the urethra.

A cyst of the seminal vesicle is uncommon. It should be removed by dissection through an incision similar to that for perineal prostatectomy.

AFFECTIONS OF COWPER'S GLANDS

Cowperitis.—The diagnosis of Cowperitis is often wanting for lack of a simple examination. On passing the forefinger into the rectum and placing the thumb first on one side and then on the other of the median raphe of the perineum, Cowper's glands can be palpated (fig. 1548). In acute cases the least pressure causes excruciating pain. In this way the condition is differentiated from an ischio-rectal or peri-urethral abscess. **Treatment.**—In acute cases antibiotic therapy, and rest in bed, often brings about resolution. Should suppuration occur, incision and drainage is necessary.

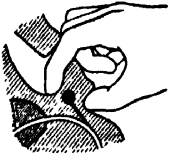


FIG. 1548 —
Palpating an en-
larged gland of
Cowper.

Fistula formation is the result of an abscess of the gland terminating in spontaneous rupture; it is necessary to excise the fistula and the remnants of the gland.

A cyst of Cowper's gland either bulges into the urethra and causes disturbances of micturition, or gives rise to a unilateral swelling in the anterior part of the perineum. The cyst should be excised.

William Cowper, 1666–1709. London Surgeon. Was the first in England to dissect a marsupial. He published anatomical works in a sumptuous fashion.

CHAPTER 48

THE URETHRA AND PENIS

THE MALE URETHRA

Embryology.—From the internal urinary meatus to the sinus pocularis (uterus masculinus) the urethra is developed from the urogenital sinus. This portion corresponds to the entire female urethra. Beyond the sinus pocularis the male urethra, as far as the glans, is formed by fusion of the edges of the medial labial folds, which also form the corpus spongiosum (fig. 1549). The part traversing the glans is formed by canalisation of a down growth of a solid pencil of ectoderm.

CONGENITAL ABNORMALITIES OF THE URETHRA

Meatal Stenosis.—The external urinary meatus, normally the narrowest part of the male urethra, is occasionally the seat of congenital stenosis which is associated with phimosis (p. 1203). In the circumcised, meatal stenosis may also be acquired or increased by fibrosis following meatal ulceration. All degrees of narrowing are encountered. When the opening is reduced to a pin-hole, back-pressure affects the whole urinary system. Pin-hole meatus is occasionally a cause of enuresis, and at any age it may result in chronic retention of urine (fig. 1550).

Treatment.—Meatal stenosis, sufficient to (a) give rise to symptoms, (b) prevent free drainage of the discharge in cases of urethritis, or (c) obstruct the passage of full-sized urethral instruments or a cystoscope, should be treated by meatotomy. Lesser degrees of narrowing respond to dilatation with Hegar's dilators.



FIG. 1550.—Pin-hole meatus causing chronic retention of urine (distended bladder outlined) in a man of fifty-one. Cured by meatotomy.

from re-uniting. Usually dilatation on two occasions at weekly intervals is sufficient in this instance.

Congenital stricture of the urethra is very rare; some cases are due to reduplication of the urethra. Usually the symptoms are delayed until adolescence. Treatment by dilatation is effective.

Congenital valves of the posterior urethra are often symmetrical and occur usually just below (fig. 1551), but occasionally above, the verumontanum. They allow the ingress of a catheter but obstruct the outflow of urine. In a few instances the valves are incomplete and the patient may reach adolescent or adult life without symptoms; however, in such cases the prostatic urethra is greatly dilated and diverticula of the bladder are present.

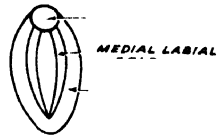


FIG. 1549.—The male and female external genitals are undifferentiated until the end of the third month.

Meatotomy.—A fine-pointed scalpel is introduced into the urethral meatus, and a cut is made in a downward direction.

In infants and children a metal bougie of suitable calibre is passed into the meatus each day for four days, and then weekly for one month.

In adults it is preferable to unite the cut edges of the urethra to the skin of the glans penis with two or three catgut sutures. These stitches control hæmorrhage and prevent the lips of the meatus

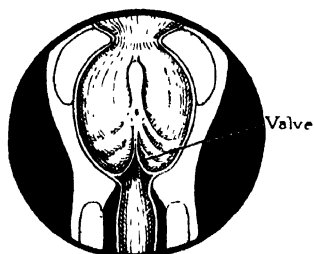


FIG. 1551.—Congenital valves of the posterior urethra.
(After D. Innes Williams.)

The valves are difficult to see at cysto-urethroscopy, because the irrigating fluid sweeps them into the open position. The most reliable method of confirming the diagnosis is voiding cysto-urethrography, by which is meant that radiographs are taken during attempts to micturate after the bladder has been filled with contrast medium. Alternatively, pressure above the pubes while the radiograph is taken achieves the same objective. In this way the dilated urethra above the valves is outlined, as well as the bladder.

Treatment.—Suprapubic drainage of the bladder is a life-saving measure; several weeks should intervene before attempting to remove the valves. At the second operation the retropubic space is displayed. A vertical incision is made into the bladder just above the prostate and continued downwards into the dilated prostatic urethra. Bleeding-points are coagulated with a diathermy needle and the valves are picked up with toothed dissecting forceps and excised with scissors. The incision into the bladder and urethra is closed with fine catgut sutures, and the bladder is drained by a suprapubic catheter for ten to fourteen days. Infection is controlled by antibiotic therapy. In older children and adults the valves can be excised transurethrally with a resectoscope.

Because of their importance in the differential diagnosis of congenital valves of the urethra, the following causes of chronic retention in childhood are included here :

Obstruction to the neck of the bladder (*syn. Marion's disease*) is analogous to hypertrophic pyloric stenosis of infants, and gives rise to symptoms identical with those of congenital valves. The diagnosis can often be appreciated cystoscopically as a hypertrophied interureteric bar. Voiding cysto-urethrography demonstrates that the posterior urethra does not fill, and clarifies the differential diagnosis between the two conditions. Treatment consists in opening the bladder and performing a Y—V plasty (p. 1171).

Neurogenic bladder must be excluded in both the above lesions. Spina bifida, and the accompanying lesions of the central nervous system, is the usual cause of retention of urine of neurological origin in childhood. As with obstruction due to congenital valves, cysto-urethrography shows the dilated prostatic urethra. In neurogenic obstruction, the case with which the bladder can be emptied by manual pressure above the pubis serves as a ready means of distinguishing chronic retention of urine due to this cause from other varieties.

HYPOSPADIAS

Hypospadias is the commonest congenital malformation of the urethra, and it occurs once in every 350 males. The external meatus is situated at some point on the *under-surface* of the penis or in the perineum.

There are five degrees of the deformity, classified as follows :

1. **Glandular Hypospadias.**—There is an ectopic opening on the under-surface of the glans, separated from a blind depression at the normal site of the external urinary meatus. Sometimes a channel connects the ectopic to the normal meatus.

2. **Coronal Hypospadias.**—The meatus is situated at the junction of the under-surface of the glans with the body of the penis.

3. **Penile Hypospadias.**—The meatus opens at some part of the under-surface of the penis (fig. 1552).

4. **Peno-scrotal Hypospadias.**—The urethra opens at the junction of the penis with the scrotum.

5. **Perineal Hypospadias.**—The scrotum is split and the urethra opens between its two halves. This variety is sometimes associated with bilateral maldescended testes, in which event the sex of the child may be difficult to determine.

Glandular hypospadias is the most frequent variety, and is due to a failure of canalisation of the glans (see embryology). The other varieties are looked upon as an absence of the urethra and corpus spongiosum distal to the ectopic orifice, the absent structures being represented by a fibrous cord. In all except the first variety the penis is curved in a downward direction (chordee). The farther away the opening is from the normal position, the more pronounced is the bowing. In all cases the inferior aspect of the prepuce is poorly developed.

Treatment.—In glandular hypospadias no treatment is required, unless the opening is too small, in which case meatotomy is performed. In other varieties a plastic operation, of which there is a great variety, should be carried out. On this account circumcision during infancy should not be performed in these cases as the redundant skin will prove useful later.

Stage 1.—*Straightening the penis* is undertaken preferably between one and a half and two years of age. A transverse incision is made on the ventral surface $\frac{1}{4}$ inch (1.25 cm.) distal to the misplaced external urinary meatus, and the skin on either side of the urethra is undermined, exposing the fibrotic corpus spongiosum, which is detached from before backwards by severing its fibrous attachment to the corpora cavernosa. This accomplished, the urethral orifice recedes towards the perineum; thus a coronal hypospadias becomes a penile, peno-scrotal, or perineal, and the penis is no longer tethered. The original transverse incision is further lengthened by carrying it into the under-surface of the prepuce on either side. The incision is then closed vertically, tension being relieved by making a longitudinal incision in the middle line through the skin of the dorsum of the penis and prepuce. The penis is wrapped in ribbon gauze soaked in flavine and liquid paraffin.

Stage 2.—*Denis Browne's method of constructing a urethra* is undertaken, preferably between five and seven years of age. Diversion of all urine from the seat of operation can be accomplished only by *perineal urethrostomy*. A Malécot catheter of correct size is passed into the bladder on a sound. The sound is withdrawn slightly, and rotated so that its beak can be felt in the perineum. Strictly in the middle line a small incision is made, and the catheter is seen within the urethra. The wall of the catheter is grasped in a hæmostat while the assistant withdraws the sound. The butt end of the catheter is then drawn along the urethra and made to emerge in the perineum. Two cotton sutures are tied around the catheter and utilised to anchor it to the edges of the perineal wound.

Fashioning a New Urethra.—An incision is made as outlined in fig. 1553 (A). The flaps are undermined, special attention being paid to freeing the skin in the direction of the perineum, so that when the flaps are united they lie well forward of the old urethral opening. When the undermining involves the scrotum, stab wounds should be made on either side to allow the ready escape of blood and serum. Next, with sharp scissors, a triangular area of glans is bared of skin on either side of the proposed new meatus (fig. 1553 (B)). An incision is made through the skin along the whole length of the dorsum of the penis. This is to relieve tension, and the wound is allowed to epithelialise. The flaps are sutured, but not too tightly. Browne employs glass beads and small sections of soft aluminium tubing, which are crushed, and hold the sutures in place (fig. 1553 (C)). No dressing is employed—only a spray of Nobecutane. Tetracycline is given for two weeks. The sutures are removed in a week, and the Malécot catheter is withdrawn on the tenth day. The perineal wound usually closes three to five days later.



FIG. 1552.—Penile hypospadias. The patient passes urine through the orifice demonstrated by the probe.

Sir Denis Browne, 1892–1966. Surgeon, Hospital for Sick Children, Great Ormond Street, London. Achille-Etienne Malécot, 1852–?. He invented his catheter while Interne des Hôpitaux de Paris.

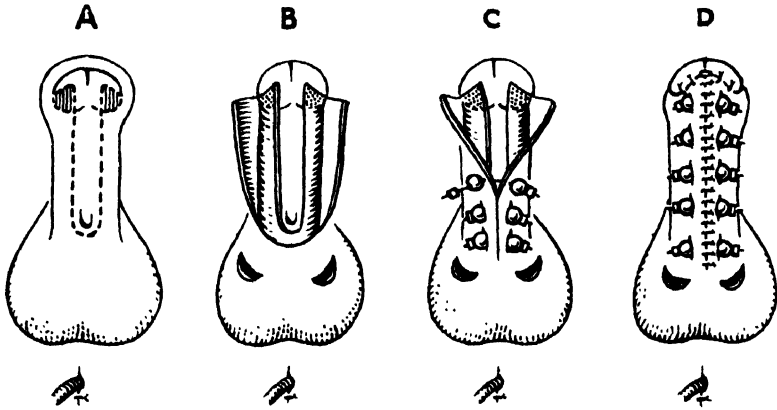


FIG. 1553.—Denis Browne's operation for hypospadias. The buried strip of skin forms the new urethra.

EPISPADIAS

Unlike hypospadias, epispadias is very rare, occurring in one in 30,000 males and one in 400,000 females. The defect may be glandular (fig. 1554), penile, or total, the latter usually associated with ectopia vesicæ. In the first two varieties the urethral orifice is situated on the dorsum, and in the second variety the penis curves upwards as well.

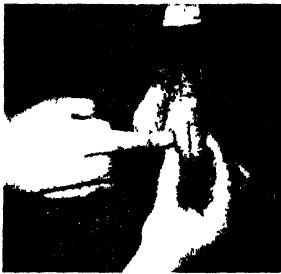


FIG. 1554.—Epispadias glandularis.

Treatment.—The operative treatment is similar in principle to that of hypospadias, and is somewhat less difficult to perform.

INJURIES

Rupture of the urethra is divided into two distinct classes—rupture of the bulbous urethra, and rupture of the membranous urethra (*syn.* intrapelvic rupture) (fig. 1555). Each is again subdivided into complete and incomplete which relates to the circumference of the urethral wall, and total and partial which relates to the thickness of the wall.

RUPTURE OF THE BULBOUS URETHRA

Rupture of the bulbous urethra is the more common accident. Almost without exception there is a history of a fall astride a projecting object. In the days of sailing-ships, the common cause was falling astride a spar from aloft. To-day a loose manhole cover (fig. 1556) and cycling accidents account for a number of cases.

Clinical Features.—The triad of signs of a ruptured bulbous urethra is urethral hæmorrhage, a perineal hæmatoma, and retention of urine.

Preliminary Treatment and Investigation.—If the condition is suspected, in order to diminish the possibility of extravasation, *the patient should be told not to try to pass urine.* No attempt should be made to catheterise him until he is fit to be taken to an operation theatre, where asepsis can be

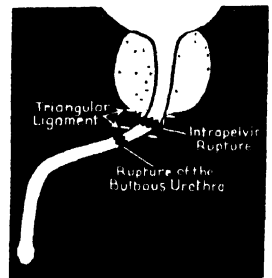


FIG. 1555.—Showing the two varieties of rupture of the urethra.

assured and operation can be undertaken in necessary cases. A suitable dose of morphine is given, a course of chemotherapy started. When circumstances are extenuating and the bladder is full, it should be emptied by suprapubic puncture (p. 1128) until the patient can be treated as above. If the patient has passed urine when first seen and there is no extravasation, then the rupture is partial and catheterisation should be avoided.



FIG. 1556.—The type of accident which results in rupture of the bulb of the urethra. (After V. J. O'Connor.)

Treatment.

1. Under strict aseptic precautions an attempt is made to pass a plastic coude catheter (14–16 F). The roof of the urethra is the most likely part to be intact, so the **beak** of the catheter should be directed towards this.

2. If the catheter passes it should be left in position for forty-eight hours. A perineal hæmatoma must be drained, and the mid-line wound packed.

3. If the catheter fails to pass, the site of the obstruction is explored, with the patient in the lithotomy position, through a mid-line perineal incision. Should it be impossible to define the proximal end of the ruptured urethra, a suprapubic cystotomy is performed and a fully curved sound passed down from the internal meatus. The exposed ends of the urethra are cleaned and the ventral aspect split longitudinally for 1.5 cm. The circumference is then sutured as a flat ribbon, with fine catgut, the knots being left on the outside of the wall (fig. 1557). The bladder, if opened, is drained by a suprapubic catheter. If the repair has been accomplished without cystostomy the catheter can be passed into the bladder from the urethrostomy (fig. 1557(B)). A small balloon Gibbon-type catheter (12 F) is very satisfactory. The perineal wound is packed, not stitched.

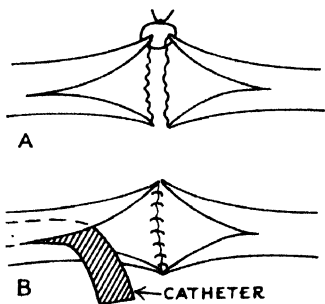


FIG. 1557.—Repair of ruptured bulbous urethra (see text).

After-treatment.—The course of chemotherapy (Urolucosil 100 mg. six-hourly) is continued until healing is complete. The urine must be recultured after a week. The perineal wound is irrigated daily with a weak Eusol (hypochlorite) solution. The catheter is removed after eight to ten days. A perineal leak will persist for a few days. Sounds must be passed two weeks after healing to calibrate the urethra, and a urethrogram, performed two months later, will reveal the result.

Complications:

Subcutaneous extravasation of urine occurs in total rupture if the patient attempts to pass urine (p. 1186).

Stricture.—This common complication is due, for the most part, to infection (p. 1193).

RUPTURE OF THE MEMBRANOUS URETHRA (syn. INTRAPELVIC RUPTURE)

Intrapelvic rupture of the urethra occurs in the membranous portion near the apex of the prostate (fig. 1555). It is a complication of a fracture of the true pelvis or dislocation of the symphysis pubis; nevertheless, only 6 per cent. of cases of fractured pelvis are thus complicated. Blood, and later urine, are extravasated into the prevesical space and, because the puboprostatic

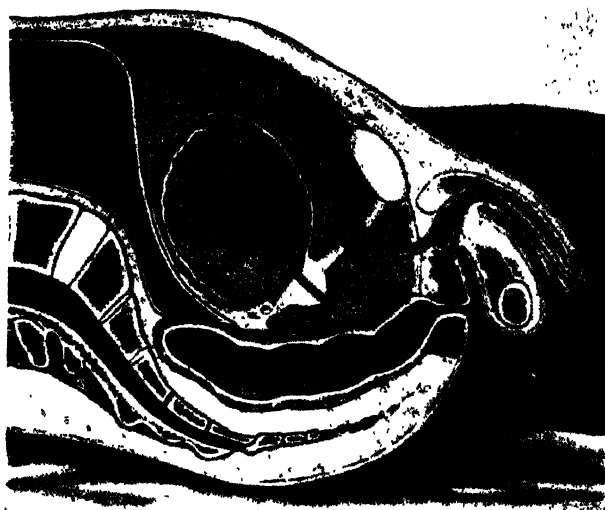


FIG. 1558.—Intrapelvic rupture of the urethra. Note the displacement of the bladder backwards, due to the tearing of the pubo-prostatic ligaments.

ligaments are torn, the prostate and the bladder are displaced backwards and upwards, thus producing wide separation of the ends of the severed urethra (fig. 1558).

Clinical Features.—

Signs of shock prevail for some hours, and those of a fractured pelvis are also present. Usually urethral hæmorrhage is trivial in amount, or absent. Often it is only when the patient has not passed urine since the accident and hypo-

gastric pain increases that signs of rupture of the urethra become manifest. On abdominal examination an ill-defined swelling is felt in the hypogastrium, usually more in evidence on one side than the other. In some cases the rounded dome of the distended bladder can be distinguished from the swelling caused by the prevesical extravasation. Per rectum the prostate cannot be felt; the area normally occupied by it is tender and unsupported.

Investigation.—(1) Radiographs of the pelvis are essential. Fractures involving the symphysis or the pubic rami may be associated with a ruptured urethra and in all such cases. (2) Catheterisation under strict aseptic precautions must be attempted. If clear urine is obtained the urethra is intact. A cystogram should be performed to exclude rupture of the bladder. Blood-stained urine indicates a rupture of the urethra or bladder. One should be mindful lest the withdrawal of a few ounces of blood-stained urine from the prevesical space (fig. 1558) be mistaken for an entry into the bladder. Careful instillation of 10 ml. weak Hypaque (diatrizoate) solution, with further radiographs, will assist the diagnosis.

Treatment of Ruptured Membranous Urethra.

Other injuries may be present and must be evaluated. Blood loss must be corrected promptly.

The first step of the operation is to make a suprapubic incision which opens the prevesical space. It is only after this has been done that it is possible to distinguish with certainty between an extraperitoneal tear of the bladder (p. 1124) and an intrapelvic rupture of the urethra. The guiding rule is, if the bladder is even moderately distended the lesion must be situated below the vesical sphincter. Thus the diagnosis of intrapelvic rupture of the urethra is confirmed. Direct suture of the membranous urethra is impracticable, yet it is imperative to bring into direct apposition the widely separated ends of the urethra. Therefore the bladder is opened suprapubically and a metal bougie is passed through the internal meatus to the seat of the rupture. A

second metal bougie is passed from the external urinary meatus. The two bougies are manipulated until their tips touch (fig. 1559). By slowly withdrawing the first bougie and steadily advancing the second, while keeping their tips in contact, it is possible to guide the second bougie into the bladder past the seat of the rupture. The first bougie is withdrawn and a piece of plain rubber tubing of such a size as to fit tightly is threaded on to the beak of the second bougie, which is withdrawn, carrying with it the rubber tubing. Outside the external urinary meatus the bougie is disengaged from the rubber tubing, and to the latter is fastened, by means of a stitch, the tip of a Foley's catheter. By pulling on the vesical end of the rubber tubing the tip of the Foley's catheter is drawn into the mouth of the suprapubic wound, where it is disconnected from the rubber tube. At this stage it is advisable to tie a long piece of silk firmly to the tip of the Foley's catheter, and to wind the excess around a wooden spatula. (Thus, should the balloon burst, the Foley's catheter can be replaced by the rail-road method just described). This completed, the balloon (30 ml.) of the catheter is moderately distended with water and the side tube is ligated (fig. 1561).



FIG. 1559.—Intrapelvic rupture of the urethra. Showing the tips of the metal bougies in contact.

Concluding the Operation.—The catheter is drawn down so that the balloon rests upon the bladder neck.

The bladder is closed around a Malécot catheter (fig. 1560), and the prevesical space is drained with corrugated rubber.



FIG. 1560.—Malécot catheter.

After-treatment:

Urological.—(1) By the mediation of a length of silk, attached to the bell end of the catheter and transfixing both walls of the catheter, extension is applied to the catheter via a pulley and a weight; thus the severed ends of the urethra are brought into contact (fig. 1561).

The weight applied is 2 lb. (900 G.) for the first three days and 1 lb. for the next ten days. Bed-cages are arranged so as to prevent the bedclothes touching the taut silk cords.

(2) Suprapubic drainage of the bladder is maintained for fourteen to twenty-one days.

(3) The urine is kept alkaline and antibiotic treatment

is given until the patient has a sterile urine and completely healed wounds.

(4) On or about the twelfth day the Foley's catheter is removed, and during the process the silk attached to its tip is unwound from the spatula; thus the silk lies along the whole length of the urethra, one end being tied to the spatula and the other emerging from the external urinary meatus. The suprapubic tube can be clipped off at about the fourteenth day, and if the

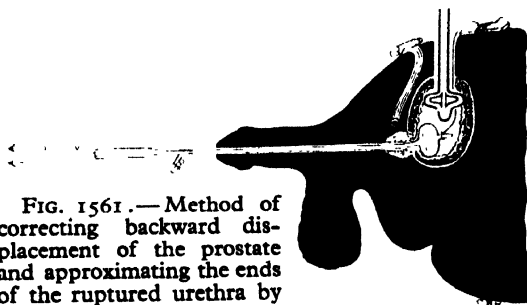


FIG. 1561.—Method of correcting backward displacement of the prostate and approximating the ends of the ruptured urethra by traction on a Foley's catheter.

patient passes urine easily is can be removed. The silk thread should be retained for the passage of bougies by the rail-road method at the end of another week, and then it should be removed, unless the insertion of another catheter becomes necessary.

Orthopædic.—For management of the fractured pelvis, see p. 213.

EXTRAVASATION OF URINE

Superficial extravasation occurs in neglected cases of complete rupture of the bulbous urethra, i.e. when operation is not undertaken within twelve to twenty-four hours, and in ruptured periurethral abscess.

The extravasated urine cannot pass (1) behind the mid-perineal point, because of the attachment of the perineal (Colles's) fascia to the triangular ligament; (2) into the thighs, for the deep layer of the superficial fascia of the abdominal wall (Scarpa's fascia) blends with the pubic portion of the fascia lata just distal to the inguinal ligament; (3) into the inguinal canals, because of the intercolumnar fibres and fascia of the external oblique (external spermatic fascia).

It therefore must pass (1) into the scrotum; (2) beneath the superficial fascia of the penis; (3) up the abdominal wall beneath the deep layer of the superficial fascia (fig. 1562).

Treatment.—Urgent operation is a necessity.

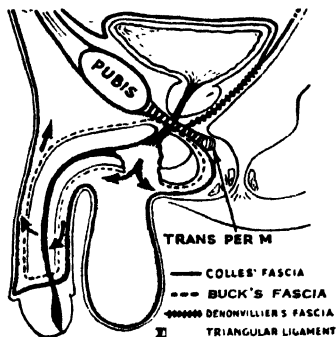


FIG. 1562.—The fascial planes concerned in superficial extravasation of urine. Trans. Per. M = Transverse Perineal muscle.

is closed around a de Pezzer catheter. The perineal wound is left widely open. The urethral catheter is removed after three or four days. In other respects the after-treatment is similar to that for complete rupture of the bulbous urethra.

Deep extravasation (fig. 1563) occurs in the case of extraperitoneal rupture of the bladder, intrapelvic rupture of the urethra, and after suprapubic puncture, when the bladder has been allowed to refill. It can also result from rupture or perforation of a ureter, or unnoticed damage to the bladder or prostatic capsule

Multiple incisions are made in the infiltrated tissues of sufficient depth to penetrate the limiting fascia. By the time extravasation has occurred it is unlikely that the urethra can be repaired in the way described already (p. 1188), for the sutures would cut out of the oedematous inflamed tissues. It is therefore often advisable to adopt the sutureless operation of Rutherford. Suprapubic cystostomy is performed and a metal bougie is passed through the internal urinary meatus. The patient having been placed in the lithotomy position, another metal bougie is passed from the external urinary meatus to the perineum, and perineal section is carried out. A Foley's catheter is introduced from the external urinary meatus into the bladder in the same way as that described for intrapelvic rupture of the urethra, and the bladder

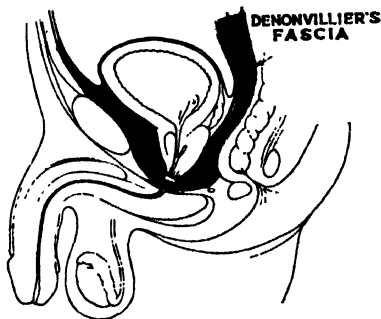


FIG. 1563.—Area occupied by extravasated blood and urine in the case of intrapelvic rupture of the urethra.

Abraham Colles, 1773–1843. Surgeon, Dr. Stevens' Hospital, Dublin. Professor of Surgery for thirty-two years.
 Antonio Scarpa, 1752–1832. Anatomist and Surgeon. Venice.
 Gurdon Buck, 1807–1877. Surgeon, New York Hospital.
 Charles Pierre Denonvilliers, 1808–1872. Professor of Anatomy, later Professor of Surgery, Paris.
 Henry Rutherford, 1861–1929. Surgeon, Glasgow Royal Infirmary.
 Oscar Michel Benvenuto de Pezzer, 1853–1917. Assistant in the Urological Department, Necker Hospital, Paris

during per-urethral resections. In these conditions urine extravasates in the layers of the pelvic fascia and in the retroperitoneal tissues.

Treatment.—When extravasation is proceeding from the bladder, it is necessary to drain the prevesical space (cave of Retzius) and to perform suprapubic cystostomy. The treatment of the various conditions which give rise to deep extravasation of urine is considered in the appropriate sections.

INFLAMMATIONS

ULCERATION OF THE URETHRAL MEATUS

Ulceration of the meatus in young male children (fig. 1564) is a common clinical entity. It is never found in the uncircumcised. It is common after circumcision, though an interval of three to eighteen months may elapse between the operation and the onset of symptoms. Lack of protection given by the prepuce is the initial cause. Friction of the clothing and ammoniacal urine are important secondary ætiological factors. The ulcer causes a scab to form which closes the meatus, and the child can only urinate by bursting this scab (fig. 1564). This process is usually accompanied by pain and screaming, and a few drops of blood may be passed. Ulceration and scab formation alternate, and if neglected, cicatricial contracture of the meatus may result eventually in an acquired pin-hole meatus.

Treatment.—A simple regimen of washing the napkins in boric acid crystals after boiling and applying 5 per cent. boracic ointment locally is usually curative. These measures neutralise the alkaline urine. A few cases require meatotomy (p. 1183).

URETHRITIS

Gonorrhœa (p. 31).

Non-gonococcal urethritis has become a world problem; it is now nearly as common as gonorrhœa in the male. The incubation period is somewhat longer than that of gonorrhœa. The cause of this infection has yet to be discovered. An organism of the pleuro-pneumonia group has been incriminated, but at the present time this is considered not to be the cause, but an early symbiotic intruder. In most instances the infection is the result of coitus with a woman suffering from leucorrhœa and a cervical erosion, and whenever possible the female partner should be examined and treated, otherwise reinfection is probable. While in many cases organisms in the discharge are absent, in others, especially those of long standing, a profuse and varied flora is found. Undoubtedly most of these organisms are the result of secondary infection.

The symptoms are identical with those of gonorrhœa, and the diagnosis can only be established by failing to find the gonococcus after intensive investigation on several occasions.

Treatment.—Non-gonococcal urethritis is a self-limiting disease, which makes the results of treatment difficult to assess. Prebble has found that urethral irrigations with oxycyanide of mercury in a strength of 1 in 8,000



FIG. 1564.—Ulceration of the urinary meatus in a child of one year.

and the temperature of the irrigating fluid 105° F. (40.5° C.) given once daily for three to seven days is successful in 85 per cent. of cases. In some quarters treatment by local irrigation is decried as old-fashioned, whereas treatment exclusively by antibiotics (which are very costly but are less troublesome to administer) are in favour generally. Good results have been obtained by oxytetracycline and also spiramycin, given orally four times a day for five days (Willcox). Furacin urethral suppositories quickly render the patient asymptomatic, but on withholding the treatment early relapse is the rule. In most instances refractory cases are due to misdiagnosis, the most common being overlooking the presence of a urethral polypus or especially a trichomonas infection.

Urethritis due to *Trichomonas Vaginalis*.—Although infection due to *Trichomonas vaginalis* is regarded as a disease of women, there is increasing evidence of its occurrence in men as well; indeed, the carrier of the disease is often a male in whom the parasite causes no symptoms. In men, the small number of parasites present in the discharge, and the difficulty in finding them, accounts for the fact that infection with *Trichomonas vaginalis* is not widely recognised, and such infection is considered to be due to non-gonococcal urethritis. The type of discharge is in no way diagnostic: it is slight to moderate in amount, greyish in colour, and of a thin or mucoid nature. It is only by examining at once a specimen of the discharge as a 'hanging drop' preparation that this protozoon (fig. 1565) can be identified. *T. vaginalis* urethritis in the male often proves a most difficult infection to eradicate, the treatment being handicapped by the high rate of reinfection, which emphasises the necessity for treating the female partner as well as the male. The most promising results have followed treatment with metronidazole, 200 mg. by mouth t.d.s. for ten to fourteen days.

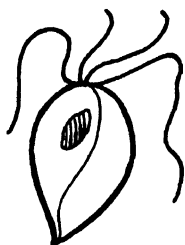


FIG. 1565.—*Trichomonas vaginalis*.

REITER'S DISEASE

The condition is venereal in origin and commences as a subacute urethritis four to six weeks after contact. The discharge is usually devoid of organisms, being clear and viscid. A few days later conjunctivitis occurs, at first unilateral, and then bilateral. Usually in ten days to two weeks arthritis supervenes (acute hydrarthrosis of at least one joint is present in every case). Another concurrent manifestation that often accompanies the onset of arthritis is keratoderma blennorrhagicum, consisting of nodules, vesicles, and pustules, frequently found on the soles of the feet. Pleuropneumonia-like organisms have been isolated from the various lesions of this condition in approximately 30 per cent. of cases.

Differential Diagnosis.—In untreated gonorrhoea, arthritis and ophthalmic infection were not rare in the past. The absence of the gonococcus is of the highest importance in coming to the conclusion that the symptoms are due to Reiter's disease. In Reiter's disease the urethritis, as well as the arthritis, is milder and the incubation period is much longer than in gonorrhoea. Furthermore, never are the ocular manifestations of Reiter's disease so destructive as those of gonorrhoea.

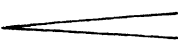
Prognosis.—The urethritis and conjunctivitis frequently subside in a few weeks, but more often than not the arthritis persists for many months.


Treatment.—While the ophthalmic complications must be treated thoroughly following the usual lines (eye-baths and shades), there is no evidence that any form of

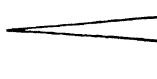
treatment at present in use has any influence on the course of the disease (Fowler). Various antibiotics and fever therapy induced by graduated doses of triple typhoid vaccine (TAB) have been tried, the last being given for the arthritis.

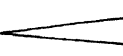
URETHRAL STRICTURE

Congenital.

Traumatic  Bulbous.
Membranous.

Inflammatory  Post-gonorrhœal
Post-urethral chancre.
Tuberculous,

Instrumental  Indwelling catheter.
Following the passage of a large calibre
endoscope, notably a resectoscope.

Post-operative  Prostatectomy.
Amputation of the penis.

Post-gonorrhœal stricture, which has become very much less common since the introduction of the antibiotic treatment of gonorrhœa, is situated most frequently (*a*) in the bulb (70 per cent.); (*b*) at the peno-scrotal junction; (*c*) in the distal part of the spongy urethra, in that order. The membranous and prostatic parts of the urethra are exempt.

Multiple strictures are relatively common. When there are two strictures, the deeper is the narrower; when there are three strictures, the deepest is the narrowest. If a stricture in the penile urethra has a very narrow orifice, there is seldom another stricture behind it.

Pathology.—Following inadequately treated gonorrhœa, infection persists in the periurethral glands, and spreads to the periglandular tissues, which become infiltrated with round cells and fibroblasts. Gradually the infiltrated periurethral tissues contract with the formation of scar tissue, localised thrombophlebitis of the corpus spongiosum playing a part in the more dense varieties. Whereas in the bulbous urethra the fibrosis is most in evidence in the roof, in the penile urethra it predominates in the floor. Most strictures develop during the first year after gonorrhœal infection, but they may not give rise to difficulty in micturition for ten to fifteen years.

Clinical Features.—In a stricture of large calibre the only symptoms are the occasional passage of flakes (desquamated epithelium) in the urine and a varying amount of urethral discharge (gleet), most in evidence in the early morning (the morning 'dew-drop'). Often these symptoms are neglected until the diminished calibre of the urethra causes considerable difficulty in micturition. In contradistinction to obstruction due to an enlarged prostate, the patient finds he must strain to empty the bladder. Another distinguishing feature is the patient's age. He is often considerably younger than the prostatic sufferer, or the symptoms date back to some time prior to the fiftieth year. The stream becomes progressively narrower, micturition is prolonged, and after it has seemingly ended dribbling occurs. This is due to urine trickling from the dilated urethra above the stricture. Increasing frequency of micturition, at first during the day and then both by day and by night, is another common complaint, due either to incomplete emptying of the bladder at each act of micturition, or to cystitis, or to both. In long-

standing cases it is often possible to palpate the stricture from without as an induration in the urethral floor. The evil effects of urethral obstruction upon the bladder, ureters, and kidneys are similar to those of prostatic obstruction (p. 1162). Untreated, sooner or later retention of urine supervenes. Sometimes acute retention sets in while the stricture is still of moderate calibre; it is then due to superadded œdema of the urethral mucous membrane in the neighbourhood of the stricture, brought about by voluntary retention, alcoholic excess, or recrudescence of local infection. In other cases narrowness of the stricture results in increasing inability to expel residual urine, and acute-on-chronic retention, or retention-with-overflow, supervenes.

Urethroscopy renders the diagnosis of urethral stricture very precise.

The stricture can be seen as a white scar of fibrous tissue, and its position in the urethra, the size of its contained lumen, and its dilatability can be judged (fig. 1566).

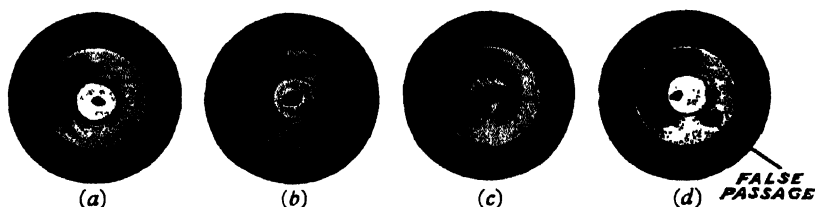


FIG. 1566.—Urethroscopic appearances. (a) Fine-bore stricture. (b) Moderate-bore stricture. (c) Crescentic stricture of the roof. (d) Stricture with false passage.

Often a stricture encircles the whole urethra, and the lumen is more or less centrally placed, but from time to time a variety of urethroscopic appearances is noted in individual cases; for instance, the stricture may take the form of a crescent.



FIG. 1567.—Urethrogram showing a stricture of the membranous urethra following fracture of the pelvis.

Urethrography (p. 1066) supplies information concerning the length of a stricture, of dilatation or diverticulum formation above the stricture, or failure of the medium to pass a stricture (figs. 1567 and 1568).

INSTRUMENTAL TREATMENT

Intermittent Dilatation.—In the majority of instances, provided the

False passages may be seen. They are recent penetrations of the urethra in front of the stricture due to unskilful attempts to pass a bougie. False passages are particularly liable to bleed, and it is essential to abandon æro-urethroscopy if urethral hæmorrhage occurs, for fatal air embolism has resulted from air being pumped into the cavernous tissue through a urethral wound.



FIG. 1568.—Gonorrhœal stricture of the bulbous urethra.

patient attends regularly, dilatation at suitable intervals is a satisfactory form of treatment. Before each dilatation the patient should pass urine, the glans penis and urinary meatus are cleansed and the urethra is filled with a mild antiseptic solution, and local anæsthetic in a methyl cellulose base (Lidothesin). Dilatation must be carried out gently with bougies of increasing size.

On no account should the stricture be forcibly or over-dilated, both of which result in traumatisation that induces inflammatory œdema and subsequent formation of more fibrous tissue.

Strictures of very small calibre should be dilated twice a week at first. With this exception it is sufficient for the patient to attend for treatment at regular intervals, and an erstwhile reminder to the patient was that he must return for treatment 'once a week for a month, once a month for a year, and every year on his birthday'. A few strictures are cured by full dilatation, but in the majority the patients must be kept under supervision for the remainder of their lives.

Gum-elastic bougies (*syn.* French bougies) (fig. 1569) should be the standard instruments in the early stages of treatment of all but the very finest strictures. It is



FIG. 1569.—Gum-elastic bougie.

usual to commence with a No. 10 French, and if this cannot be passed, progressively smaller, or even filiform bougies are employed.

Filiform bougies are gum-elastic bougies varying in size from 1 to 3 French. If one fails to pass, it is often valuable to insert several as far as the stricture. By manipulating each back and forth, frequently one of them can be induced to negotiate the stricture (fig. 1570). To distend the urethra with sterile olive-oil and apply a penile clamp to the glans penis prior to attempting the foregoing method adds to the chances of its success, because obstructing folds of mucous membrane are separated by the oil distension. If this method fails, it is sometimes possible to pass a filiform bougie through the stricture under the vision afforded by a urethroscope. If any



FIG. 1570.—'Faggot method of introducing a bougie through a stricture.

bleeding has been occasioned by the attempt to pass bougies, urethroscopy should be postponed for several days. Very few urethral strictures are impassable, but it often requires patience to insinuate even the finest urethral guide: only when even a filiform bougie cannot be passed on three successive occasions is a stricture held to be impassable.



FIG. 1571.—Filiform bougie with follower.

Filiform Bougies with Followers.—Filiform bougies furnished with threaded hollow mounts at their proximal ends are to be preferred, because screw-ended gum-elastic bougies of a larger size can be attached and guided by the filiform through the stricture. In this way many strictures of very small calibre can be

dilated sufficiently to render subsequent dilatations less tedious.

By means of gum-elastic bougies a stricture is dilated up to the size of 14 French. Thereafter metal bougies are to be preferred.

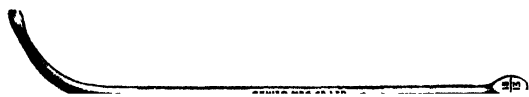


FIG. 1572.—Lister's metal bougie.

English bougies) (fig. 1572) are indicated after the second or third attendance of the patient, when it has been proved that the stricture is responding to dilatation by the French bougies. Metal bougies of a smaller size than 7/9 English should not be used, for fear of making a false passage.

Lister's metal bougies (*syn.*

Continuous dilatation necessitates some days of in-patient treatment, but it is of immense benefit in cases where little or very slow progress is made by intermittent dilatation. In a number of instances continuous dilatation obviates the necessity for operative treatment. After dilatation with gum-elastic bougies a catheter of corresponding size is tied into the urethra. On its removal two or three days later it will be found possible to pass much larger bougies.

OPERATIVE TREATMENT

1. External urethrotomy (Wheelhouse's operation) is the operation of choice when a patient presents with acute retention and the stricture proves impassable.

With the patient in the lithotomy position the Wheelhouse staff (fig. 1573) is passed down to the face of the stricture, and the urethra distal to the stricture opened by cutting down on to the groove in the staff. Stay sutures hold the edges open to display the face of the stricture. The mid-line incision is then carried backwards

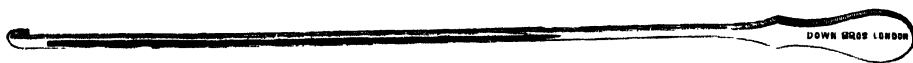


FIG. 1573.—The Wheelhouse staff.

until the dilated proximal dilated urethra is opened. Urine will usually leak out under some pressure. A catheter is now passed from the external meatus across the opened urethra into the bladder, and the wound packed lightly. *Alternatively* the operation can be concluded formally as the first stage of a urethroplasty (see below) by stitching skin edge to mucosal edge.



FIG. 1574.—Thomson-Walker's urethrotome.

2. Internal urethrotomy is now a rare procedure. It can only be used if the stricture is passable. A triangular cutting knife (fig. 1574) runs in a grooved staff, which is guided through the stricture by a fine gum-elastic bougie. The stricture is

divided by sharp thrusts of the knife. Intermittent dilatation by bougies should be commenced fourteen days later.

3. Urethroplasty.—Nowadays surgical cure by means of a two-stage urethroplasty is applicable to all strictures of the bulbar and penile portions, and is the treatment of choice.

Johanson's Urethroplasty.—The circular constricting tissues should be completely divided, and the normal urethra opened at either end for a distance of about 2 cm. Skin and mucosal edges are united to prevent the urethral edges from rejoining (fig. 1575). Contraction of the damaged tissues and of the incisional scar will occur, but three months later—employing the principle used in the Denis Browne repair of a hypospadias (p. 1185)—a strip of skin, which has obtained a blood supply from deep tissues and the residual urethral wall, can be buried to form a new penile urethra and distal bulb.

For the second stage of the repair of the posterior part of the bulb, a funnel of scrotal skin will have to be used to reach the upper end of the urethral defect (fig. 1576).

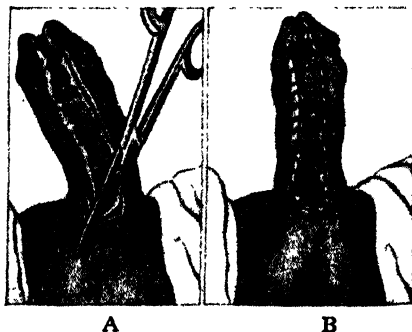
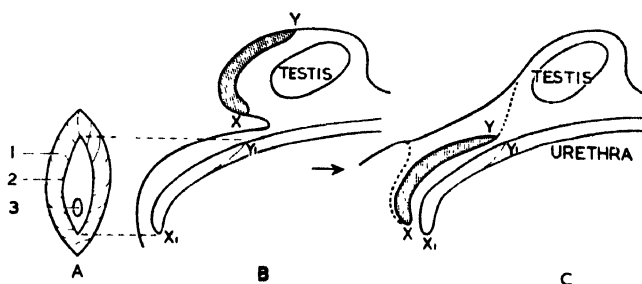


FIG. 1575.—Johanson's urethroplasty.

FIG. 1576.—Urethroplasty for the posterior part of the bulb using a funnel of scrotal skin.



OTHER CAUSES OF URETHRAL STRICTURE

Congenital stricture has been considered on p. 1183.

Traumatic stricture follows unskilful or delayed treatment of rupture of the bulbous urethra. If dilatation is unsatisfactory, a urethroplasty should be done.

The so-called stricture following rupture of the membranous urethra is often not a stricture at all but a complete loss of continuity of the urethra (fig. 1558). If, as frequently occurs, it proves impossible to keep the passage dilated by the usual methods, the only alternative to transplantation of the ureters is Badenoch's pull-through operation. After the bladder has been opened suprapubically, with the patient in the lithotomy position, a metal sound is passed up the urethra and when arrested by the stricture its tip is cut down upon in the perineum. The distal urethra is then freed as a tube for 4 to 6 cm. A large metal sound is passed suprapubically down the urethra and cut down upon from the perineum until it can be forced through the area of fibrosis. A rubber catheter is attached to it by the butt end and pulled back through the suprapubic opening. The freed distal urethra is threaded over the lower end of the catheter and its end stitched to the catheter some 10 cm. (4 inches) from its tip with interrupted chromic catgut stitches. Traction on the catheter then guides the distal urethra into the prostatic urethra where it is held by attaching an artery forceps to the catheter as it emerges on to the abdominal wall (fig. 1577). Finally, the perineum is closed and a suprapubic cystostomy temporarily established.



FIG. 1577.—Badenoch's pull-through operation completed.

Post-instrumental stricture can occur in any part of the urethra from trauma due to the passage of a large-calibred endoscope, or from urethritis from an indwelling catheter. To avoid the former, some surgeons advise performing perineal urethrostomy in order to pass a resectoscope in patients with a narrow urethra.

Post-operative Stricture.—A stricture develops in about 4 per cent. of cases after prostatectomy, irrespective of the method employed. The stricture is usually situated in the proximal end of the prostatic urethra. In many cases the stricture can be dilated by regular intermittent dilatation. When the stricture takes the form of a shelf at the junction of the bladder with the prostatic bed, the bladder must be opened and the shelf excised.

Post-operative stricture can also follow partial or complete amputation of the penis. Methods of avoiding this complication are described in the section dealing with these operations (p. 1211). Regular dilatation is satisfactory.

COMPLICATIONS OF URETHRAL STRICTURE

1. Retention of urine (p. 1126).
2. Urethral diverticulum.
3. Periurethral abscess.
4. Urethral fistula.

5. All the attendant evils of 'back pressure', culminating in bilateral hydronephrosis, combined with a susceptibility to urinary infection and an increased liability to urinary calculus.

6. Hernia, hæmorrhoids, or rectal prolapse may be induced by the straining.



FIG. 1578.—A urethral diverticulum.

Diverticulum of the male urethra (*syn.* urethral pouch)

1. Congenital.
2. Due to increased intraurethral pressure behind a stricture.
3. Due to the long-continued presence of a urethral calculus.
4. Indwelling catheters in paraplegics.

In many cases the pouch can be seen (fig. 1578), and those which are not obvious at first become so when the patient interrupts the stream of urine.

Treatment is excision of the diverticulum and removal of the cause if possible.

PERIURETHRAL ABSCESS

There are two main varieties of acute abscess:

Penile periurethral abscess arises as an infection of one of the glands of Littre, and is usually a complication of acute gonococcal urethritis. A tender induration can be felt on the under-surface of the penis (fig. 1579). Left to nature, frequently the abscess bursts externally, and a urinary fistula is liable to result.

Treatment.—The passage of a bougie often causes the abscess to burst into the urethra. When this is unsuccessful, a ureteric meatotome is passed through a urethroscope and the abscess is opened by diathermy. When an abscess lies behind a stricture of the urethra it must be drained externally.

Bulbous periurethral abscess runs a variable course. Its most acute form, formerly termed periurethral abscess with extravasation of urine, is better termed periurethral phlegmon, because in 50 per cent. of cases there is no stricture of the urethra present. Of the remainder, the majority have a passable stricture, while in the minority the stricture is impermeable. Consequently, in the majority of cases there is no reason why urine should extravasate. The condition is due to a spreading cellulitis caused by streptococci and anaërobic organisms invading the same cellular plane as that of superficial extravasation of urine (p. 1190).

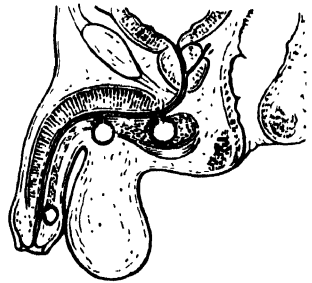


FIG. 1579.—Common situations of a periurethral abscess. (After F. Papin.)

Clinical Features.—There is pain in the perineum, a high temperature, repeated rigors, and a rapid pulse-rate. In early cases the tenderness and swelling are limited to the perineum. Later, often in a matter of hours, the scrotum and then the penis become œdematous, and the infection spreads beneath the superficial fascia of the abdominal wall (p. 1024).

Treatment.—Antibiotics have greatly improved the prognosis.

Operation.—The perineal abscess should be opened. In addition, if spreading cellulitis is present, incisions deep enough to divide the superficial fascia are made into the infiltrated scrotum, penis, and abdominal wall, wherever considered necessary.

When there is partial or complete retention of urine, if the infection is limited to the scrotum and the penis, after changing gloves and instruments, suprapubic cystostomy should be performed. If the superficial layer of the abdominal wall is implicated, it is best to drain the bladder by performing perineal urethrostomy. It should be noted that the abscess usually communicates with the urethra by a minute opening which cannot be found.

After-treatment.—After instilling hydrogen peroxide into them, the various incisions are irrigated with weak potassium permanganate solution. Later sitz baths¹ of the same solution are given. When the infection has been overcome, the stricture of the urethra, if present, is treated by one of the methods described already.



FIG. 1580.—Chronic periurethral abscess.

Chronic periurethral abscess is nearly always situated in the perineum (fig. 1580) and is associated with much periurethritis. It is almost invariably the result of a long-standing stricture of the bulbous urethra. The abscess should be opened, together with the various pockets that are often present. Later the associated stricture must receive adequate treatment. The condition is liable to be complicated by a urethral fistula which occurs either spontaneously or as a result of incision of the abscess.

URETHRAL FISTULA

The most frequent cause of an acquired external urethral fistula is bursting or incision of a periurethral abscess. When the opening is situated in the penile urethra or at the peno-scrotal junction, the amount of urine that escapes at each act of micturition is often small. A fistula following a periurethral abscess of the bulbous urethra can be either single or multiple. In the latter case the fistulae originate behind a tight stricture and the patient passes most or all of his urine through the various fistulae (watering-can perineum). A fistula can also follow external urethrotomy when there is a stricture situated more distally.

Treatment.—A small fistula often closes spontaneously after repeated dilatation of the urethra. Sealing the track with the diathermy needle often encourages closure. Occasionally urethroplasty (p. 1196) is indicated.

URETHRAL CALCULUS

Calculi occur less frequently in the urethra than in any other part of the urinary tract. A urethral calculus can arise primarily in the urethra behind a stricture or in an infected urethral diverticulum. The latter are composed of phosphates. Less rarely a calculus which has migrated from the ureter becomes arrested in the prostatic, bulbous, or penile portions of the urethra. Migratory calculi are arrested in the urethra relatively frequently in children under the age of two, the explanation being that the comparatively large vesical neck allows them to pass out of the bladder.

Clinical Features.—In the case of a migratory calculus arrested in the urethra, typically there is a history of an attack of renal colic two or three days previously. During micturition the patient experiences sudden pain in the urethra and the stream of urine ceases abruptly. A few drops of blood-stained urine follow, and then there is retention of urine. A stone can be palpated readily through the floor of the urethra. When the stone has been arrested in the prostatic urethra a rectal examination usually reveals a tender, hard nodule in the middle line of the prostate, generally near its apex. A calculus forming behind a urethral stricture often does so without causing much additional discomfort and it sometimes attains a considerable size before giving rise to retention of urine or painful dysuria. In some cases such

¹ Sitz bath — in which the patient sits in a bath-tub, bathing only the hips and the buttocks.

a stone can be felt easily ; in others, owing to periurethral thickening, its presence is not suspected until it is seen at urethroscopy, or a grating sensation is experienced on passing a gum-elastic bougie or a characteristic click is heard if a metal bougie has been employed. In all but superficially placed calculi giving rise to acute symptoms, radiography is necessary to confirm the presence, and particularly to reveal the size of the calculus (fig. 1581) before commencing treatment.

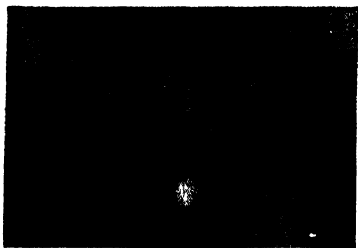


FIG. 1581.—Radiograph showing calculus impacted in the bulbous urethra.

Treatment :

(1) *Prostatic Urethra*.—When the stone is arrested in the prostatic urethra a general anæsthetic is given, and a metal bougie is inserted along the urethra as far as the calculus. In cases of recent impaction the stone can be pushed into the bladder, where it can be crushed by litholapaxy and the fragments evacuated. In cases of long-standing the stone is hour-glass shaped, and impacted firmly, in which event it is best approached through the bladder.

(2) *Membranous* and (3) *Bulbous Urethra*.—Occasionally a small stone can be removed from the deeper parts of the urethra by means of urethral forceps passed through a urethroscope. On no account must aero-urethroscopy be employed (danger of air embolism if bleeding occurs). A stone impacted in the proximal urethra sometimes can be manipulated forward if the urethra is distended with lotion. Another method is to pass a number of filiform bougies beyond the stone, twist them, and endeavour to dislodge the calculus by pulling all the bougies out together. A stone impacted in the fossa navicularis, or manipulated into this position, sometimes can be removed by urethral forceps, or by inserting a probe behind it.

Operative Measures (situations 2 and 3).—Meatotomy is often required to permit the stone to be withdrawn. When a stone cannot be removed by one of the expedients described above, an incision is made through the floor of the urethra on to the stone (external urethrotomy). After the stone has been extracted, in the absence of a stricture and gross infection, the urethra can be closed in layers.

NEOPLASMS OF THE MALE URETHRA

Polypi are usually multiple and occur most frequently in the region of the verumontanum. As seen at urethroscopy, each polypus is a pale, finger-like projection (fig. 1582) with blood-vessels coursing over it. Polypi are never found without chronic infection. Sometimes, if the infection can be cured, the polypi disappear; conversely, if polypi are destroyed, chronic urethritis, which previously resisted treatment, often responds to it. Possibly this condition should be classified as a granuloma.

Papilloma.—From time to time a solitary papilloma occurs, most often within the fossa navicularis and, as it enlarges, it protrudes from the external urinary meatus (fig. 1583).

Multiple papillomas associated with penile papillomas (p. 1209) sometimes spring from mucous membrane just within the external urinary meatus.

Papillomatosis of the urethra is most often associated with papillomas of the bladder—the posterior urethra is considerably more often affected than the anterior urethra.

The typical symptom of papilloma of the urethra is slight hæmaturia immediately preceding micturition.

Angioma is rare. The hæmaturia to which it gives rise is often profuse, and may occur independently of micturition.

Treatment of all the foregoing neoplasms is diathermy coagulation through a urethroscope. Exceptionally the neoplasm is so accessible that it can be fulgurated without employing a urethroscope.

Carcinoma is rare. Usually a urethral stricture is antecedent to the carcinoma.



FIG. 1582.—Polypi in a male posterior urethra. They occur also in the posterior urethra of females.

Spread to the inguinal lymph nodes often occurs. Blood-borne distant metastases are uncommon unless the cavernous tissue of the penis becomes implicated.

Clinical Features.—As a rule, the first and only symptom is a profuse urethral discharge. Carcinoma of the urethra is therefore one of the many causes of 'urethritis'. Later, the discharge becomes blood-stained and the symptoms of stricture of the urethra supervene. The chronic nature of the condition in a man past forty years of age, the presence of blood in the discharge or in the urine, a tendency to bleed easily during instrumentation, and above all palpable induration, are factors that lead to a suspicion of carcinoma. In the deeper parts of the urethra, sometimes the first manifestation is an indolent periurethral abscess that after incision not only fails to heal, but the periurethral induration increases.

Biopsy then establishes the diagnosis.

Treatment.—When the growth is in the anterior part of the penile urethra partial amputation of the penis can be carried out, and provided the growth has been diagnosed reasonably early, long survival can be expected. Complete amputation with the construction of a perineal meatus is required when the neoplasm is situated more posteriorly.

When the carcinoma is situated further back a still more extensive operation, which includes radical prostatectomy, is the only hope of eradicating the disease.



FIG. 1583.—Papilloma of the fossa navicularis extruding from the external urinary meatus.

THE FEMALE URETHRA

Urethritis.—As in the male, acute urethritis can be due to gonorrhœa or to non-gonococcal urethritis. Likewise chronic urethritis may or may not be due to gonorrhœa, and in its severe forms is a urethro-trigonitis. The symptoms are increased frequency, pain on micturition, urgency, terminal hæmaturia, and low back pain. Urethral tenderness is an important sign of infection of Skene's tubules. Pressure on the urethra causes pus to exude from the orifices of the ducts surrounding the urethral opening. Incomplete cure of the infection in these tubules is a source of chronicity in anterior urethritis. In cases of posterior urethritis, urethral polypi are often present and can be seen on urethroscopy.

Treatment.—In addition to appropriate antibiotic treatment, local treatment is necessary. In cases of non-gonococcal urethritis furacin urethral suppositories are sometimes beneficial. Intermittent dilatation of the urethra, by promoting drainage of infected foci within the urethra, often helps to eradicate the infection. If polypi are present, the condition is seldom remedied unless they are removed by fulguration. Swabbing the urethra with 10 per cent. solution of silver nitrate is also helpful. Excision of the distal 1 cm. of the urethral mucous membrane is the best method of curing chronic infection proved to be due to infection of Skene's tubules.

T. Vaginalis Urethritis.—About 12 per cent. of women, one-third of whom are unmarried, suffer from *T. vaginalis* vaginitis. In a large percentage of cases *Trichomonas vaginalis* infection must be regarded as a venereal disease; the way in which the remaining patients become infected is speculative. Lavatory seats are often blamed, and their culpability is almost impossible to disprove for *T. vaginalis* can survive

thereon for forty-five minutes (Whittington). On the other hand, the fact that virgins are never infected with this protozoon adds to the scepticism with which this explanation is received. Trichomonal vaginitis is complicated by urethritis in many instances. This form of urethritis responds to metronidazole (p. 1192), but the vaginitis must be treated also.

Senile urethritis is an atrophic lesion due to deficiency in endogenous ovarian hormone. Suppositories containing diethylstilbæstrol 0.1 mg. in a topical anæsthetic agent are extremely effective.

Prolapse of the Urethra.—Prolapse of the posterior margin of the urethra occurs in many women past the menopause, and is symptomless unless it is associated with senile urethritis when, on account of straining on micturition, the condition is progressive. Prolapse of the urethral mucous membrane occurs also as a congenital condition. When half or more of the circumference prolapses the local discomfort, especially on walking, is proportionate to the degree of the prolapse. The urinary meatus is examined while the patient strains: when the prolapse is complete the opening of the urethra is central; when it is partial, the opening is eccentric.

Treatment.—The associated urethritis, if present, must be treated first. Often lesser degrees of prolapse can be cured by making one or more linear grooves in the long axis of the mucous membrane of the urethra with a diathermy needle, employing the coagulating current. Subsequent contraction of the scar reduces or obliterates the prolapse. More advanced cases are treated by transfixing the whole thickness of the prolapse as far from its extremity as possible with four catgut sutures placed at equidistant intervals. Redundant urethral mucous membrane is excised distal to the sutures, which prevent retraction of the stump into the canal. The cut edges of the mucous membrane are then united.

Urethral Stricture.—An adult female urethra which fails to admit freely a No. 20 French bougie is the seat of a urethral stricture.

The causes are (a) urethritis, not necessarily gonococcal, and (b) trauma as the result of difficult labour. Inflammatory stricture is situated at the external meatus; traumatic stricture usually affects the middle or posterior part of the urethra.

Sometimes the stricture will only admit a guide, and in these cases acute retention of urine is particularly prone to occur. Dilatation of the stricture yields satisfactory results. The recognition and treatment of a stricture often clears up an obscure case of pain on micturition.

Urethral diverticulum (*syn.* urethrocele) is more common in women than in men. Arguments favour a congenital origin in one of Gartner's ducts¹, but some diverticula are acquired by (a) rupture of a distended urethral gland or (b) injury of the urethra during parturition. A small uninfected pouch may be symptomless. As the diverticulum enlarges, inability to pass all the urine at one time, or dribbling after micturition, occurs. Once infected—and infection is almost inevitable if the diverticulum is not evacuated completely at each micturition—recurrent attacks of cystitis perpetuated by reinfection from the diverticulum continue in spite of antibiotics and chemotherapy. On digital examination a swelling can be felt on the anterior vaginal wall in the line of the urethra, and when it is compressed, urine, usually obviously purulent, is expressed. If the beak of a curved metal bougie is passed gently along the floor of the urethra, from time to time the orifice of the diverticulum is large enough for the beak to enter the sac. In 10 per cent. of cases a stone forms in the diverticulum, and hæmaturia may then occur.

Treatment.—The simplest method is to open the diverticulum through an incision in the vaginal wall. After irrigating the interior of the diverticulum, the mucous membrane is curetted, and the cavity is packed lightly with oxycel. The incision in the diverticulum and vaginal wall is then closed by interrupted catgut sutures. An indwelling catheter remains in place for a week. It is presumed that fibrosis obliterates the sac (Lane). This procedure is so often successful that formal excision is seldom required, and is reserved for diverticula with wide mouths.

Calculus impacted in the female urethra is exceptional. When it occurs it can be removed by grasping it with forceps while a finger on the anterior vaginal wall presses it forward.

Urethral caruncle is common in middle-aged and elderly women. The condition presents as a soft, raspberry-like, pedunculated, granulomatous mass about

¹ The unobliterated distal end of the mesonephric duct.

the size of a pea, attached to the posterior urethral wall near the external urinary meatus. Histologically it is composed of highly vascular connective tissue stroma infiltrated with polymorphonuclear leucocytes and covered by squamous epithelium.

Clinical Features.—There is increased frequency of micturition, and often great pain during and after micturition. Terminal hæmaturia often occurs, and there may be a blood-stained discharge independent of micturition. The condition can be diagnosed on inspection, although it must be differentiated from prolapse of the mucous membrane. With a probe it can be determined that the protrusion arises from a pedicle attached to the posterior urethral wall. The mass is exquisitely tender and bleeds readily.

Treatment.—The pedicle should be divided flush with the floor of the urethra with a diathermy needle, using a cutting current, after which that portion of the urethra from which the pedicle arose is coagulated with the diathermy current. The chronic urethritis with which the condition is always associated should be treated by Furacin suppositories and intermittent urethral dilatation until the patient is symptom-free.

Papillomas acuminata occur on the external urinary meatus, and spread on to the labia minora. They differ in no respect from, are acquired in the same way as, and the treatment is similar to that of, papillomas acuminata of the penis (p. 1209). In female Africans papillomas acuminata are common, and they increase at an alarming rate during pregnancy. Towards term, so great does the neoplastic mass become that it may obstruct labour and necessitate Cæsarean section (Bowesman).

Carcinoma of the urethra occurs twice as frequently in the female as in the male. Whether or not a caruncle can become malignant is disputed, but a visible bleeding protrusion is the commonest manifestation of a carcinoma of the urethra, and the most common site of the carcinoma in its early stages is the external urinary meatus. A carcinoma commencing within the urethra gives rise to dysuria, hæmaturia, and sometimes retention of urine. Usually induration distinguishes it from an innocent tumour.

Treatment.—Interstitial irradiation by gold grains or radium needles is the method of choice. If this fails to cure the condition, the advanced state of the disease necessitates total urethro-cystectomy.

THE PENIS

PHIMOSIS

While the condition can be acquired as a result of chronic or acute inflammation of the lining of the prepuce, usually it is due to congenital narrowing of the preputial orifice, often associated with an unduly long foreskin. In extreme examples of congenital phimosis, when the patient micturates the prepuce balloons out first, and a thin, weak stream of urine follows. Dysuria with residual urine, hydroureters, and hydronephroses are rarely due to phimosis, but more often occur as a result of atresia meati which may lie hidden by the phimosis. The treatment is circumcision.

CIRCUMCISION¹

Indications.—(a) *In infants and young boys*, because of a request by the parents (religious and personal); because of recurrent balanitis with inability to retract the prepuce; and, rarely, because of a very long prepuce. Except



FIG. 1584.—A urethral caruncle.

¹ Apparently circumcision did not originate among the Jews: they took the custom from either the Babylonians or the Negroes, probably the latter. It has been practised in West Africa for over 5,000 years.

for the ritual operation most circumcisions are unnecessary: it is normal for the prepuce to be long and adherent to the glans within, for these parts become satisfactorily separated and the prepuce mobile in the first few years of life. Recurrent balanoposthitis and phimosis often follow attempts by the parents forcibly to retract the prepuce. (b) *In adults*, because of inability to retract for intercourse, a tight frenum, balanitis, and sometimes prior to radiotherapy for carcinoma (a dorsal slit may suffice).

Technique in an Infant.—The parts are washed with soap and water, and painted with a non-irritating antiseptic. If it is not possible to retract the prepuce, the preputial orifice should be stretched with a hæmostat in order that the post-coronal sulcus can be cleansed of contained smegma. A pair of sinus forceps or, better still, bone

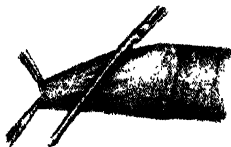


FIG. 1585.—Applying sinus forceps preparatory to excising the redundant skin.

forceps, is applied obliquely to the prepuce, parallel to the corona, just beyond the tip of the penis (fig. 1585), and the skin distal to the forceps is excised with scissors close to the forceps, which are then removed. The outer layer of the preputial skin retracts, leaving the glans covered by the inner layer, which is divided in the middle line anteriorly by introducing a blade of delicate scissors between it and the glans. Each leaf of the deep layer of skin (formerly called the mucosa) is excised, leaving a narrow collar around the corona. The frænal artery usually requires ligation (fig. 1586), as well as several small vessels on the dorsum. The cut edges of the deep and superficial layers are

approximated with fine catgut sutures. A wisp of cottonwool soaked in tinct. benzoin co. makes an excellent waterproof dressing, which becomes detached when the patient is sat in a bath six days later.

In adolescents and adults the following method is preferable. The prepuce is retracted until its tense orifice is apparent, or until the tip of the glans comes into view, and on to the edge of the prepuce are placed three hæmostats, one in the middle line ventrally and two on either side of the middle line dorsally. The prepuce is then slit up in the middle line dorsally to within $\frac{1}{2}$ inch (1.25 cm.) of the corona. The under-surface of the prepuce having been completely separated from the glans and the corona, the layers of each flap are excised, keeping $\frac{1}{2}$ inch distal to the corona. The superficial layer is retracted and bleeding-points are secured and ligated. The inner layer of the prepuce having been trimmed to $\frac{1}{4}$ inch (0.3 cm.) from the corona, the two cut edges are approximated accurately with fine interrupted catgut stitches



FIG. 1586.—The 'four-in-one' frænal stitch.



FIG. 1587.—Method of circumcision in an adult described in the text.

(fig. 1587). The cut edges in the immediate vicinity of the frænum can be neatly drawn together by a mattress suture. The ends of appropriately placed stitches can be left long to anchor a circular dressing of ribbon tulle gras.

PREPUTIAL CALCULI

Late in life, chronic posthitis resulting in fibrosis further constricts an already inadequate orifice of a preputial sac. When the prepuce has not been retracted nor

its interior cleansed for many years, preputial calculi are wont to form. Three types are described: 1. Those resulting from inspissated smegma. 2. Those consisting of a mixture of smegma and urinary salts. 3. Those consisting entirely of urinary salts. The treatment is circumcision.

PARAPHIMOSIS

The tight prepuce has been retracted but cannot be returned, and it is constricting the glans which is engorged and œdematous. The diagnosis is apparent at a glance.

Treatment.—One ml. of normal saline containing 150 turbidity units of hyaluronidase is injected into each lateral aspect of the swollen ring of prepuce. Fifteen minutes later the swelling is much reduced, and in early cases reduction (fig. 1588) can be accomplished with ease. If this is un-



FIG. 1588.—Reducing a paraphimosis.

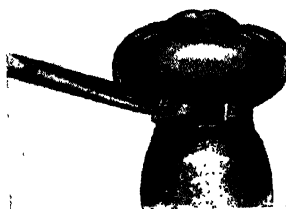


FIG. 1589.—Excising the constricting band.

successful, a general anæsthetic must be given and the constricting band is incised, and the narrow cuff of skin which formed the constricting band excised (fig. 1589). There is now no obstruction to reduction, and there remains only to remove a broad cuff of normal skin on the proximal side of the gap and unite the cut free edges, thus performing circumcision.

INJURIES

Avulsion of the Skin of the Penis.—Entanglement of clothing in rotating machinery accounts for the majority of these injuries. Repair can be carried out by burying the shaft of the penis in the scrotum (fig. 1590), with subsequent surgical release at a propitious time.

Fracture of the penis is a very uncommon accident due, most often, to the erect organ being bent forcibly downwards. Following trauma, the erect organ suddenly becomes flaccid. The extravasation of blood, which is considerable, causes great pain and swelling. In early cases incision, clearing out blood-clot, and suture of the ruptured corpus cavernosum has yielded good results.



FIG. 1590.—Covering the denuded shaft of the penis by burying it in a scrotal tunnel.

STRANGULATION OF THE PENIS BY RINGS (fig. 1591).

Removal is prevented by venous engorgement. Consequently aspiration of the corpora cavernosa may assist in removal of the ring in early cases, otherwise a ring cutter or a hacksaw must be employed. If a tight ring is not removed within six hours, unless the bladder is drained suprapubically, or perineal urethrostomy performed, rupture of the urethra with extravasation may ensue.

INFLAMMATIONS

Balanoposthitis.—An inflammation of the prepuce is known as posthitis; an inflammation of the glans penis is called balanitis. Frequently the opposing surfaces of the prepuce and the glans are implicated in the inflammatory process—hence the term balanoposthitis.

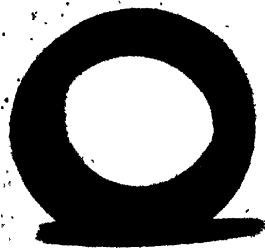
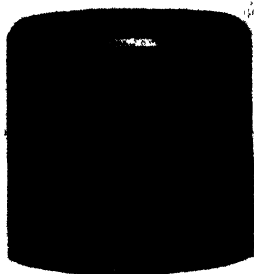


Fig. 1591.—This hard steel ring was removed one Christmas Eve from the base of the penis after aspiration of the corpora to remove blood, and multiple punctures with hyaluronidase had allowed the œdema to be reduced by squeezing (two-thirds actual size). (*P. F. Philip F.R.C.S., London.*)

It is imperative to bear in mind that cancer, chancre, or sugar in the urine may be the primary factor, and that the condition is nearly always associated with some degree of phimosis (fig. 1592). In mild cases the only symptom is itching and some discharge. Acute cases show a red, swollen and tender foreskin with exuding pus.



(A)

(B)

FIG. 1592.—(A) A discharge was the patient's complaint. On retracting the prepuce a Hunterian chancre (B) was revealed. (*Dr. D. Erskine, London.*)

Treatment.—A dorsal slit of the prepuce is often essential to promote drainage, but it should be delayed twelve hours while a broad-spectrum antibiotic is given. Local cleaning can then be effective. A formal circumcision may be necessary later.

HARD CHANCRE (*syn.* HUNTERIAN CHANCRE)

Hunterian chancre is described on p. 28, and soft chancre on p. 31.

PENILE HERPES

This is an uncommon infection which is due to a virus and occurs on the glans and the prepuce. Rarely is it seen in the vesicular phase because the vesicles rupture early. The resulting erosions are superficial, red, and frequently take the form of multiple circular lesions encroaching upon one another, which is characteristic. Unlike other conditions from which it must be differentiated, penile herpes is painful and tender. Should doubt exist as to the diagnosis, infection by the virus of herpes can be confirmed by inoculating embryo chicks with some of the exudate.

LYMPHOGRANULOMA INGUINALE (*syn.* LYMPHOGRANULOMA VENEREUM)

Although predominantly a tropical venereal disease, lymphogranuloma inguinale is not rare in temperate climates where there is a mixed population of white and negro races. The condition is encountered in England mainly in seafarers whose ships have come from afar. The infection is caused by a filtrable virus of the psittacosis-lymphogranuloma inguinale group. It is transmitted by sexual intercourse, but accidental infection has occurred (e.g. in surgeons operating on infected patients).

John Hunter, 1728–1793. Surgeon, St. George's Hospital, London. He bequeathed 13,682 specimens prepared and mounted by his own hands. Many were destroyed by enemy action in 1941, but the remainder now form part of the Hunterian Museum of the Royal College of Surgeons of England.

The Primary Lesion.—Because the symptoms are so slight, rarely does the patient present with the primary lesion, but a careful history unfolds the occurrence of a primary sore on the genitals in about 25 per cent. of cases. The sore takes the form of fleeting, painless, herpetic vesicles. Exceptionally it is within the anterior urethra, when it constitutes one of the many causes of non-gonococcal urethritis.

The Secondary Lesion.—Almost invariably the patient presents only when the secondary lesion has appeared, i.e. two to six weeks after exposure to infection. Constitutional symptoms are generally absent, although in white people pyrexia and anaemia are sometimes in evidence. The secondary local lesion is as follows :

In the Male.—A lymph node in one groin or both groins enlarges, usually in the medial part of the groin. The infection spreads to other nodes and palpable enlargement of the iliac lymph nodes is also found in some cases. Soon periadenitis occurs, and a brawny mass presents, increases in size, and the overlying skin (in white races) becomes purple. Untreated, the mass proceeds to liquefy. Unless treated by aspiration, the mass breaks down and discharges thick yellowish-white pus free from organisms. The resulting sinus (fig. 1593) or sinuses persist for months or years. An occasional aftermath is lymphatic obstruction, resulting in elephantiasis of the scrotum and penis. With this exception, complications are rare in the male, and consist of transient joint effusions. Should the rectum be affected (cf. the female), homosexuality is implied.

In the Female.—If the primary lesion lies in the anterior part of the vulva, an inguinal bubo follows, as in the male. When, as is much more usual, the primary lesion is on the vaginal wall or cervix, oedematous thickening of the posterior vaginal wall occurs and it is not long before the para-rectal lymph nodes enlarge and suppurate. As a result of this intense para-rectal inflammation, dense fibrosis of the rectal wall follows with the formation of a stricture of the rectum (p. 1010). Elephantiasis of the vulva sometimes develops in chronic cases (Esthiomene). Ischio-rectal abscess and recto-vaginal fistula are not infrequent complications, and perianal polypos can occur.

Confirmatory Tests:

1. **The Frei Test.**—The original Frei antigen consisted of bacteriologically sterile pus obtained from an unruptured bubo of a patient suffering from the disease. This is more reliable than the now frequently employed lygranum, which is an extract of the content of yolk sacs of embryo chicks infected with the virus. One or 2 minims (0.05 or 0.1 ml.) of either of these is injected intradermally. A positive reaction appears within forty-eight hours, and is a red papule of at least $\frac{1}{4}$ inch (6 mm.) in diameter. The control site should be unchanged.
2. **The complement fixation test**¹ is more sensitive than the foregoing, and has the merit of giving a positive result earlier in the course of the disease.
3. **Biopsy** is occasionally necessary to differentiate other diseases of lymph nodes.



FIG. 1593.—Lymphogranuloma inguinale. (Professor F. A. R. Stammers, Birmingham, England.)

¹ Unfortunately, these tests, unlike a Wassermann reaction in syphilis, cannot be used as a test for cure.

Wilhelm Siegmund Frei, 1885–1943. Dermatologist, State Hospital, Spandau, Berlin; later emigrated to New York.

Treatment.—The disease responds both to sulphonamides and to several antibiotics. Many patients derive more benefit when two different drugs are exhibited successively than from any single drug.

(a) A course of sulphadimidine, 1 G. four times a day for ten days, to be followed by

(b) A course of oxytetracycline, 250 mg. six-hourly for a similar period, in patients who respond poorly or incompletely to (a).

If resolution has not been effected by the end of the dual course, other antibiotics should be tried after a suitable interval.

Fomentation of the bubo should be eschewed, as it favours softening and increases the necessity for aspiration (*not incision*), which should be carried out before the skin becomes involved. If general treatment is commenced reasonably early, aspiration will be required in only about 10 per cent. of cases.

Other complications such as stricture of the rectum must be treated on their merits (p. 1010).

GRANULOMA INGUINALE (*syn.* GRANULOMA VENEREUM)

This disease is a different entity from *lymphogranuloma* inguinale. It affects the genital, inguinal, crural, perineal, and perianal regions but not the lymph nodes, and is due to *Donovania granulomatis*, a Donovan body that can be seen as a Gram-negative rod found characteristically in the cytoplasm of large mononuclear tissue cells. It occurs in people living in or coming from sub-tropical or tropical areas.

Clinical Features.—The primary lesion is a vesicle surrounded by an area of erythema and induration, and looks a bright, beefy red. The overlying epithelium soon disintegrates and the lesion develops, either as a slightly raised ulcer or a mass of exuberant granulation tissue. Pain and tenderness are singularly absent unless secondary infection occurs. The lesion progresses slowly by peripheral extension, and also by satellites due to auto-inoculation.

Extragenital granuloma inguinale (by which is meant that the lesion is distant from all the commonest sites set out above) occurs in about 6 per cent. of cases, but usually these lesions are due to auto-inoculation. Disseminated disease from a genital primary lesion is particularly liable to occur in pregnant women, who thereby become extremely emaciated, with sores in various parts of the body. In general it can be stated that granuloma venereum of the cervix uteri is a most serious lesion, and not a few women die from the effects of involvement of the pelvic viscera. In none of the manifestations of the disease does complete recovery occur without thorough treatment, but sometimes there is partial healing with the formation of thick or keloid scar tissue. Other complications are rectovaginal fistula, pyelitis, cystitis, and urethral stricture. Scarring may cause lymphatic obstruction and oedema of the genitalia.

Confirmatory Test.—If a needle is drawn through the tissues near the edge of the lesion, sufficient material is obtained for microscopical examination after staining.

Treatment.—The treatment of choice is oxytetracycline (Terramycin) 2 G. daily for twenty days. In resistant cases, after an interval, a further course is sometimes required.

CHORDEE¹

Chordee is a fixed bending or bowing of the penis due to (a) hypospadias or (b) acutely as a result of general urethritis. Erection is grotesque and very painful. The treatment is stilbœstrol 6 mg. daily.

INDURATIO-PENIS-PLASTICA (syn. PEYRONIE'S DISEASE)

Usually the patient is over forty years of age and seldom is there a history of venereal disease. At the onset pain and curving of the penis on erection cause the patient to seek advice, but after a few weeks the pain disappears. On palpation an indurated plaque can be felt on the dorsal surface of one corpus cavernosum. The condition is slowly progressive and sometimes extends across the middle line. Its ætiology is unknown; bygone trauma is a possible explanation.

Treatment has remained an unsolved problem for over 200 years. In some cases there is a tendency to slow resolution over three to five years.

PERSISTENT PRIAPISM

The penis remains erect, and is painful. Most often the erection is due to idiopathic thrombosis occurring in the prostatic venous plexus. Less frequently it is associated with leukæmia or sickle-celled anæmia. Secondary malignant deposits in the corpora cavernosa, or in the pelvis, account for about 7 per cent. of cases. In another completely different category are cases due to spinal injury or disease.

Diagnosis.—A low spinal anæsthetic will cause priapism of neurogenic origin to abate temporarily. A blood-count is essential to exclude leukæmia.

Treatment.—Local applications are useless. Anticoagulant therapy together with adequate sedation for fourteen days bring about restitution in the majority of cases. Should this treatment not bid fair after a trial of several days, the repeated aspiration of the corpora cavernosa is of value. When this fails, or is unsatisfactory, a small incision into a corpus cavernosum should be made, and blood and blood-clot evacuated. Should gangrene occur amputation is necessary.

NEOPLASMS OF THE PENIS

Papillomas Acuminata² (syn. venereal warts), the commonest benign growths of the penis, occur both in the uncircumcised and the circumcised. Most often they are situated in the coronal sulcus (fig. 1594), but in the male they can occur anywhere on the penis, scrotum, perineum, and the anal region. Usually they are moist, and attended by an offensive serous discharge. Often they are transmitted by sexual contact and are possibly due to a virus infection.

Treatment:

(a) **Fulguration** of the growths by diathermy.

(b) **By Podophyllin.**—The parts are cleansed thoroughly and then unaffected skin should be protected by smearing with a zinc oxide ointment. A 10 or 20 per cent. solution of podophyllin in tinct. benzoin is applied with a sterile applicator, and the prepuce is held back for three to five minutes until the tincture dries. The patient is instructed to wash the part thoroughly in thirty minutes. Thereafter he



FIG. 1594.—Penile warts. (Dr. D. Erskine, London.)

¹ *Cordee* (Fr.) = corded.

² *Acuminatus* (Lat.) = sharp pointed.

must pay particular attention to cleanliness. The papillomas gradually disappear. Often such treatment is followed by considerable local reaction, and is painful. For this reason fulguration is advisable, especially if it is necessary to circumcise the patient.

CARCINOMA OF THE PENIS

Ætiology.—For a reason that is only partially understood, circumcision correctly performed soon after birth confers almost total immunity against carcinoma of the penis. On the other hand (and this is difficult of explanation) circumcision after early infancy does not provide the same degree of protection. Mohammedans, who are circumcised between the ages of four and nine years, exemplify this.

For practical purposes, then, carcinoma of the penis occurs only in men who have not been circumcised in early infancy, and undoubtedly the development of this neoplasm is favoured by chronic balanoposthitis. Furthermore, there are definite pre-carcinomatous states, viz. :

(a) **Leukoplakia of the glans** is exactly comparable to the well-known condition of the tongue (p. 496).

(b) **Long-standing penile papillomas** (p. 1209).

(c) **Paget's Disease of the Penis** (*syn. Erythroplasia of Quérat*).—"I have seen a persistent rawness of the glans like a long-standing balanitis followed by cancer of the substance of the penis" (Sir James Paget). It is possible that penile Paget's disease escapes detection until carcinoma has developed. Treatment is by radium or diathermy excision.

Pathology.—There are two types of squamous carcinoma of the penis—the flat or infiltrating, and the papilliferous. The former may be associated with leukoplakia; the latter commences in a papilloma of long standing. The growth remains purely local for months. The earliest spread is to the inguinal, and then to the iliac lymph nodes. Direct spread to the body of the penis is prevented for many months by the fascial sheath of the corpora cavernosa, but once this barrier becomes broken the growth extends more rapidly and the iliac lymph nodes (fig. 1595) become involved. Distant metastatic deposits are infrequent.

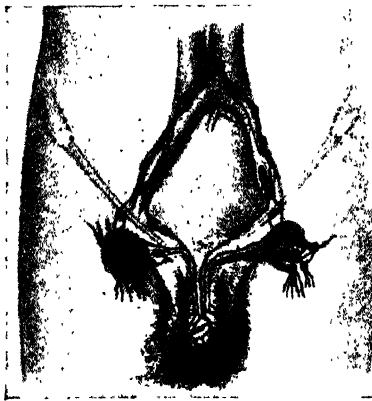


FIG. 1595.—The lymphatic drainage of the penis. The superficial lymphatic vessels drain into the inguinal while the deep lymphatics drain into the iliac lymph nodes. (After Archie L. Dean.)

Clinical Features.—It is a mistake to believe that this is a disease confined to the elderly—40 per cent. of the sufferers are under forty years of age. The progress of the disease is slow; the first symptoms are a mild irritation and a purulent discharge from the prepuce. These symptoms are often neglected (fig. 1596), and by the time the patient reports, sometimes more than a year after symptoms have appeared, there is a blood-stained, foul discharge. Pain is singularly absent. The inguinal lymph nodes are enlarged in over 60 per cent. of

cases, but in only half of these is the enlargement due to secondary deposits, the remainder being due to sepsis. In most cases the prepuce cannot be retracted, and in order to view the lesion the prepuce must be slit. In all cases a biopsy should be performed.

Untreated, the whole glans becomes a fungating and particularly foul-smelling mass. Later, the inguinal lymph nodes fungate through the skin of the groin, and often death relieves the victim by torrential hæmorrhage following erosion of the femoral or external iliac artery.

Treatment:

Radiotherapy gives good results (60 to 70 per cent. five-year survival) with small well-differentiated tumours. **Surgery** is needed for large anaplastic growths; if there is infiltration of the shaft of the penis; when radiotherapy has failed; in elderly men who do not mind the mutilation as much as the pain to be expected from the extensive reaction to radiotherapy. *Partial amputation* is used for distal growths providing there is at least 2 cm. of the dependant shaft which is not involved. With an advanced, infiltrating, or anaplastic lesion *total amputation* must be performed.



FIG. 1596.—Carcinoma of the penis.

Radiotherapy methods are: implantation with flexible radio-active tantalum wires (total dose 6,000 r. in five to seven days); medium or high-voltage X-rays (5,000 to 6,000 r. given in divided doses over five weeks); by radium mould applicators worn intermittently or continuously (not exceeding 6,000 r. in seven to ten days). If not already performed, a dorsal slit is a prerequisite for treatment and nursing.



FIG. 1597.—Partial amputation of the penis.

Partial Amputation Technique (guillotine amputation).—A tourniquet, in the form of a No. 6 French rubber catheter, is placed around the most proximal portion of the penis, being tied once, and the knot clipped in a hæmostat. An abdominal pack is placed over the glans penis and with a piece of strong silk is tied tightly to the shaft of the penis at the proposed level of section. A guillotine amputation is then performed, and the pack, with its contents, is dropped into a receptacle. The exposed cross-section of the penile stump is examined for extensions of the growth. If, as is usual, none is present, the dorsal vessels are ligated. The corpus spongiosum is dissected from the corpora cavernosa for 1 cm. A long straight needle carrying a double stout

catgut suture is passed from the ventral to the dorsal aspect of the fibrous septum separating the two corpora cavernosa, the needle entering just distal to the attached portion of the corpus spongiosum. The needle having been removed (fig. 1597), each corpus cavernosum is ligated firmly. The free portion of the urethra and its surrounding corpus spongiosum is divided on the dorsal aspect for nearly 1 cm. The tourniquet is now removed. The urethra is carefully attached to the adjacent skin by interrupted stitches that pass through the whole thickness of the corpus spongiosum, thereby avoiding a post-operative stricture. Bleeding-points having been ligated, the remaining portions of the skin edges are approximated vertically over the cut corpora cavernosa. An indwelling catheter can be employed until the dressings are dispensed with.

Complete amputation includes the removal of the corpora cavernosa from the pubic bones and division of the corpus spongiosum so as to leave at least $\frac{1}{2}$ inch (1.3 cm.)

protruding from the perineum, as a perineal urethrostomy via an incision behind the scrotum. Careful suture of the divided urethra to the skin avoids a subsequent stricture.

The Treatment of Associated Enlarged Inguinal Lymph Nodes.—

It is advisable to wait for at least three weeks after the local lesion has been dealt with by one of the methods described. If the enlargement is due to inflammation, the lymph nodes will decrease in size or disappear with antibiotic treatment. When these lymph nodes remain unaltered at the end of this period, block dissection of the more affected side should be undertaken, followed by contralateral block dissection at a later date. The five-year survival rate falls to some 35 per cent. in these cases.

If the enlarged lymph nodes are massive and fixed (inoperable), X-ray therapy causes some temporary regression.

Secondary carcinoma of the penis occurs from time to time, and about a hundred cases have been reported. The primary source of the disease is usually the bladder, rectum, or prostate.

The metastasis arrives in the penis in one of three ways : (a) by direct spread ; (b) by retrograde lymphatic transport ; (c) by retrograde venous embolism via the dorsal vein of the penis. The condition must be differentiated from Peyronie's disease. As would be expected, secondary carcinoma of the penis occurs only late in the course of the disease ; nevertheless, in many instances active treatment is worth while.

CHAPTER 49

THE TESTES AND THE SCROTUM

CONGENITAL ABNORMALITIES

Anterior inversion is said to be present in one in every fifty males. The epididymis lies anteriorly, and the body of the testis and the tunica vaginalis posteriorly. When the organ is diseased this anomaly may cause confusion in diagnosis (p. 1229).

Polar inversion is less frequent. In some cases when there is complete inversion the globus major lies inferiorly (fig. 1598(B)). In other cases it lies horizontally (fig. 1598(C)). Both varieties predispose to torsion (p. 1218). Complete inversion, impossible to detect clinically, is an incidental finding at operation, when it is observed that the hydatid of Morgagni is situated at the lower, instead of the upper, pole.

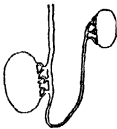


FIG. 1599.—Polyorchism; usual arrangement. (After P. St. G. Anderson.)

Supernumerary testis (*syn.* polyorchism) is exceedingly rare. The accessory testis is small, usually on the left side communicating with a common epididymis (fig. 1599). Only at operation can the diagnosis be made with certainty.

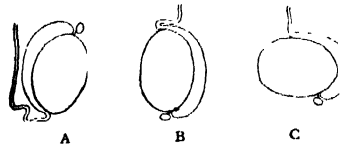


FIG. 1598.—A. Normal. B. Inverted. C. Horizontal inversion.

IMPERFECT DESCENT OF THE TESTIS

Under this heading are included two conditions :

Incomplete Descent.—The testis is arrested in some part of its path to the scrotum.

Maldescended (*syn.* *Ectopic*) **Testis.**—The testis is abnormally placed outside this path.

Development of the Testes.—The testes develop from the genital fold, which lies medial to the mesonephros (Wolfian body), and therefore in early foetal life they lie in the coelomic cavity behind the peritoneum and below the developing kidneys. The primitive testis is attached to the posterior abdominal wall by a mesentery, the mesorchium, which contains the testicular blood-vessels and nerves, derived from the tenth and twelfth dorsal segments respectively. About the tenth week of intra-uterine life some of the transverse tubules of the mesonephros unite with the rete testis to form the vasa efferentia; others remain rudimentary. Most of the Wolfian body disappears, but the Wolfian duct becomes the epididymis and vas deferens. About this time tissue in a fold of peritoneum (*plica inguinalis*) develops, containing the precursor of the gubernaculum¹; it is attached to the junction of the vas deferens and epididymis, and can be traced through the abdominal wall to end in the region of the developing phallus. The processus vaginalis appears as a dimple of peritoneum about the tenth week and precedes the testis through the layers of the abdominal wall down into the scrotum. Muscular fibres develop in the mesoderm of

¹ Gubernaculum = a rudder. It was first described by John Hunter, who believed that its function was to guide the testis into the scrotum.

Giovanni Battista Morgagni, 1682–1771. Professor of Medicine and Anatomy, Padua. He held the chair for fifty-six years, and was the founder of Pathological Anatomy.
Kaspar Friedrich Wolff, 1733–1794. Professor of Anatomy and Physiology, St. Petersburg (now Leningrad).
John Hunter, 1728–1793. Surgeon St. George's Hospital, London. Founder of the Hunterian Museum.

the plica inguinalis to form the gubernaculum but there is still no certainty as to the part this structure plays in regulating testicular descent.

Chorionic gonadotrophin from the maternal circulation stimulates the growth of the testis and may play some part in the migration of the organ. Undoubtedly imperfect development of a testis is a significant factor in imperfect descent of the organ.

Morphology.—Most of the misplacements of the testes in man are a counterpart of the varying normal placements of the testes in animals. In the whale and the elephant the testes remain in the undescended abdominal position throughout life. Rodents and hibernating animals, such as the hedgehog, the mole and the bat, maintain open inguinal canals, and the testes are housed in the abdomen, to descend into the scrotum only during the breeding season. Man is the only member of the animal kingdom that has testes in the scrotum at birth.

INCOMPLETELY DESCENDED TESTIS

Incidence:

In the Neonatal Period.—At birth, and for a variable number of weeks afterwards, the cremasteric reflex, which is so active in young boys, is absent. Scorer examined 2,000 newborn male infants and found that the incidence of imperfect descent on one or both sides was 4 per cent. in full-term infants and 30 per cent. in premature infants. A follow-up of all cases showed that in more than 50 per cent. the testis or testes reached the scrotum during the first month of life. More often than not imperfect descent of the testis is not detected during infancy.

In later childhood and puberty the incidence is 2 per cent. Frequently the condition still remains unrecognised unless a routine examination is made by the school medical officer. In a few cases the presence of a hernia, pain in the region, or acute torsion, in that order of frequency, directs attention to the abnormality.

In Adult Life.—It is inconceivable that a man can fail to notice the absence of one or both testes in his scrotum, yet there must be many with this abnormality who do not seek advice about it unless symptoms develop. Sometimes the condition is first discovered at the medical examination for entrance to one of the Public Services. In an examination of 10,000 recruits during World War II the incidence was found to be 0.8 per cent. In 10 per cent. of unilateral cases there is a familial history.

Pathology.—Up to the age of six years there are no macroscopical differences between an incompletely descended testis and a normal testis. After that time, due, it is believed, to the higher temperature to which it is subjected, the development of the incompletely descended organ becomes progressively retarded.¹ By the time puberty has been reached the incompletely descended testis is flabby and hardly more than half the size of its intrascrotal counterpart. Histologically, the epithelial elements are grossly immature, and by the age of sixteen irreversible destructive changes have occurred in the germinal epithelium.

The *external secretory mechanism* of an incompletely descended testis functions but feebly, and often after a few months or years ceases; thus its power of spermatogenesis is negligible².

The *internal secretory activity* of an incompletely descended testis is reduced. In bilateral cryptorchism about half the normal amount of androgen is produced: notwithstanding, the secondary sexual attributes of a cryptorchid are seldom noticeably in abeyance.

If an incompletely descended testis is brought down satisfactorily *before* puberty it often develops and functions satisfactorily.

Clinical Features.—The right testis alone is affected in 50 per cent. of cases, the left alone in 30 per cent., while double arrested descent occurs in 20 per cent. Other abnormalities of the urinary tract may be present. Felton found that intravenous pyelograms demonstrated major abnormalities in 13·5 per cent. of children with cryptorchism. The testis may be:

1. *Retained within the abdomen* extraperitoneally, usually just above the internal abdominal ring.

2. *In the Inguinal Canal.*—Early in life the testis is a soft structure, and when, as in this instance, it is submerged in the non-resisting floor of the inguinal canal, and shielded by the overlying tendinous aponeurosis of the external oblique, it cannot be felt.

When both testes are in the abdomen or the inguinal canals and are consequently impalpable, the condition is known as cryptorchidism (hidden testes) (fig. 1600).

3. *In the Superficial Subinguinal Pouch.*—Very frequently during childhood the testes are mobile, each being withdrawn by the contraction of the cremasters into the superficial subinguinal pouch (fig. 1601), a space lined by loose areolar tissue lying beneath Scarpa's fascia and superficial to the external oblique aponeurosis (Denis Browne), or into the inguinal canal. Reflex retraction occurs from a very

slight stimulus such as touching the thigh or the abdomen, or even by exposure of the parts. Not infrequently this testicular

mobility continues throughout childhood on both sides, or less frequently on one side. At all times it is liable to be mistaken for arrested descent unless a special method of examination

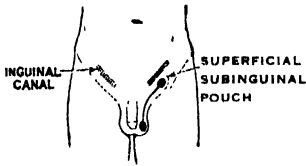


FIG. 1601.—Mechanism of retractile testis. (After Sir Denis Browne.)



FIG. 1600.—A cryptorchid, aged 12. Note the retracted, underdeveloped scrotum. In cases of retractile testes the scrotum is comparatively well-developed.

is undertaken, if necessary on more than one occasion. Retractable testes should be suspected if the scrotum is normal; in arrested descent the corresponding side of the scrotum is undeveloped. This inspection completed, the pulps of two fingers are placed over the superficial inguinal pouch, exerting moderate pressure. By drawing the fingers towards the neck of the scrotum a testis of the retractile type can be pushed into the upper part of the scrotum, where it is grasped between the finger and thumb of the other hand. *Only if the testis cannot be made to touch the bottom of the scrotum¹ is it imperfectly descended.* When the testis can be placed in a normal position temporarily it is not truly an imperfectly descended testis, and it should be

known by its fitting title '*retractile testis*'. Such testes require neither endocrine nor operative treatment. In course of time (it may be delayed until puberty), the testis will take up a normal position permanently.

This is borne out by the results of an extensive survey into the problem of undescended testes in school children. It was found that between five years and eight years about 4.4 per cent. had one testis undescended, while on leaving school this figure had dropped to 0.58 per cent. The investigators concluded that, between five years and eight years, group assessment of descent of the testes is difficult owing to the instability of position of the organ. Similarly, at five years 2.03 per cent. had bilateral non-descent of testes and on leaving school this was present in 0.07 per cent.

In early life 80 per cent. of non-apparent testes are retractile. In the remaining 20 per cent. true incomplete descent is present.

The Hazards of Incomplete Descent

1. *Sterility* in bilateral cases.
2. *Pain*. An inguinal testis is liable to oft-repeated trauma.
3. *An associated indirect inguinal or interstitial hernia* is present in 70 per cent. of cases, and in adolescent and adult patients it is frequently the hernia that causes the symptoms.
4. *Torsion* (p. 1218).
5. *Epididymo-orchitis*.—Right-sided epididymo-orchitis occurring in an incompletely descended organ is extremely difficult, if not impossible, to differentiate from acute appendicitis.
6. *Atrophy*, even before puberty, sometimes occurs when the testis is situated in the inguinal canal. This is attributed to recurrent trauma.
7. *Increased Liability to Malignant Disease*.—All types of malignant testicular tumour have been recorded as developing in undescended testes, and in those brought down surgically. It is definite that seminoma is more common possibly because the testis is frequently histologically abnormal. Some compute that 1 in 20 abdominal and 1 in 80 inguinal testes become malignant; others say this estimation is far too high. Reliable statistical evidence as to whether orchiopexy diminishes the liability to tumour formation later, is not available. However, at the worst it should improve the prospect of early diagnosis.

Treatment by Orchiopexy.—Most surgeons favour performing the operation between the ages of six and eight years. Many choose the earlier age in order to reduce the incidence of thermal damage to the germinal epithelium. After the age of puberty the percentage of successful operations, which at the optimum age approaches 80 per cent., falls considerably. In cryptorchidism one side should be operated upon at a time, with an interval of six months between the operations.

The operation consists of (1) mobilising the spermatic cord and the testicular vessels; (2) retaining the mobilised testis in the descended position.

1. *Mobilising the Spermatic Cord*.—The inguinal canal is opened. The cord and testis are freed from surrounding structures and a concomitant inguinal hernial sac is dealt with. The dissection of the diaphanous sac from the cord is greatly facilitated by opening the sac and injecting a small amount of normal saline just

underneath that part which is adherent. Near the internal inguinal ring all the coverings of the cord are divided; every muscle fibre and fibrous band is severed, leaving only the vas and its blood-vessels and the spermatic blood-vessels. Sometimes this provides sufficient length for the testis to be placed in the scrotum. If not, the spermatic vessels are dissected from the peritoneum to which they are adherent. This abolishes the wide, outward curve of these vessels, thereby gaining up to 1 inch (2.5 cm.). It is most exceptional for the above measures to fail to elongate the pedicle sufficiently for the testis to be placed in the scrotum, and even beyond it: the avoidance of vascular tension is the keystone of plastic surgery. The empty half of the scrotum is stretched thoroughly with the index finger passed into it through the inguinal incision.

2. *Retaining the testis in the scrotum* is effected by one of a number of ingenious methods, of which the following are popular:

(a) *Narrowing the neck of the scrotum and fixation of the testis to the bottom of the scrotum*, is all that is needed in many cases. The neck of the scrotum is narrowed with catgut sutures, and the testis is anchored below by an unabsorbable suture passed through the scrotal skin. This suture can then be attached, if need be, to an elastic band, the lower end of which is fixed to the skin of the lower thigh for up to seven days. The anchoring suture is removed about twelve days after the operation.



FIG. 1602.—Method of passing the suture attached to the testis through the septum dartos and the skin of the opposite side of the scrotum.

(b) *Ombreëdanne's operation* is suitable for patients under the age of ten years. A suture is attached to the lower end of the tunica vaginalis of the mobilised testis, and the two ends are left long. A short vertical incision about 1 inch (2.5 cm.) in length is made in the opposite side of the scrotum, $\frac{1}{2}$ inch lateral to the median raphe, and deepened until the corresponding tunica vaginalis is seen. Between this and the septum dartos, a bed is prepared by blunt dissection. The free ends of the suture are seized in a hæmostat, which is passed through the inguinal incision into the scrotum. The tip of the hæmostat is thrust against the septum dartos, permitting a small incision to be made through the septum exposing the tip of the hæmostat and the suture it grasps (fig. 1602). The jaws are loosened and the ends of the suture are drawn through the scrotal wound. Before removing the hæmostat its jaws are opened sufficiently to stretch the tiny opening in the septum to about half the size of the testis, which is

pulled through the opening into the opposite side of the scrotum by traction on the suture. The hole in the septum is narrowed around the cord just sufficiently to prevent the testis escaping from its new abode, but not tightly enough to compress the cord (fig. 1603).

(c) *The Keetley-Torek Operation*.—The testis is brought down through the scrotum and stitched to the deep fascia of the thigh. A skin tunnel is fashioned for this purpose, fig. 1604. After six months or less the testis and the scrotum are freed from the thigh. The special indication for this operation is when the patient is a youth or an adult, but some specialists in child surgery employ the Keetley-Torek operation in those of tender years.

Failure to bring the testis down.—(1) Sometimes a two-stage procedure is successful: mobilisation as far as possible, and anchorage with an unabsorbable suture, followed six months later by further mobilisation. (2) *Orchiectomy*, if the

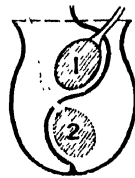


FIG. 1603.—The disposition of the testes in relation to the septum dartos following bilateral Ombreëdanne's operation.



FIG. 1604.—The Keetley-Torek operation.

other testis is normal, and the incompletely descended testis is hopelessly atrophic, or, after puberty.

Orchio-coelioplasty (abdominal replacement of the organ). This measure is justifiable when the contralateral organ has been removed previously and orchiopexy is found to be impossible. It is then the only method of preserving a supply of internal testicular secretion and secluding the organ from injury.

Hormone Treatment.—The only indications for hormone treatment are:

1. Cases of bilateral incomplete descent associated with hypogenitalism and obesity.
2. In lieu of waiting for retractile testes to descend, thereby avoiding jibes that small boys endure from others in the changing rooms of swimming baths, etc. (Scowen).
3. In cases of uncertainty as to whether the case is one of testes retracting into the inguinal canal or one of true incomplete descent.

Of many preparations having a gonadotrophic action, chorionic gonadotrophin extracted from the placenta (Pregnyl) is the one usually employed. It should be given at about eight years of age, and 1,000 international units are given twice a week until the testes descend, the limit of the course being ten weeks, after which it must never be repeated, otherwise secondary male characteristics may develop prematurely.

ECTOPIC TESTIS (*syn.* MALDESCENDED TESTIS)

An ectopic testis may be found, in order of frequency:

1. Anchored in the superficial inguinal pouch.
2. In the perineum.
3. At the root of the penis.
4. In the femoral triangle.

Unlike the imperfectly descended testis, an ectopic organ often develops well, if not fully. Its main hazard is that it is liable to injury.

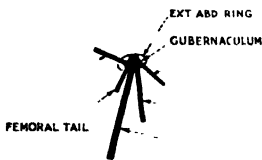


FIG. 1605.—The 'gubernacular tails' of Lockwood.

Ætiology.—To explain the appearance of the testis in these anomalous positions, Lockwood advanced the ingenious theory of many gubernacular tails. His theory postulates that in ectopic testis the scrotal tail becomes ruptured. As a consequence the testis, adrift from its usual mooring, follows one of the accessory rudders. Lockwood stated that the accessory gubernacula depicted in fig. 1605 could be demonstrated in the fœtus.

INJURIES OF THE TESTIS

Closed rupture of the testis from blows and the like is an uncommon accident owing to the extreme mobility of the organ within the scrotum. Severe contusion and rupture are each associated with a hæmatocele which surrounds the testis, rendering the differential diagnosis impossible without exploration. Operation is advisable in these cases in order to:

1. Evacuate the hæmatoma.
2. Repair the tunica albuginea, if split, excising only that part of the testicular tissue that extrudes. If the testis is severely damaged, orchiectomy should be performed.

Hæmatocele (p. 1226).

Traumatic Dislocation of the Testis.—As a result of a blow the testis occasionally becomes dislocated and by reason of blood-clot anchored, usually in one of the positions described under ectopic testis. Unless the dislocation is reduced early by manipulation, an operation is required to replace the organ in the scrotum.

TORSION OF THE TESTIS (*syn.* TORSION OF THE SPERMATIC CORD)

Predisposing Causes.—Torsion of the testis (fig. 1606) is uncommon. It does not occur in a normal fully descended testis: its anchorage prevents rotation. Therefore one of several anomalies must be present.

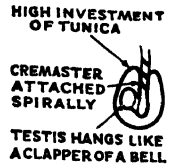
(a) *Inversion of the Testis* (p. 1213).—This appears to be the commonest predisposing cause.



FIG. 1606.—Torsion of the testis. Note the inversion of the organ.

(b) *High investment of the tunica vaginalis* causes the testis to hang within the tunica like a clapper in a bell, viz. —————→

Very occasionally torsion is extravaginal.



(c) *In cases where the body of the testis is separated from the epididymis*, torsion of the body can occur without involving the cord. The twisting is confined to the mesentery that joins the testis to the epididymis, viz. —————→



Exciting Causes.—Normally the cremaster contracts *pari passu* with violent contraction of the abdominal musculature. Contractions of the spirally attached cremaster favour rotation around the vertical axis in relevant cases. Straining at stool, lifting a heavy weight, and coitus are all exciting causes, but often the history fails to reveal any one of them. Occasionally torsion develops during sleep.

Clinical Features.—The highest incidence is between fifteen and twenty-five years of age, and the second most common age period is during infancy. The symptoms vary with the degree of torsion present. Most commonly the patient experiences sudden and agonising pain in the groin and lower abdomen,¹ and vomits. Upon theoretical grounds one might think that the diagnosis is simple, but sometimes it is practically impossible to distinguish *torsion of an imperfectly descended testis* from a strangulated inguinal hernia until the parts have been displayed by operation. The fact that the side of the scrotum is empty and œdematous is certainly in favour of the tender lump at the external abdominal ring being the testis with its twisted cord. *Torsion of a completely descended testis* is a less difficult problem. Sometimes the actual twists in the cord can be felt, thus establishing the diagnosis. At other times the condition can be mimicked exactly by a small, tense, strangulated inguinal hernia compressing the cord and causing congestion of the pampiniform plexus, viz. —————→



Torsion of the fully descended testis can also simulate closely acute epididymo-orchitis; after a lapse of six hours or so the skin of the scrotum becomes reddened (fig. 1607) and the temperature is raised slightly—99° F. (37·2° C.). Elevation of the scrotum usually relieves the pain in epididymitis, but increases it in torsion of the spermatic cord. Therefore in a boy (if mumps has been excluded) and in a man (if urethritis has been eliminated), a diagnosis of torsion of the testis should be insisted upon,

¹ But, in about 23 per cent. of K. H. Smith's patients, the first symptom was a dull ache in the loin or hypogastrium. It is insufficiently appreciated that true testicular pain is situated in the lower abdomen at the level of the internal inguinal ring in accordance with Brown's law.²

² Brown's law: Pain, produced in an organ which has migrated from its primary position, and which has not acquired an additional nerve supply in its secondary or permanent position, is invariably localised in the primary relative position of that organ.



FIG. 1607.—Torsion of the right testis of twenty-four hours' duration in a boy aged thirteen. The acutely tender testis and the inflamed scrotum simulate acute epididymo-orchitis.

and almost invariably when the testis is exposed the diagnosis will be substantiated.

Treatment.—Urgent operation is indicated, as after six hours of strangulation the chance of the testis recovering completely is small (O'Flynn). In a few early cases it may be possible to untwist the cord under vision (fig. 1608), in which case, should an improvement in the colour of the organ result,

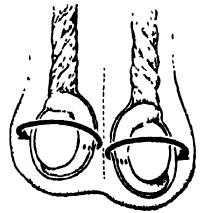


FIG. 1608.—Usually the testis *twists* in a direction away from the septum dartos, so *untwisting* must be effected in the opposite direction.

and the testis is fully descended, it can be spared, but it is essential to fix the testis in an anatomically correct position. More usually orchiectomy must be performed. Experience has shown that in cases where torsion occurs, the congenital abnormality that permits twisting (p. 1213) appertains to both sides. Therefore, the contralateral organ should be fixed in the scrotum (fig. 1609) even at the same operation. By failing to observe this precaution patients may lose both testes from torsion.

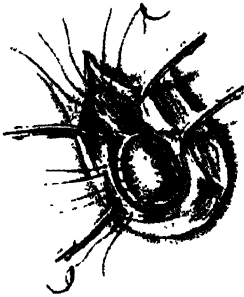


FIG. 1609.—Method of anchoring the testis with non-absorbable sutures to prevent torsion.

or at the age of puberty. The condition is often mistaken for acute epididymo-orchitis of unknown origin and frequently, although the symptoms are comparatively mild, they cannot be distinguished with certainty from those of torsion of the testis. Immediate operation with ligation of the pedicle and amputation of the twisted appendage cures the condition.

Torsion of an Appendage of the Testis.—Vestigial structures related to the testis and epididymis are liable to undergo axial rotation. The most common of these structures to twist is the appendix of the epididymis (the pedunculated hydatid) (fig. 1610). Torsion of an appendage of the testis is essentially a lesion occurring before

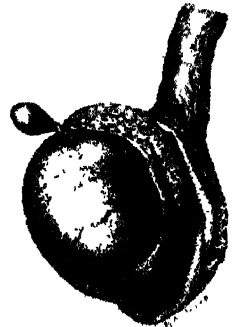


FIG. 1610.—Torsion of the appendix of the epididymis (pedunculated hydatid of Morgagni).

VARICOCELE

Varicocele is a state of varicosity of the cremasteric and occasionally the testicular veins.

Surgical Anatomy.—The veins of the testis and epididymis form an anastomosing plexus—the pampiniform plexus—which is the most bulky constituent of the spermatic cord. As the veins pass upward through the inguinal canal they become reduced in number to between four and eight. Coalescence in the neighbourhood of the internal inguinal ring brings about further reduction in their number, and by the time the posterior abdominal wall is reached, a single, or a duplicated testicular vein pursues its cephalad course behind the peritoneum, to empty into the renal vein on the left, and the inferior vena cava on the right. Only near their terminations are the testicular veins provided with valves, and not infrequently even these valves are absent, in which case valves will be found nearby in the renal veins. The cremasteric veins

anastomose with the testicular veins, so providing an alternative (collateral) venous return and draining mainly into the inferior epigastric veins.

Ætiology.—(1) Most varicoceles are noted in the 'teens' or early adult life. The left side is affected in 95 per cent. of cases. The left testicular vein joins the renal vein and reflux into this vein can be demonstrated by angiograms. Nathan has shown that the left testicular artery arches over the left renal vein in 16 per cent. of cases and may obstruct it (fig. 1612).

(2) Harrison and Hanley, however, maintain that in many cases the dilated vessels are cremasteric veins, and not veins of the pampiniform plexus. These veins drain into the inferior epigastric veins mainly, but also communicate freely with the veins in the pampiniform plexus.

(3) Obstruction of the left testicular vein by growth of a hypernephroma along the left renal vein is a rare cause (0.4 per cent. Riches) of a varicocele arising in middle life.

Clinical Features.—Varicocele is more frequent and more troublesome in hot climates; in all parts of the world tall, thin, visceroptotic men are frequently affected, whereas short, fat individuals are seldom so. Usually symptoms are entirely absent, but in some cases the patient experiences a dragging pain in the

affected side. Pain is due to the fact that the elongated scrotum no longer supports the testis, whose full weight is now borne by the cord. The scrotum, particularly the left side, hangs lower than normal (fig. 1613), and on palpation with the patient standing, the varicose plexus feels like a bag of worms. If, while lightly holding the varicocele between the fingers and thumb, the patient is instructed to bow (fig. 1614), tension within the veins becomes appreciably less. An impulse is often felt when the patient coughs. When the patient lies down and the scrotum is elevated, the veins will be emptied by gravity and the opportunity of comparing the size of the left testis with its fellow should be taken. In long-standing cases the left testis is somewhat smaller and softer than the right, due to a minor degree of atrophy. Infertility can be caused by a varicocele.

Varicocele and Spermatogenesis.—The testis is supported in the scrotum

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 Howard Granville Hanley, *Contemporary*. Surgeon, St. Peter's and St. Paul's Hospitals, London.
 Sir Eric Riches, *Contemporary*. Honorary Consulting Surgeon and Urologist, Middlesex Hospital, London.

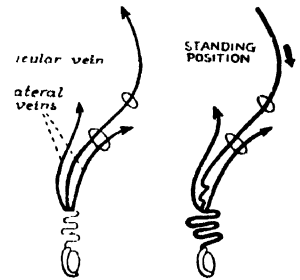


FIG. 1611.—(a) Normal drainage of the pampiniform plexus; (b) reflux along the testicular vein in varicocele. (After J. W. Lane.)

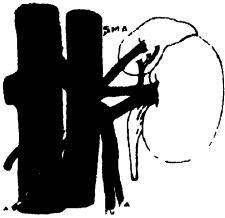


FIG. 1612.—Arching left testicular artery compressing the renal vein of the corresponding side. (After H. Nathan.)



FIG. 1613.—A large varicocele. A left inguinal hernia is also present.

by the cremaster and the dartos muscles. The main function of the cremaster is one of defence, for a fright or threat of trauma will cause reflex retraction of the testis towards the external inguinal ring, especially in the young. The dartos bears the weight of the testis and is reflexly controlled by temperature changes and acts as a kind of thermostat for efficient spermatogenesis at some 2.5° C. lower than rectal temperature. On exposure to cold it raises the testis to the body so conserving heat, while a too warm environment causes relaxation of the dartos.



FIG. 1614.—The bow sign. (After E. B. Ribeiro.)

Hanley and Harrison, who have reawakened interest in the effects and treatment of varicocele, explain the characteristic elongation of the scrotum in this condition on the grounds that venous engorgement in the

varicosities reduces the temperature differential (scrotum/rectum) to about 0.1° C. which may seriously depress spermatogenesis. The dartos relaxes in a vain attempt to maintain the temperature differential. It also follows that the modern tendency to wear tight and poorly ventilated pants and scrotal supports is to be deprecated.

Treatment.—Operation for varicocele is not indicated unless there is (1) pain; (2) serious depression of spermatogenesis (oligospermia).

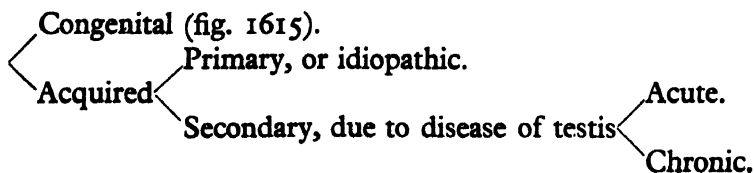
Technique.—The testis and varices are best exposed by a scrotal incision. The operating table is tilted downwards to distend the varicosities. Tortuous and varicose veins are divided and ligated individually. If there are any distended testicular veins of the pampiniform plexus, the enclosing glistening capsule is incised and each affected vein divided and ligated. Sufficient veins must be left to maintain venous return. Some maintain that these veins are more easily ligated within the inguinal canal.

Hæmatoma formation after operation on the scrotal contents is usually due to bleeding from the dartos muscle. This should be sutured meticulously with a continuous everting catgut stitch; the scrotum should not be drained. The skin edges will lie together and need not be stitched. The scrotum should be supported on a bridge of 'Elastoplast' (fig. 1626) across the thighs, and the patient should be kept in bed for forty-eight hours.

As a result of this operation the testis becomes cooler, and the dartos contracts, supports the testis, and relieves pain, while cases of oligospermia show a 70 to 80 per cent. improvement with a conception rate of about 30 per cent.

The older operations employing mass ligation, transfixion, and wide resection, are no longer advocated.

HYDROCELE



A hydrocele is a collection of serous fluid in some part of the processus vaginalis, usually in the tunica. Four anatomical varieties of congenital hydrocele are encountered.

Ætiology.—A hydrocele can be produced in four ways :

1. By excessive production of fluid within the sac, e.g. secondary hydrocele.
2. By defective absorption of hydrocele fluid by the tunica vaginalis. Defective absorption appears to be the most common cause of the common variety of primary hydrocele, but the reason for the defective absorption is obscure. Damage to the endothelial wall by low-grade infection seems to be the probable explanation.
3. By interference with the drainage of fluid by the lymphatic vessels of the cord.
4. By connection with the peritoneal cavity, as in the congenital variety.

Hydrocele fluid is amber coloured, and registers a specific gravity of 1.022 to 1.024. It contains water, inorganic salts, 6 per cent. of albumin, and a quantity of

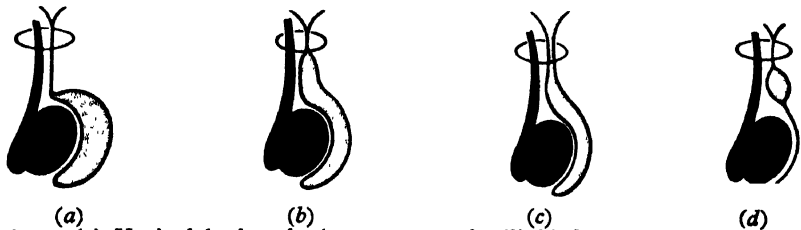


FIG. 1615.—(a) Vaginal hydrocele (very common); (b) 'infantile' hydrocele (unusual); (c) congenital hydrocele; (d) encysted hydrocele of the cord.

fibrinogen. The last constituent confers upon the fluid a characteristic feature. If the contents of a hydrocele are allowed to run through a cannula into a receptacle, the fluid does *not* clot, but if a few drops of blood which have come into contact with cut tissues are stirred into even a large quantity of hydrocele fluid, the whole clots firmly. In old-standing cases the fluid sometimes contains so much cholesterol that it is iridescent, and occasionally tyrosine crystals are found.

DIAGNOSTIC RULES FOR ALL HYDROCELES

Ninety-nine out of every 100 hydroceles are translucent. On examination it is possible 'to get above the swelling'.

(a) **Primary vaginal hydrocele** most often appears in middle-aged (fig. 1616) or elderly men, but it is not uncommon in early childhood (fig. 1617). The condition is particularly common in tropical countries. The only complaint of the patient is the swelling, and occasionally he does not seek relief until the sac has attained enormous dimensions.

About 5 per cent. of inguinal herniæ are associated with a vaginal hydrocele of the same side.¹ Often a large hydrocele obscures a small inguinal hernia, even if the latter has been especially looked for.

(b) **Infantile hydrocele** does not necessarily appear in infants. The tunica and the processus vaginalis are distended up to the internal abdominal ring, but the sac has no connection with the general peritoneal cavity.



FIG. 1616.—Large bilateral hydroceles of many years' duration.

¹ Captain Edward Gibbon (1737–1794) of the South Hampshire Militia and Member of Parliament was greatly embarrassed by a large hydrocele. He wrote 'The History of the Decline and Fall of the Roman Empire'. After the second tapping of his hydrocele he died of infection. The hydrocele seems to have been associated with a large scrotal hernia which, probably, was punctured.

(c) **Congenital Hydrocele.**—The processus vaginalis communicates with the peritoneal cavity. Usually the communicating orifice is too small for the development of a hernia. When the patient is horizontal, e.g. at night, the fluid in the pouch disappears within the abdominal cavity, but it returns when the erect posture is resumed. The hydrocele cannot be emptied by digital pressure, as this results in an 'inverted ink bottle' effect at the internal ring. Especially in bilateral cases, ascites or ascitic tuberculous peritonitis should be suspected.



FIG. 1617.—Right vaginal hydrocele in an infant. It is possible to get above the swelling which is brilliantly translucent.

(d) **Encysted Hydrocele of the Cord.**—

There is a smooth, oval swelling associated with the spermatic cord, which is liable to be mistaken for an irreducible inguinal hernia. If, with gentle traction upon the testis, the swelling moves downwards and becomes less mobile, the diagnosis of hydrocele of the cord is confirmed.

Hydrocele of the canal of Nuck is a condition comparable to the foregoing. It occurs in females, the cyst being in relationship to the round ligament. Unlike a hydrocele of the cord, a hydrocele of the canal of Nuck is always wholly, or partially, in the inguinal canal.

Hydrocele en Bisac.—A rare condition where the hydrocele has two intercommunicating sacs, one above and one below the neck of the scrotum. The upper lies superficial to or partly within the inguinal canal and it may insinuate itself in between the muscle layers.

COMPLICATIONS OF A HYDROCELE

1. *Rupture* is usually traumatic, but may possibly be spontaneous. On rare occasions a cure results after absorption of the fluid.
2. *Hernia of the hydrocele sac* sometimes occurs in old-standing cases. Tension of fluid within the tunica causes herniation through the dartos muscle (fig. 1618).
3. *Transformation into a hæmatocele* occurs both spontaneously and as the result of trauma (p. 1226).
4. *Calcification* of the sac wall sometimes occurs in long-standing cases.

Treatment.—*Tapping.*—After transillumination, the swelling is made tense by manual compression. A fine trocar and cannula is inserted into an unquestionably translucent area and the fluid evacuated. After a varying interval of weeks or months the sac usually refills. Many patients are content to be relieved at regular intervals in this way. Repeated tapping is liable to be followed by a little oozing into the sac. Deposits of blood on the walls of the sac in time increase its thickness, and so diminish its translucency. After tapping, the testis should always be examined.

Subtotal excision is the best means of ensuring against recurrence, and it is the only satisfactory method for thick-walled hydroceles. An incision is



FIG. 1618.—A hernia of a hydrocele.

made over most the prominent part of the swelling and the hydrocele together with the testis is separated from its coverings by gauze dissection. The sac is opened with a scalpel and is then split along its length. Each half of the tunica vaginalis is excised, preferably with a diathermy knife, a little at a time (fig. 1620) close to its



FIG. 1619.—Tapping a hydrocele.

reflection on to the epididymis. Each segment held in a hæmostat is likely to contain blood-vessels, so it is ligated by transfixion. Complete hæmorrhage is essential. The testis is replaced. The wound is closed without drainage as described on p. 1222.

Partial excision with eversion (syn. Jaboulay's operation) is suitable for thin-walled hydroceles. Sufficient of the sac is excised to allow the cut edges to be united without tension behind the epididymis, so that the endothelial lining faces outwards.

Secondary hydrocele is an effusion into the tunica vaginalis that accompanies certain affections of the testis. It is frequently associated with acute and chronic epididymo-orchitis. It

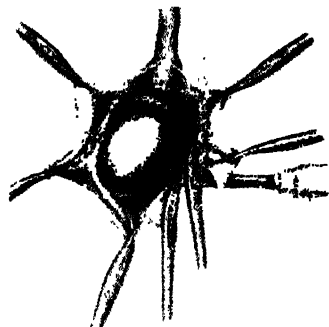


FIG. 1620.—Excision of vaginal hydrocele.

is nearly always present in the syphilitic affections of the testis, and occasionally complicates malignant disease of the organ. A secondary hydrocele rarely attains a large size, and in most instances is lax and does not interfere unduly with the palpation of the testis and its epididymis. Occasionally the question as to whether a hydrocele is or is not secondary to some underlying disease of the testis can be settled only after the contents of the hydrocele have been aspirated.

In the case of acute epididymo-orchitis usually a secondary hydrocele subsides *pari passu* with the primary lesion.

Post-herniorrhaphy hydrocele is a secondary hydrocele that appears after an operation for inguinal hernia in 0.2 per cent. of cases in which there was no evidence of a hydrocele pre-operatively. Possibly this is due to damage of the lymphatic vessels of the tunica vaginalis, which pass with those of the testis, along the spermatic cord, mostly superficial to the blood-vessels.

Hydrocele of a Hernial Sac.—The neck of a hernial sac becomes plugged with omentum or occluded by adhesions, and a hydrocele results (fig. 1359).

Filarial Hydroceles and Chyloceles.—As one proceeds from temperate climates towards the Equator, the incidence of vaginal hydrocele increases. Filariasis (p. 34) accounts for 80 per cent. of hydroceles in some tropical countries (fig. 1621). In most instances neither are microfilariae discovered in nocturnal samples of blood, nor is a history of filariasis elicited. Usually filarial hydroceles follow repeated attacks of filarial epididymitis, and develop rapidly or gradually and can be large or small: frequently they are bilateral. In early cases the hydrocele fluid is similar to that found in the idiopathic variety. In cases of some standing, if the fluid is placed

in a tall glass, after a few hours a film of liquid fat (chyle) will be floating on the surface. This is rich in cholesterol, and is derived from rupture of a lymph varix into the tunica. The presence of chyle is proof-positive of the filarial origin of a hydrocele. Adult worms of the *Wuchereria bancrofti* have been found in the epididymis removed by operation, or at necropsy on these cases. In long-standing chyloceles dense adhesions form between the scrotum and its contents. Filarial elephantiasis of the scrotum (p. 1238) supervenes in a small proportion of cases.

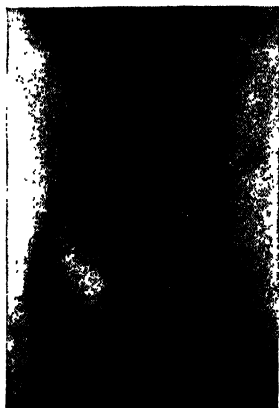


FIG. 1621. — Bilateral filarial hydrocele. Note œdematous scrotal skin at the lowermost part of the left side due to early stage of elephantiasis of the skin. (G. D. Adhia, F.R.C.S., Bombay.)

Treatment.—Acute cases should be treated by rest and aspiration. The more usual chronic cases can be cured by excision of the sac.

Hæmatocele can be either recent, or present as the clinical entity 'old clotted hæmatocele'.

Recent hæmatocele is usually the result of injury of a small blood-vessel during tapping or aspiration of a hydrocele. Prompt refilling of the sac with considerable pain and tenderness, and poor or absent transillumination, leave no doubt as to the diagnosis. The treatment should be urgent operation, with evacuation of blood and excision or eversion of the sac. If this course is not followed a clotted hæmatocele will almost certainly result.

Acute hæmorrhage into a previously normal tunica vaginalis can result from a blow on the testis. The treatment should be similar to the above

for the reasons given, and also because without exploration it is impossible to tell whether or not the testicle is ruptured. Furthermore, a neoplasm can sometimes present in this way.

Old Clotted Hæmatocele.—Curiously, slow hæmorrhage into the tunica vaginalis can occur spontaneously, and apparently painlessly, for most of the patients presenting with an old clotted hæmatocele give neither a history of trauma to the testis nor of pain in the organ. An old clotted hæmatocele simulates a neoplasm of the testis so closely that pre-operative differential diagnosis is sometimes impossible.

Treatment.—Unless exposure of the organ leaves no doubt as to the innocent nature of the swelling, unquestionably orchietomy should be performed. As a rule it is impossible to be certain of the diagnosis until the mass has been bisected. As can be seen in fig. 1622, the testicle is often so compressed as to be virtually functionless, so the responsibilities of removing it unnecessarily are not great.

Russell Howard, renowned in his day as a surgical diagnostician and teacher, was wont to recount how he had performed orchietomy for old clotted hæmatocele on the mistaken diagnosis of malignant testis on three consecutive occasions.

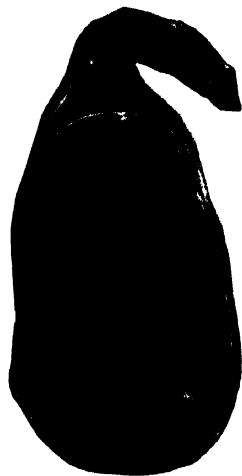


FIG. 1622.—An old clotted hæmatocele. The long-standing pressure has flattened the testis.

CYSTS CONNECTED WITH THE EPIDIDYMIS

1. Cysts of the Epididymis.—A cyst of the epididymis, filled with crystal-clear fluid (as opposed to the barley-water-like fluid of a spermatocele or the amber-coloured fluid of a vaginal hydrocele), is a common condition.

Ætiology.—A cyst of the epididymis is due to cystic degeneration of:

- (a) The appendix of the epididymis (pendunculated hydatid of Morgagni) (fig. 1624).
- (b) The appendix of the testis (sessile hydatid of Morgagni).

(c) The vas aberrans (of Haller).

(d) The paradidymis (or organ of Giraldès), which is the most likely cause.

Clinical Features.—Cysts of the epididymis usually are found during middle life and, due no doubt to their congenital origin, they are often bilateral. They are tense, as opposed to the softness of a spermatocele, and they consist of an aggregation of a number of small cysts which on careful palpation feel like a bunch of tiny grapes (fig. 1623). Cysts of the epididymis are situated *behind* the body of the testis, and on transillumination, because of the numerous septa, their brilliant translucency is finely tessellated, giving an appearance like that of a Chinese lantern.

Treatment.—As epididymis cysts are multilocular, aspiration is useless. If giving trouble, which is unusual, they must be excised.

Cyst of an appendage of the testis is a separate clinical entity. It forms a small globular swelling at the superior pole of the testis; it is usually unilateral, and when pedunculated (fig. 1624) it is liable to undergo axial rotation (p. 1220). The treatment of this variety of cyst is removal by ligation and severance of its pedicle.



FIG. 1624.—Cyst of the pedunculated hydatid of Morgagni.

2. Spermatocele is a unilocular retention cyst derived from some portion of the sperm-conducting mechanism of the epididymis.

Clinical Features.—A spermatocele is nearly always situated in the head of the epididymis, and is therefore above and behind the body of the testis. Filled with fluid resembling barley water and containing spermatozoa, the swelling is typically softer than other cysts occurring within the scrotum but, like them, is translucent. Spermatoceles give rise to few symptoms, and are usually small and unobtrusive. Less frequently they are large enough to attract notice.¹

Treatment.—Small spermatoceles can be ignored. Larger ones, if causing symptoms, can be aspirated or excised via a scrotal incision (see under varicocele). The thin coverings of the cyst are carefully peeled off, layer by layer, and any residual attachments are ligated and divided.



FIG. 1623.—Cystic disease of the epididymis. (After I. Abell.)



FIG. 1625.—A large spermatocele.

ACUTE EPIDIDYMO-ORCHITIS

In many instances inflammation remains confined to the epididymis, in which case it is referred to as epididymitis; when the infection spreads to the body of the testis the condition is known as epididymo-orchitis. In cases where the inflammation is

¹ Sometimes the patient may believe that he has a third testicle (fig. 1625). It is recorded that in the fourteenth century the Pope granted a petition from a gentleman to marry two wives because he possessed three testicles.

situated mainly, if not entirely, in the body of the testis, the term orchitis is employed.

Mode of Infection.—As a rule infection reaches the epididymis via the lumen of the vas, and affects the globus minor first. It is secondary to infection of the urethra, prostate and seminal vesicles. Blood-borne infections affect the globus major of the testis primarily.

Clinical Features.—The initial symptoms are those of acute prostatitis (p. 1178). Some days later an ache in the groin with some increase in fever heralds the onset of epididymitis. Severe pain and swelling of the testis occurs rapidly: the scrotal wall becomes red, oedematous and shiny. The epididymis may become adherent to the scrotal skin and may soften and, later, discharge. Resolution is heralded by scaling of the scrotal skin.

Acute epididymitis due to *Esch. coli*, streptococcus, staphylococcus or pneumococcus is a not uncommon clinical entity. As a rule there is no evidence of urethritis. Especially in *Esch. coli* infection, it is sometimes unaccompanied by infection of the urine (a mid-stream specimen must be sent for bacteriological examination). In the absence of any local cause, the infection is assumed to be blood-borne.

Instrumental acute epididymitis can follow any form of urethral instrumentation. It is most liable to occur when a catheter is tied into the urethra without the necessary aseptic precautions.

Epididymitis complicating non-gonococcal urethritis is more frequent than it is in gonorrhœa, and unless correct antibiotic therapy (p. 1191) is given early, suppuration is usual.

Acute gonococcal epididymitis is much less common than formerly, as a result of the antibiotic treatment of acute gonorrhœa. Epididymitis usually commences during the second or third week of gonococcal posterior urethritis. In 90 per cent. of cases the infection is unilateral. When the epididymis becomes involved the urethral discharge lessens and pain commences in the testis. On the following day the urethral discharge ceases, but the urine is hazy with pus cells. The temperature rises to 102° to 103° F. (38·9° to 39·5° C.), and the patient feels so ill that he goes to bed. About the third day the skin of the scrotum often becomes reddened and adherent to the globus minor, but in cases where the gonococcus is the sole infecting organism suppuration does not occur. After the fifth day the symptoms commence to abate, and by the eighth to tenth day the condition subsides. Induration of the globus minor sometimes persists for months. In cases of persistent infection of the corresponding vesicle, subacute attacks of recurrent epididymitis are not infrequent.

Epididymitis from Retrograde Passage of Urine.—Epididymitis can develop during an unusual exertion or violent strain while the bladder is full, and it is caused by the urine being forced along the vas deferens. The degree of inflammation that results depends upon whether the urine is infected, or if there is an active or latent infection in the prostate or seminal vesicles. This 'reflux' epididymitis can also occur due to the presence of a stricture in the urethra.

Acute Tuberculous Epididymitis.—In cases where the vas is thickened and there is little response to the usual treatment, the possibility of the infection being due to tuberculosis of the epididymis, which is not rare, must receive due consideration.

Acute epididymo-orchitis of mumps develops in about 18 per cent. of males suffering from mumps, usually as the parotid inflammation is waning. The testis, usually the right, becomes swollen and painful. Rarely, the testicular precede the salivary manifestations. Resolution or fibrosis, as opposed to suppuration, nearly always occurs, but in 55 per cent. of cases atrophy of the testis follows. It often takes many months for signs of testicular atrophy to become apparent. A small incision into the tunica vaginalis and drainage, if undertaken early in the attack, prevents this atrophy and relieves the intense pain. It is often stated that the body of the testis is alone involved, but the epididymis is also acutely inflamed, as has been witnessed at operation. Especially in infants, epididymo-orchitis of mumps can occur without parotitis.¹

¹ A complement fixation test for the virus of mumps is sometimes positive.

Epididymo-orchitis in Bornholm disease¹ should be considered in cases of epididymo-orchitis of undetermined aetiology when appearing in epidemic form. The testicular lesion appears especially during the second week of the illness.

Epididymo-orchitis due to the virus of lymphogranuloma venereum.—The infection is bilateral, and is often followed by abscess formation. The diagnosis presents little difficulty, because of the massive inguinal adenitis, but confirmation by the Frei intradermal test is required (p. 1207).

Epididymo-orchitis due to Brucellosis (undulant, abortus or Malta fevers). Acute epididymo-orchitis is sometimes the first sign of this disease. The diagnosis is clarified by appropriate agglutination and skin tests and cultures of the urine and blood.

Acute post-operative epididymitis was a serious and frequent complication of prostatectomy. It has been practically abolished by prophylactic division of the vasa deferentia (p. 1169).

Treatment.—The patient must be kept in bed until the acute symptoms have abated. A broad spectrum antibiotic such as tetracycline should be given (500 mg. q.d.s.) until the result of the urine culture is available. The scrotum is supported on a sling formed by broad adhesive plaster attached across the thighs (fig. 1626). Upon the sling is placed a nest of cotton wool, and in this the inflamed organ rests and cooling lotions are applied. The urine is rendered alkaline, and a high fluid intake is necessary.

Antibiotic treatment should be continued for two weeks or until the inflammation has subsided. If suppuration occurs, drainage is necessary. Atrophy of the testis may follow as a sequela in any case of epididymo-orchitis.



FIG. 1626.—Treatment of acute epididymo-orchitis. Broad adhesive strapping is applied to the thighs to support the testicles.

CHRONIC EPIDIDYMO-ORCHITIS

Chronic Tuberculous Epididymitis.—Nearly 90 per cent. of cases of chronic epididymo-orchitis are tuberculous, and the great majority commence insidiously.

Ætiology.—The frequency with which the globus minor is first attacked indicates that infection is commonly retrograde along the vas deferens from an infected seminal vesicle. Blood-borne infection commences in the globus major.



FIG. 1627.—Tuberculous epididymitis.

Clinical Features.—A slight ache in the testis or a trivial injury, calls the patient's attention to the swelling in relation to the testis. An early manifestation is a discrete, indurated, slightly tender nodule in the globus minor—rarely in the globus major. As the disease progresses other nodules appear, and eventually the entire epididymis becomes involved, and is felt as a firm, craggy, often painless, mass situated behind the testis, which feels normal. In 30 per cent. of cases a lax secondary hydrocele is present. When the testis is anteverted, these changes are found in front of the testis. The vas on the affected

¹ Epidemic myalgia. Bornholm is a Danish island in the Baltic sea.

side sometimes becomes 'beaded' due to submucous tubercles. Rectal examination reveals a thickened, and later an irregular, indurated seminal vesicle of the corresponding side, and sometimes of the contralateral vesicle as well. The prostate in advanced cases contains one or more discrete nodules. In 20 per cent. of cases the patient first presents himself with a 'cold' abscess in the lower and posterior aspect of the scrotum (fig. 1627), or with a discharging sinus, which may have healed and reopened several times (fig. 1628). Untreated, after a varying time (from a few months to several years), the contralateral epididymis becomes similarly diseased. The body of the testis remains uninvolved for a long period, often years.



FIG. 1628. — Discharging sinus from a tuberculous right epididymis.

In two-thirds of the cases there is either active tuberculosis in the renal tract or evidence of previous disease. In the remainder the patient is apparently healthy.

In all cases of chronic epididymitis the urine is examined for tubercle bacilli, if necessary on several occasions, and pyelography should be undertaken. The chest is X-rayed (evidence of disease in about 50 per cent.).

Treatment.—When tuberculous epididymitis is secondary, the primary lesion should receive priority. The epididymitis may well resolve during the treatment.

When renal tuberculosis is present, the renal lesion is treated as detailed on p. 1111. Rarely active pulmonary tuberculosis will be present.

Treatment with streptomycin and associated anti-tuberculous drugs (p. 23) is less effective in genital tuberculosis than in urinary tuberculosis. If there is no sign of resolution within two months, epididymectomy or orchiectomy is advisable. Subsequently, in the absence of evidence of active tuberculosis elsewhere, a further three months' course of suitable and anti-tuberculous drugs is all that is necessary. Care of the patient in a sanatorium is an insurance that he will adhere to the treatment.

Chronic non-tuberculous epididymitis either follows an acute attack or is chronic from the commencement. The condition is difficult to distinguish from tuberculosis, but as a rule the epididymis in non-specific epididymitis is larger and smoother. It is essential to exclude a urethral stricture.

Treatment.—In most cases associated posterior urethritis, vesiculitis, or prostatitis must be eradicated. If the condition fails to resolve after six to eight weeks of conservative treatment and antibiotic therapy, epididymectomy should be performed.

Epididymectomy.—When there is a sinus, or on palpation the epididymis is involved extensively, permission for orchiectomy should be obtained, as it is sometimes impossible to determine whether the body of the testis is implicated before the organ has been displayed. An incision is made from the external inguinal ring to below the testis. If a sinus is present, or the skin is adherent to the epididymis,

the affected skin is included in an ellipse. The testis is withdrawn and the tunica vaginalis is opened. Commencing at the globus minor, the epididymis is separated from the body of the testis by blunt dissection, aided by the diathermy needle. As the globus major is approached great care is exercised: the vascular pedicle entering the testis does so between the upper pole of the testis and the globus major, but it is possible to separate the globus major from the testis without injuring the spermatic vessels. The testis, with its vessels intact, is wrapped in a swab while the vas, still attached to the epididymis, is separated from the spermatic cord as far as the external inguinal ring. The vas is then divided between hæmostats near the epididymis, which is removed. The tip of the hæmostat containing the proximal end of the vas is thrust beneath the skin of the inguinal region in an upward and outward direction for about 1 inch (2.5 cm.). A small nick is made in the skin over the tip of the hæmostat, and the latter, together with the vas, is pushed through. The end of the vas is grasped in a second hæmostat and the redundant vas is drawn on to the surface. This small incision is closed with two sutures, one of which passes through the outer coat of the vas. The protruding portion of the vas is allowed to separate at skin level, and it shrivels and separates in about seven days. In this way infection of the scrotal wound from the lumen of the vas is prevented. The swab covering the testis is removed and any bleeding vessels are ligated, or controlled by diathermy coagulation. If hæmostasis is perfect, the wound is closed without drainage.

Orchiectomy is reserved for those cases where the body of the testis is involved. It is advisable to deal with the vas as has been described in epididymectomy.

Syphilitic Orchitis.—Syphilis attacks the body of the testis, and like other late manifestations of the disease, all three varieties have become uncommon, interstitial fibrosis the least so. The three varieties of syphilitic orchitis are:

(a) *Bilateral orchitis* occurs in congenital syphilis (p. 32).

(b) *Interstitial fibrosis* is bilateral, symptomless, and leads to gradual destruction of the seminiferous tubules. The testes are not enlarged. They are firmer than normal, and there is a gradual loss of testicular sensation. In bisecting the testis, irregularly distributed streaks of fibrosis are seen traversing the parenchyma.

(c) *Gumma.*—The condition is nearly always unilateral. The body of the testis enlarges slowly and painlessly. At this stage it feels extremely hard. Testicular sensation is lost, and there is nearly always an associated secondary hydrocele. Later there is softening anteriorly and the skin of the scrotum becomes inflamed, and a gummatous ulcer forms. If the response to treatment is not rapid it is better to perform orchiectomy than risk the possibility of overlooking a neoplasm with purely coincidental positive serological tests.

Leprous Orchitis.—Over 25 per cent. of male lepers have testicular atrophy due to fibrosis engendered by the direct action of the *Mycobacterium lepræ*, and in 20 per cent. of those so affected the orchitis is associated with gynæcomazia.

NEOPLASMS OF THE TESTIS

The lymphatics of the testis run upwards in the spermatic cord, and, passing through the internal inguinal ring, they branch fountain-wise (behind and adherent to the posterior peritoneum) towards the para-aortic lymph nodes in the region of the origin of the spermatic vessels. Above this level the lymph drains into the thoracic duct which extends through the mediastinum to the left supraclavicular fossa, where it drains into the left innominate vein. Lymphatics from the medial side of the testis may run with the artery to the vas and drain into a lymph node at the bifurcation of the common iliac artery.

When lymphatic spread of a neoplasm occurs, contralateral lymph nodes can be affected. Inguinal lymph nodes are only affected if the scrotum is involved.



FIG. 1629.—The lymphatics of the testes. (After Rouvier.)

MALIGNANT NEOPLASMS OF THE TESTIS

About 99 per cent. of neoplasms of the testis are malignant, and though they only constitute about 1-2 per cent. of malignant tumours in the male, they are one of the commonest forms of cancer in young adult males. Malescent undoubtedly predisposes to malignancy (p. 1216). It is curious that a testicle bearing such a tumour, contained as it is in a bag of skin bereft of fat, and thus more readily accessible to the examining fingers than a tumour of any other organ in the body, only too often escapes detection until it has metastasised. In this connection, perhaps the worst error is to plunge a trocar and cannula into the enlarged testis in the belief that it is a hydrocele, for want of applying the test of translucency. This has been done more often than the reader may be inclined to believe.

Types of Tumour.—Tumours of the testis are classified as follows:

(1) Seminoma	40 per cent.
(2) Teratoma	32 per cent.
(3) Combined Seminoma and Teratoma	14 per cent.
(4) Interstitial tumours	1.5 per cent.
(5) Lymphoma	7 per cent.
(6) Other tumours	5.5 per cent.

The percentage of the first two subdivisions varies in different series. Those series emanating from hospitals for Service personnel show a higher percentage of teratomata. The reason for this is that patients in such hospitals are likely to be of a younger age-group than found in the community at large; the **peak incidence** of teratoma being between twenty and twenty-five years of age, while that of seminoma is between thirty and thirty-five. Seminoma has only once been recorded before puberty.

Seminoma commences in the mediastinum of the testis, and as it increases in size the neighbouring testicular tissue is compressed (fig. 1630). Macroscopically the enlarged testis is firm and smooth, and on section homogeneous and pink or cream in colour. Occasionally fibrous septa give it a lobulated appearance. In rapidly growing tumours areas of necrosis are sometimes present, in which case the consistency is variable.



FIG. 1630.—Seminoma of the testis.

Histologically it is composed of rounded, slightly oval cells with clear protoplasm and large rounded nuclei containing acidophile nucleoli. The cells are arranged in sheets separated by a fine fibrous stroma. Derived from the seminiferous tubules, the cells resemble spermatocytes.

A seminoma metastasises almost exclusively by the lymph vessels to lymph nodes; only occasionally does it disseminate by the blood-stream and give rise to secondary deposits in other situations.

Teratoma arises in the rete testis from toti-potent cells; accordingly, elements of ectoderm, mesoderm, or endoderm are often represented. Usually one of these predominates and attains malignancy. The tumour varies in size from that of a marble to that of a coconut, and even when large, as it is moulded by the tunica vaginalis, the shape of the testis is more or less retained but the surface is usually slightly irregular.

The most usual variety¹ is yellowish in colour and shows cystic spaces (fig. 1631) containing gelatinous fluid. Cartilaginous nodules are often present.

Histology.—The Testicular Tumour Panel have classified teratomas as follows:

(1) **Teratoma Differentiated (T.D.)** (1 per cent.²).—The best-known variety of this type of tumour is a dermoid cyst. If a teratoma has no histologically recognisable malignant components, it is termed differentiated, but it cannot be considered to be benign, because such growths may have metastasised. Many patients have been cured following simple orchiectomy. With radical orchiectomy the outlook is improved, the five-year survival rate being over 80 per cent.

(2) **Malignant Teratoma Intermediate (M.T.I.—A and B)** (30 per cent.), consisting of definitely malignant and incompletely differentiated tissues. In type A some mature tissue will be seen. Type B is more malignant and no fully differentiated tissue can be found. The tumours are mostly radio-resistant (see prognosis, p. 1237).

(3) **Malignant Teratoma Anaplastic (M.T.A.)** (1.5 per cent.) is composed of quite undifferentiated cells, which are radio-resistant.

(4) **Malignant Teratoma Trophoblastic (M.T.T.)** (1 per cent.) contains within the other tissue types a syncytial cell mass with malignant villous or papillary cytotrophoblast (choriocarcinoma). In some cases it is associated with the production of chorionic gonadotrophin and gynæcomazia. Spread is by the blood-stream as well as by lymphatics while the primary tumour is small, and it is often described as the most malignant tumour known. It is usually radio-resistant. Methotrexate may produce a temporary regression but death results within months. However, a choriocarcinoma is frequently associated with teratoid tissue in the form of a terato-choriocarcinoma, and patients with this hybrid choriocarcinoma have survived five years following radical orchiectomy (p. 1236).

Interstitial Cell Tumour.—A tumour of the testis arising in the interstitial cells of Leydig occurs at an early age or the prime of life, and arises either in the cells of Leydig or those of Sertoli.

A Leydig cell tumour masculinises; a Sertoli cell tumour feminises. For clinical reasons these tumours are classified as those occurring before or after puberty.

Prepubertal tumour is characterised by an excessive output of androgen, which is responsible for sexual precocity and extreme muscular development. This results in an infant Hercules, in all respects similar to the condition produced by an adrenal cortical tumour (see p. 608), except that on palpation a swelling of the testis can be elicited. Exceptionally the tumour is bilateral, but it must be noted that in this condition the contralateral normal testis frequently becomes hypertrophied. In so far as the neoplasm is concerned, removal of the testis is curative; in some cases the symptoms regress, in others (where the contralateral testis is hypertrophied) the regression is incomplete.

Post-Pubertal Tumour.—In a high percentage of cases the tumour arises in the cells



FIG. 1631.—Teratoma of the testis containing solid and cystic areas. (Dr. Keith Simpson, London.)

¹ Formerly known as 'fibro-cystic' disease.

² The percentages given refer to all cases of testicular neoplasm.

of Sertoli, and consequently the output of feminising hormone becomes excessive. Gynæcomazia, loss of libido, and aspermia result : in such cases the Aschheim-Zondek test is positive. As a rule the tumour is innocent, and orchiectomy is curative.

The Aschheim-Zondek Pregnancy Test.—The test should be carried out on a twenty-four-hour specimen of the urine and the quantitatively adapted test is of considerable value; thus the highest reading (from 10,000 to 200,000 mouse units/L) is registered by choriocarcinoma, and the lowest reading (less than 500 mouse units/L) by seminomas and adult teratomas.

A positive A.Z. before treatment, which becomes negative afterwards, is an indication that the malignant process may have been ablated. A positive reading during follow-up is a strong indication of recurrence.

Clinical Features of Testicular Tumours.—Various clinical types are met with.

1. **Typical.**—The patient may not seek advice until four to six months after the first symptom, which is enlargement of the testicle. A sensation of heaviness is complained of when the testis has reached two to three times its normal size. Pain is complained of in only about 30 per cent. of cases (Fergusson). There is a history of trauma to the affected side in over 10 per cent. It is generally conceded that injury merely calls attention to the testicular enlargement and in no way initiates the neoplasm.

On examination the body of the testis is found to be enlarged, firm, smooth, and heavy. Later one or more softer bosses may be palpable. In no other disease is testicular sensation lost so early or so completely, but the greatest gentleness must be exercised in eliciting this sign for fear of disseminating the neoplasm. A secondary hydrocele is present in 10 per cent. of cases; it is usually lax, and seldom obscures the enlarged testis. The epididymis is normal at first; later it becomes so flattened or incorporated in the growth that it is imperceptible. The spermatic cord remains normal for a considerable time. It then becomes thickened, due to cremasteric hypertrophy and enlargement of testicular vessels. The vas is never thickened. Rectal examination reveals no abnormality in the prostate or seminal vesicles.

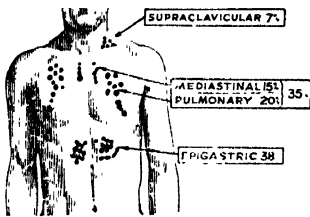


FIG. 1632.—Distribution of metastases in teratoma of the testis. (R. S. Ferguson's figures.)

Next, the sites of possible metastases (fig. 1632) should be examined. The abdomen is palpated for secondary retroperitoneal deposits, their most usual situation being just *above the umbilicus* on the same side, the opposite side, or both sides. The left supraclavicular region is examined, and occasionally the first presenting sign is an enlarged supraclavicular lymph node. An X-ray of the chest may show metastases in the lungs, particularly in cases of teratoma.

2. **When the predominant symptoms are due to metastases:** (a) with a seminoma the patient complains of abdominal or lumbar pain, and on examination a mass is discovered in the epigastrium. (b) Pulmonary metastases from a teratoma of the testis give rise to few, if any, early symptoms. Later the patient may complain of pain in the chest, dyspnoea, and perhaps hæmoptysis.

In either case the enlargement of the testis may not have been noticed by

the patient, and is discovered only by a thoughtful clinician. Sometimes the primary growth is tiny and will only be discovered by operative exploration.

3. Atypical Cases.—The variations are:

(a) Some cases (7 per cent.) simulate *epididymo-orchitis*; there may even be organisms in the urine. Failure to respond to a short sharp course of antibiotics should make one say 'this could be a tumour' and action must be taken. Rarely, patients will present with severe pain and acute swelling due to a hæmorrhage. Between 1 to 5 per cent. of cases have gynæcomastia (mainly the teratomas).

(b) *The Hurricane Type*.—Metastases from a highly malignant tumour bring about a fatal termination in a matter of weeks (Gordon-Taylor).

(c) *The Slowly Growing Type*.—In a few cases of teratomas, the patient notices an increasing enlargement of the testis for two or three years.

MANAGEMENT OF A SUSPECTED CASE OF NEOPLASM

The action to be taken when the clinical diagnosis is made is:

- (1) Orchiectomy (or exploration).
- (2) Confirmation of pathology by serial histology.
- (3) Radiography.
- (4) Radiotherapy.

1. Orchiectomy (retrograde simple¹ orchiectomy).—This should be done at once. Through an inguino-scrotal incision the inguinal canal is opened to display the spermatic cord, which is divided and ligated at the level of the internal inguinal ring. The spermatic cord and the testis are then removed from above downwards by separating them from their attachments. The wound is closed, and radiotherapy starts as soon as the pathology is confirmed and the sutures have been removed.

Exploration.—On occasion when the clinical diagnosis is in doubt, delay is still dangerous, and nothing is lost by exposing the testis first, providing a soft clamp is placed across the cord above to prevent dissemination. The testis may have to be split open and any doubtful material subjected to immediate frozen section. Wedge biopsy without frozen section and needling should not be attempted for fear of involving the scrotum and opening up an alternative way of lymphatic spread (to inguinal lymph nodes) (p. 1231).

2. Confirmation of Pathology.—The pathologist will report the type and grade of the tumour (p. 1233).

3. Radiography should be used as follows:

- (a) X-ray of the chest, to reveal any secondary deposits.
- (b) Excretory urography (I.V.P.). This has two purposes: (1) Protection of kidney from radiotherapy, for besides ascertaining the function of the kidneys, their position must be marked out so that they may be shielded during irradiation in order to prevent radiation nephritis and hypertension.
- (2) Detecting the presence and extent of retroperitoneal metastases by the displacement of the ureters or deformity of a renal pelvis. Changes in radiographs after irradiation may indicate the radio-sensitivity of the tumour.

¹ Simple, as opposed to the radical operation p. 1236.

(c) Lymphangiography can demonstrate secondary deposits in para-aortic lymph-nodes.

4. **Radiotherapy.**—Most, but not all, seminomas are highly radio-sensitive. Most, but not all, teratomas are radio-resistant. Well over 80 per cent. of patients suffering from seminoma of the testis without pulmonary metastases survive more than five years after simple orchiectomy followed by radiotherapy.

From its inception, radiotherapy of tumours of the testis has been based on the assumption that retroperitoneal lymphatic metastases have occurred whether or not they are palpable. Such irradiation should be given by a cobalt or linear accelerator X-ray unit to permit the desired deep dose to be given in shorter time, with greater certainty, with less injury to superficial tissues, and with less general reaction than is possible with machines of lower voltage. When there is no clinical or radiological evidence of metastases the radiation is confined to the area between the scrotum and the 10th dorsal vertebra, with suitable screening of the remaining testis. In patients who are eager to have more children the remaining testis is shielded by lead blocks and moulds. Even so there is some danger of mutations. Only careful follow-up of descendants will show whether this procedure is the correct one. The maximum tumour dose is 3,000 r. for a seminoma, and 4,500 r. for a teratoma, given over some five or six weeks. It may take longer if interrupted by an excessive fall in the platelet count.

In more advanced cases radiotherapy is given through one inguinal, four abdominal, two mediastinal and two left supraclavicular portals. Chlorpromazine may be given to control the vomiting of radiation sickness, so common in patients receiving radiotherapy in high dosage.

Pulmonary Metastases.—Sometimes irradiation results in regression, but almost invariably this is followed by fresh deposits. Frequently the dose of X-rays converts a condition that was symptomless into one accompanied by severe pulmonitis, discomfort and respiratory deterioration, and it is doubtful if the X-ray treatment of pulmonary metastases is worth while (Cox).

Block Dissection of Retroperitoneal Lymph Nodes (radical operation).—With improvements in radiotherapeutic apparatus and techniques this operation is not often employed. It supplements simple orchiectomy and radiotherapy in resistant cases of teratoma intermediate, teratoma anaplastic, and teratoma trophoblastic (p. 1233), provided, of course, that there are no demonstrable secondary deposits in the thorax or a large fixed mass in the epigastrium, and the patient is in other respects fit to undergo an extensive operation. The best time is after the patient has recovered sufficiently from the effects of radiotherapy, which helps to devitalise metastases, if present.

Principles of Technique.—An extensive retrograde exposure is required to allow the removal of spermatic vessels and lymphatics from the internal ring up to the region of the renal vessels (Anatomy, p. 1231). The vas is transected above the bladder. Only part of the incision is made to start with, for through this the peritoneum is opened and the upper abdomen is explored to exclude very high fixed lymph nodes which would make the case inoperable.

Prognosis of Testicular Tumours.—The histological appearance of the tumour and the presence or absence of metastases at the time of operation are the two main factors in assessing prognosis:—

Seminoma.—No evidence of metastases; with orchietomy and radiotherapy 85 per cent. survive five years. If metastases are present, only 25 per cent. survive five years.

Teratoma.—The histological appearances are very important (p. 1233). Excluding Teratoma Differentiated, if no metastases are present, up to 40 per cent. survive five years. If metastases are present only 1 to 5 per cent. survive three years.

Testicular tumours in children most commonly appear during the first year of life and are exceedingly rare after the third birthday. The tumours at this time of life are teratomas, many of these being anaplastic (T.A.) in which event, in spite of treatment, the patient dies within a few months. Now and then the tumour proves to be differentiated (T.D.) when, following orchietomy alone, the prognosis is exceptionally good. On rarer occasions still, the tumour proves to be an interstitial cell tumour (p. 1233), in which case, following orchietomy, the prognosis is also excellent.

Tumours of the Epididymis.—Mesothelioma is a unique innocent tumour that usually springs from the globus minor: most examples have been about the size of a cherry. Sarcoma, and very rarely carcinoma, arise in the epididymis, and those who put their trust in the aphorism that swellings of the epididymis are invariably inflammatory may one day rue it. If the swelling in question does not respond to rest and antibiotic therapy quickly, it should be treated by epididymectomy or, if obviously malignant, by orchietomy.

THE SCROTUM

Prepenile scrotum is an exceedingly rare congenital condition. The scrotum, suspended from the mons pubis, is situated anterior to the penis.

Idiopathic gangrene of the scrotum (*syn.* Fournier's gangrene) is uncommon but fearful in effect. It is a vascular disaster of infective origin. The three cardinal characteristics of this disease are: (1) sudden appearance of scrotal inflammation in the midst of apparently good health; (2) rapid onset of gangrene (fig. 1633); (3) absence of any cause in over half the cases. It has been known to follow minor injuries or procedures in the perineum, such as a bruise, scratch, dilation of stricture, injection of anal fissure or opening a periurethral abscess.



FIG. 1633.—Fournier's gangrene.
(Max Pemberton, F.R.C.S., London.)

The hæmolytic streptococcus (sometimes micro-aerophilic) associated with other organisms (staphylococci, *E. coli*, *Cl. Welchii*) set up a fulminating inflammation within the scrotal subcutaneous tissues that results in obliterative arteritis of the arterioles supplying the overlying skin (cf. gangrene of the abdominal wall, p. 1029).

The clinical features are: sudden pain in the scrotum, prostration, pallor, and pyrexia. At first only the scrotum is involved: still unchecked, if the patient survives, the cellulitis spreads along those planes so well known in superficial extravasation of urine (p. 1190). Sometimes the entire scrotal coverings slough, leaving the testes hanging exposed, bared to their tunica, though, remarkably, free from gangrene.

Treatment.—Pending the bacteriological report, chloramphenicol is the antibiotic of choice as the organisms are usually sensitive to it. A topical application of tyrothricin is also of help. Ceporin in large doses, may be used if the organisms are sensitive.

If this treatment does not produce any improvement within six to eight hours, the scrotum should be incised. If gangrene has commenced, wide excision of all sloughing areas saves time, rids the patient of scrotal tissue that is doomed, provides the freest possible drainage, and stops the spread of gangrene.

Filarial elephantiasis of the scrotum is confined to those who dwell, or have dwelt, in tropical and subtropical countries, and is due to obstruction of the lymphatic vessels of the pelvis by *Wuchereria bancrofti* (p. 35), with superadded infection and lymphangitis. The earliest manifestation is an attack of funiculitis with the development of a secondary hydrocele. Repeated attacks cause the scrotum to remain œdematous and the scrotal skin and subcutaneous tissue greatly thicken. In long-standing cases the scrotum becomes immense, and the penis is buried within it (fig. 1634).



FIG. 1634.—Elephantiasis of the scrotum, burying the penis. (Professor M. Bahadur Khan, Hyderabad, India.)

of the penis, if necessary, is the only curative treatment. It should be especially noted that the *inner layer* of the prepuce is rarely involved: it should therefore be spared and in many instances it can be utilised to cover the bare shaft of the penis (Bowesman). In all cases concomitant hydroceles are dealt with by excision of the sac at the time of the operation.

Non-filarial elephantiasis can be acquired in non-tropical as well as tropical climates, and it is consequent upon sclerosis of lymphatic vessels due to lymphogranuloma venereum. Non-filarial elephantiasis never reaches the extravagant proportions of tropical elephantiasis. There is no effective medical treatment: the scrotum-thigh cutaneous tunnel operation described above is recommended.

Sebaceous cysts are common in the skin of the scrotum. They are usually small and multiple (fig. 1636). Sometimes a large sebaceous cyst suppurates, and when it does so the odour emitted is particularly obnoxious. A suppurating scrotal sebaceous cyst may be mistaken for a carcinoma of the scrotum, which calls to mind Cock's peculiar tumour (p. 101).



FIG. 1636.—Sebaceous cysts of the scrotum.

Treatment.—There is no medical treatment for the condition. The principle of operative treatment is the construction of new lymphatic pathways. This can be accomplished by cellular-cutaneous bridges of generous size uniting the scrotum to the thigh, as in the Keetley-Torek operation, thus permitting the scrotal lymph to be absorbed via the uninvolved thighs. In very advanced cases excision of all involved skin (fig. 1635), together with implantation of the testes into the thighs and skin grafting of the bared shaft

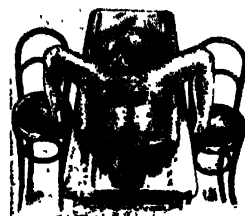


FIG. 1635.—Position of patient for removal of an enormous scrotal

Carcinoma of the Scrotum.—In the last century squamous epithelioma of the scrotum was rife amongst chimney sweeps.¹ With improved working conditions, this occupational disease ceased to exist in as far as chimney sweeps were concerned, but with the mechanisation of the cotton industry, lubricating oil from the spinning jenny soaked the crutch of the mule spinner's trousers, and proved more carcinogenic than soot. Now that a lubricating oil free from

¹ The disease was described by Percival Pott (1714–1788), Surgeon, St. Bartholomew's Hospital. In those days the chimney sweep's apprentice climbed up the chimney.

Charles Bowesman, Contemporary. Formerly Surgical Specialist, Colonial Medical Service, Kumasi, Ghana.
Edward Cock, 1806–1892. Surgeon, Guy's Hospital, London.

impurities has been substituted it is hoped that carcinoma of the scrotum will no longer attack¹ mule spinners in particular. A few cases still occur in tar and shale oil workers, but in the majority of cases encountered at the present time there is no known ætiological factor. It is remarkable that, in contradistinction to carcinoma of the penis, carcinoma of the scrotum is almost unknown in India and Asiatic countries.

Clinical Features.—The growth commences in a wart or an ulcer (fig. 1637). As it extends it may involve the underlying testis.

Treatment.—The growth, together with a considerable margin of healthy tissue, is excised by diathermy. If, as is often the case, the inguinal lymph nodes are enlarged and do not subside with antibiotic treatment, a block dissection of these nodes, and of the external iliac nodes, should be carried out, usually on both sides.



FIG. 1637.—Carcinoma of the scrotum with bilateral metastases in the inguinal nodes. (Department of Medical Photography, Cardiff Royal Infirmary.)

MALE INFERTILITY

Causes.—These are as follows :

1. *Impotence* due to (a) malformations or loss of the penis ; (b) psychic causes.
2. *Oligospermia or aspermia* due to small (fibrotic) or soft (partially atrophic) testes resulting from mumps (quarter of cases), varicocele (p. 1220), cryptorchism, over-exposure to X-rays, and occasionally other varieties of bilateral epididymo-orchitis.
3. *Aspermia Due to Faulty Sperm-conducting Mechanism.*—Leading examples are (a) urethral fistula, (b) tight urethral stricture, (c) obliteration by scar tissue of both common ejaculatory ducts or both vasa deferentia. Fifteen per cent. of all cases of infertility are due to gonorrhœa.

Investigation includes : (a) three sperm counts at weekly intervals, and an adjudication of the average. An average volume of 3 ml. of semen is good evidence that there is no deficiency in circulating androgen ; (b) testicular biopsy.

Treatment.—*Hormone Therapy.*—Recent experience has shown that low sperm counts in the absence of infection and obstruction may be helped by pituitary gonadotrophins.

Operative treatment of varicocele has proved encouraging (p. 1222).

Vaso-epididymal Anastomosis.—The criteria for doing this are :

1. Aspermia must be demonstrated on three separate occasions.
2. Testicular biopsy must show that the aspermia is not due to defective spermatocytes.
3. The secretion from the seminal vesicle must be present in the ejaculate. Its absence indicates a block in the region of the pelvic portion of the vas, which would render the operation useless.
4. The patient should be under forty years of age.

Operation.—The vas is divided at least 2.5 cm. above the portion considered to be obliterated, and in order to make certain of its patency, the proximal end is cathe-

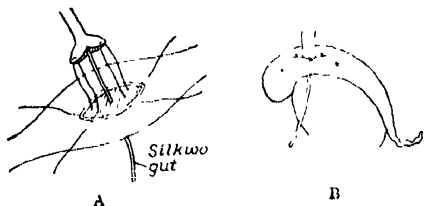


FIG. 1638.—End-to-side vaso-epididymal anastomosis. (After S. Popelka et al.)

¹ As it takes up to twenty years for the result of exposure to carcinogens to become manifest, the outcome as yet is uncertain.

terised with a suitably-sized piece of silkworm gut. The silkworm gut is removed and the free end of the vas is split for a short distance. The silkworm gut is then reinserted. End-to-side anastomosis is carried out with 4/0 silk, the flaps resulting from the bifurcation being carried into the lumen of the epididymis (fig. 1638A). The silkworm gut splint is brought out between the epididymis and the body of the testis (fig. 1638B), and it is also well to pass this through a minute puncture in the scrotum and withdraw it after several days, when the anastomosis can be presumed to have united. By this method, over 50 per cent. of cases show copious sperm in the ejaculate.

INDEX

A

- Abdomen, blast injuries of, 897
 burst, 733, 1028-1029
 following splenectomy, 777
 diabetic, 964
 Abdomino-perineal excision for rectal carcinoma, 1018
 Abdominal wall, 1024, 1027-1030
 Abortion, peritonitis following, 877
 Abrams' pleural biopsy punch, 655
 Abscess, 1, 126
 acute, 12-14
 alveolar, 466-468, 472
 amoebic liver, 798-801
 ano-rectal, 1003-1006
 appendicular, 964-965, 970-971
 breast, 1, 593, 604
 cerebral, 376-381, 670
 cold, 155, 238, 239, 287, 289, 529, 650-651, 1181
 collar-stud, 154, 163, 518, 529, 530, 531
 dysenteric, 798-801
 extradural, 376-377
 formation in acute pyogenic osteomyelitis, 230
 in Pott's disease, 240, 241
 in suppurative parotitis, 509
 gas, 46
 intra-cranial, 376-381
 ischio-rectal, 906, 909, 1004-1005
 lung, 669-671, 679
 of abdominal wall, 1024
 of gall bladder, 830
 of hand, 162-163, 167-168
 of liver, 797-801
 of seminal vesicle, 1181
 of spleen, 789
 parapharyngeal, 527, 621
 pelvic, 873, 971
 pelvi-rectal, 1006
 perianal, 911, 1004
 perigastric, 753
 perinephric, 1107-1108
 peritonsillar, 620-621
 periurethral, 1198-1199
 plantar, deep, 174
 pleural, 655
 prostatic, 1178, 1181
 psoas, 240
 diagnosis from femoral hernia, 1049
 pulmonary, 669-671, 679
 retropharyngeal, 621-622
 tuberculous, 622
 root, 468
 spinal epidural, 5411
 subareolar, 594
 subcortical, 378
 subdural, 376-377
 submucous, of rectum, 1005
 subperiosteal, 230
 Abscess—*contd.*
 subphrenic, 753, 873-874
 subpal, 378
 tropical, 798-801
 diagnosis from subphrenic, 875
 tuberculous, of lymphatics, 154
 Acetabulum, fractures of, 214
 travelling, 265, 293
 Achalasia of oesophagus, 721-723
 Achondroplasia, 252
 Acid-base balance, 95-97
 Acid-phosphatase estimations, 1161
 Actinosis, 95, 96-97
 Actinosis, diaphyseal, 253
 Actin, 42
 Actocyanosis, 140
 Actomegaly, 253, 394
 Acromial tip, fracture of, 197
 Acromio-clavicular joint, dislocations of, 267
 Acrosclerosis, 139
 Actinomycosis, 22-23
 cervico-facial, 22, 473
 differential diagnosis, 23, 530, 915
 of breast, 595
 of jaw, 472, 473
 of liver, 801
 of neck, 531-532
 of right iliac fossa, 914-915, 965
 of thorax, 22-23
 treatment of, 3, 23, 532
 Adamantinoma, 474, 475
 Addison's disease, 575, 578
 Adenitis, cervical, tuberculous, 528
 non-specific mesenteric, acute, 885-886
 of facial lymph node, 493
 simple or non-specific, 153
 specific, 34, 154-155
 Adenocarcinoma, 58
 of bladder, 1151
 of kidney, 1115-1118
 Adenoids, 617-620
 Adenolymphoma, 511-512
 Adenoma, 55-56, 392-395
 of bronchus, 663
 pancreatic, 861
 parathyroid, 570
 pituitary, 392
 sebaceous, 103
 thyroid, 549-550, 555-557, 560-561
 umbilical, 1026-1027
 Adiposa dolorosa, 54
 Adrenal cortex, 576-582
 tests, 576-577
 gland, 575-586
 medulla, 575, 583
 tumours of, 583-586
 Adrenalectomy, 138, 578, 581-583, 613-614, 1177
 Adrenaline, 583
 in cardiac arrest, 688, 689

- Adreno-genital syndrome, 580, 581
 Afferent loop, distension, 759
 obstruction following gastrectomy, 852
 syndrome, 762
 Agranulocytosis, 1, 545
 Ainhum, 47
 Air embolism, 132-133, 526
 Albee's bone-graft operation, 403
 Albers-Schönberg's disease, 253
 Albright's syndrome, 248
 Aldosterone, 84, 576
 Aldosteronism, primary, 579, 581
 Alkalosis, 87, 95-96
 Allergic reactions to blood transfusion, 76
 Alveolus, tumours of, 470
 Amazia, 588, 590
 Ameloblastoma, 474
 Amœbiasis, intestinal, surgical aspects of,
 908-910
 of liver, 798-801
 Amputation, in arthritis, acute suppurative,
 281
 in bone tumours, 259, 261, 262
 in carcinoma of penis, 1211-1212
 in diabetic gangrene, 41, 42-43
 in fractures, indications for, 192
 in gangrene, 41, 42, 45, 47
 in Madura foot, 175
 in osteoclastoma, 259
 in osteomyelitis, 235
 in sores and ulcers of foot, 439
 in tuberculosis, 238, 291
 'neuromata', 57
 Amyloid disease, 35, 494
 Anæmia, 489
 following gastrectomy, 764
 hæmolytic, 780-782
 hypochromic, 721
 Anal canal and rectum, 975-979
 squamous-celled carcinoma of, 1021
 chancres, 38, 987
 condylomas, 29, 996
 crypts, 976
 fissure, 985-988
 injuries, 984-985
 papilla, hypertrophied, 988
 stricture, 995, 1010-1011
 valves, 976
 Anaphylaxis, acute and local, 11, 17, 18
 Anderson-Hynes operation, 1087
 Aneurysmorrhaphy, 705
 Aneurysms, 124-128
 aortic, 125, 126, 127-128, 705
 arterio-venous, 129, 484
 cirroid, 352, 484
 dissecting, 120, 124, 128, 706
 false, 124, 125, 128
 fusiform, 124, 125
 intracranial, 398-400, 484
 mycotic, 125
 of hepatic artery, 795
 of renal artery, 1083
 of splenic artery, 777-778
 saccular, 124, 125, 705
 true, 124, 125
 ventricular, 705
 Angina cruris, 118
 pectoris, 118, 450, 704
 Angiocardiography, 686
 Angiofibroma, nasopharyngeal, 625
 of larynx, 640
 Angioma, 57, 103-104
 cavernous, 104
 intracranial, 399-400
 plexiform, 104
 renal, 1113
 sclerosing, 102
 Angiomyoneuroma, 57
 Ankle joint, dislocations, 278
 fractures of, 225-227
 tuberculosis of, 294
 Ankyloglossia, 501
 Ankylosis, bony, 289
 fibrous, 271, 289
 in tuberculous arthritis, 289, 290, 294
 Ano-rectal ring, 977
 syphilis, 987, 1002
 Anoxia, myocardial, 688
 Anthrax, 11, 12, 17
 Antibacterial therapy, 2-4
 'Antibioma', 13
 Antibiotic agents, 2, 3-4
 resistant organisms, 1-2, 1103
 therapy, 4
 dangers of, 3, 4, 971
 Anticoagulant therapy, 123, 131-132
 Antigens and antibodies, 73
 Antrectomy, 749-750
 Antrostomy, intranasal, 480
 palatal, 477
 Antrum, maxillary, growths of, 476-477
 infection of, 478-480
 Anuria, 1067-1073, 1169
 Anus, carcinoma of, fissure simulated by,
 987
 congenital abnormalities, 979-982
 imperforate, 979-981
 papilla of, hypertrophied, 988
 tubercule of, hypertrophic, 996
 tumours of, malignant, 1021-1022
 Anxiety states, surgical treatment of, 400
 Aorta, coarctation of, 693-695
 incompetence of, 703
 stenosis, 702
 Aortography, of aorta, 686
 translumbar, 120, 1066-1067
 Aorto-iliac disobliteration, 136
 Apoplexy, adrenal, in newborn, 577
 Appendicectomy, 966-968, 971
 complications of, 972
 Appendicitis, acute, 885, 954-970
 conditions simulating, 887-888, 909,
 962
 differential diagnosis of, 960-964, 1101
 in aged, infants, and pregnancy, 959-
 960
 position of appendix in, 953-954
 treatment of, 966-968
 postoperative complications, 972
 chronic, 973
 recurrent, 972-973
 subacute, 972
 Appendicular colic, 958
 dyspepsia, 973
 Appendix, vermiform, 953-974
 abnormalities, congenital, 954
 abscess of, 964-965, 970-971
 carcinoid tumour of, 916, 974

- Appendix, vermiform—*contd.***
 carcinoma of, 974
 diverticula of, 973
 endometriosis of, 974
 intussusception of, 973
 mass, 964–966, 968
 mucocele of, 973
 tumours of, 974
- Arachnoiditis, spinal, 417**
- Argentaffin tumour of appendix, 916, 974**
 of ileum, 916
 of liver, 816
- Arnold-Chiari malformation, 396**
- Arteries, diseases of 123–141**
 investigations, 118–121
 grafting of, by-pass, 121, 127, 136
 injuries of, 123–124, 185
 occlusion of, 118
 surgery of, 117–141
 operations on, 121
- Arteriography, 120–121, 129, 134**
 cerebral, 363
 in atherosclerosis, 144
 in tumour, carotid, 533
 cerebral, 387
 pituitary, 393
 renal, 119, 1066–1067
- Arteriotomy, 121**
- Arteritis, 137–138**
- Artery, common carotid, ligation of, 531**
 for aneurysm, 129, 398
 for intracranial angioma, 400
 resection with, for carotid body
 tumour, 534
 grafting, 121, 127, 136
 hepatic, aneurysm of, 795
 internal carotid, ligation and division of,
 for aneurysm, 129, 398
 renal, aneurysm of, 1083
 serpentine, 126
 splenic, aneurysm of, 777–778
 stenosis of, 119, 137
 swellings, 125–126
 vertebral, stenosis of, 137
- Arthritis, acute, 282**
 rheumatic, 231
 suppurative, 231, 232, 279–281
 chronic, 282, 285–302
 gonococcal, 282
 in typhoid, 910
 rheumatoid, 295–296
 tuberculous, 285–295
- Arthrodesis, extra-articular, 293**
 in flat-foot, 343
 in infantile paralysis, 350
 in injury of sciatic nerve, 439
 in joint tuberculosis, 290, 291, 293, 294
 in osteoarthritis, 300
 in parasyphilis, 283
 in Pott's disease, 241
 in rheumatoid arthritis, 296
- Artificial pneumothorax, 652**
- Arytenoidectomy, unilateral extralaryngeal,
 638**
- Aschheim-Zondek pregnancy test, 1234**
- Ascites, 807, 880–882**
- Asclitic tuberculous peritonitis, 879–880**
- Aspermia, 1238**
- Asthma, thymic, 685**
- Astragalus, fractures of, 228**
- Astrocytoma, 382, 385, 386, 388, 389, 482**
- Astrup apparatus, 97**
- Atelectasis, 679, 777**
- Atherosclerotic obstruction, 133–137**
 of legs, 133–136
- Athlete's foot, 34**
- Atlanto-axial joint, pathological dislocation
 of, 403**
- Atrial septal defect, 696**
- Atrio-femoral, left, by-pass, 691**
- Auricle, cervical, 520**
- Auriculo-temporal syndrome, 514**
- Auscultation, arterial, 119**
- Auto-antibodies, thyroid, 564**
- Axonotmesis, 185, 434–436, 446**

B

- Bacilluria, tuberculous, 1108**
- Bacillus coli shock, 78, 79**
- Bacteriemia, 15**
- Bagdad sore, 35**
- Baker's cyst, 54, 299, 318**
- Baker's common bile duct dilators, 857**
- Balanoposthitis, 1205–1206**
- Ballance's sign in ruptured spleen, 776**
- Bankart's operation, 270**
- Barrel bandage for jaw fracture, 196–197**
- Basal metabolic rate, as thyroid function
 test, 538**
- Baseball finger, 307**
- Bassini's herniorrhaphy, 1040–1041**
- Battle's operation for double cleft-lip
 and unfused premaxilla, 454**
- Bazin's disease, 141**
- Bedside biochemistry, 97**
- Bedsores, 46**
 in spinal cord injury, 406, 408, 410
- Bee stings, 17–18**
- Bell's nerve, 443**
- Bell's palsy, 431, 432**
- Belsey's operation for hiatus hernia, 718**
- 'Bends', 133**
- Bennett's fracture, 211**
- Bezoars, 734**
- Bigelow's evacuator, 1136, 1169**
- Bile ducts, abnormalities of, congenital,
 822–824**
 atresia, 823–824
 and gall bladder, 818–845
 carcinoma of, 844–845
 injuries of, 825
 stones in, 820, 836–840
 stricture of, 841–842
- Bile, 'white', 838**
- Bilharziasis of bladder, 1146–1148**
 of liver and spleen, 786
 of rectum, 1002–1003
- Biliary stasis, 826, 843–844**
- Biligradin, in cholecystography, 820**
- Billingsgate hump, 318**
- Billroth operation for gastric ulcer,
 747–748, 750, 758, 759, 760**
- Bisgaard treatment of venous ulcer, 149–150**
- Bites, 18, 164**
- Bladder, 1121–1159**
 accidental wounding of during operation,
 1126, 1140, 1169

- Carcinoma, 37, 49, 50-53, 57-58, 60-61
 basal-celled, 105. *See* *Rodent ulcer*
 colloids, 58, 768
 glandular, 58
 methods of spread, 50-52, 502, 603, 919
 of bone, 262-264
 papilliferous, causing intussusception, 941
 simplex, 58
 squamous-celled, 49, 58, 107, 457, 460, 462
See also under names of organs and regions
- Cardiac arrest, 75, 681, 688-689
 infarction, shock and, 79
 investigation, dye dilution studies in, 687
 ischaemia, 704
- Cardiochalasia, oesophageal, 731
- Cardiospasm, 721-723
- Caries, dental, 465-466
 sicca, 237, 286, 295
- Carotid body, tumour of, 60, 532-534
 stenosis, 137
- Carpal bones, dislocation of, 272-273
 fracture of, 210-211
 tunnel compression, 312-313
- Cartilage, 301
 branchial, 520
 injuries of, 276-278, 525
 loose in joints, 301
 semilunar, cyst of, 278
- Caruncle of female urethra, 1202-1203
- Casoni's intradermal test, 804
- Cat-scratch fever, 34
- Catheter, insertion of, 1128
 life, 1167
 suprapubic, technique of inserting, 1128
- Catheterisation, arterial, 120
 cardiac, 686-687
 retrograde percutaneous, 120
 suprapubic, 1128
 ureteric, 1098, 1156
 urethral, insertion of catheter, 1098
- Cations, 84
- Cauda equina, injuries of, results of, 409-410
 tumours of, 416, 417
- Causalgia, 440, 448, 449
- Cavernotomy, renal, 1112
- Cavernous sinus, thrombophlebitis of, 458, 485
- Celestin tube, 727
- Cellulitis, 1, 14-15, 99, 161
 of abdominal wall, 1029
 of hand, 160, 161
 of neck, 15, 526-527
 of stomach, acute suppurative, 736
 orbital, 14-15, 484-485
 submandibular, 467
- Central venous pressure, measurement of, 69
- Cerebellar tumours, 385
- Cerebral compression, 361-362
 contusion, 357-361
 puncture, 388
 tumours, 381-396
- Cervical disc protrusions, 424
 fractures, 407
 lymphadenitis, 528-530
 plexus, injuries to, 440
 rib, 139, 522-524
- Chancere, 28-29
 hard, 1206
 of anus, 38, 987, 1002
 of lip, 37, 456, 457
 of nipple, 589
 of tongue, 497
- Chancroid, 31
- Charcot's biliary triad, 837
 intermittent hepatic fever, 796
 joints, 265, 283-284
- Charnley compression arthrodesis of knee, 300
- Cheilosis, 490
- Chemodectoma, 533
- Chemosis, 484, 548
- Chemotherapy, 2-3, 631-632
- Chest, injuries to, 645-650
 wall, diseases of, 650-652
- Chiari's syndrome, 805
- Chilblains, 139, 140
- Chimney sweep's cancer, 49, 1239
- Chloroma, 158
- Choanal polyp, 626
- Chokes, 133
- Cholangio-adenomata, 815
- Cholangio-hepatitis, asiatic, 842-843
- Cholangiography, 820-822, 823, 833-834, 845
- Cholangioma, 816
- Cholangitis, 796
 suppurative, 838
- Cholecystejunostomy, 858, 866
- Cholecystectomy, 829, 831-832, 833
 persistent symptoms after, 843
 technique of, 834-835
- Cholecystitis, 829, 874
 acute, 829-832, 910
 chronic, 832
 diagnosis from acute appendicitis, 961, 1101
 glandularis proliferans, 833
- Cholecystography, 819-820
- Cholecysto-hepatic duct, 835
- Cholecystostomy, 832, 835, 862
- Choledochoduodenostomy, 840, 842
- Choledocholithotomy, 839
- Choledochotomy, 835, 838-840
- Choledochus cyst, 824
- Cholelithiasis, 825. *See also* *Gall stones*
- Cholera, staphylococcal, 5
- Cholesteatoma, of middle ear, 462
- Cholesterol polyposis of gall-bladder, 833
 serum, test, 540
- Cholesterosis, 833
- Chondroma, 255-256, 645, 651
- Chondromatosis, synovial, 301
- Chordee, 1209
- Chordotomy, 420
- Christmas disease, 76
- Chromaffin tissue, 583
- Chromium, radioactive, in haematological investigation, 781
- Chvostek's sign, 569
- Chyloceles, filarial, 1225-1226
- Chylothorax, 655
- Chylous reflux, in lymphoedema, 152, 153
- Cine-cystography, micturating, 1105
- Circle of Willis, aneurysm of, 398
- Circulation, collateral, 117
 extra-corporeal, 690, 703

- Circumcision, 1203-1204
 Cirrhosis of liver, 805-808, 881
 Cisternal puncture and myelography, in
 spinal canal tumours, 418-419
 Claudication, distance, 118
 intermittent, 117-118, 123, 134, 137
 Clavicle, fracture of, 197-199
 Claw-foot, 343-344
 Cleft-lip, and cleft-palate, 451-454
 Cloquet's hernia, 1049
Clostridium sporogenes, 44
 tetani, 7
 welchii, 44
 Club-foot, 344-346
 Clutton's joints, 31, 282-283, 286
 Cobalt, radioactive, therapy, 505, 1155
 Cocaine medication, 61
 Coccydynia, 214
 Coccyx, fractures of, 214
 Cochlear concussion, 462
 Cockett and Dodd operation, for venous
 ulcer, 150
 Cock's 'peculiar tumour', 101
 Codman's triangle, 260
 Coeliac rickets, 245
 Cold water treatment, of burns, 113-114
 Colectomy, in ulcerative colitis, 908
 Coleman's postural drainage, for
 unconscious, 359
 Colic, appendicular, 958, 961
 gall-stone, 828-829
 renal, 1061, 1086
 ureteric, 963, 1061, 1090-1091, 1096, 1100
 Colitis, entero-, acute, 941
 ischæmic, 949
 ulcerative, 903-908, 1010
 Collar-stud abscess, 154, 163, 518, 529,
 530, 531
 diagnosis from branchial cyst, 518
 Colles's fascia, 1190
 fracture, 178, 205-207
 Colon, adenocarcinoma of, invading
 abdominal wall, 1030
 adenoma of, 917
 carcinoma of, 903, 905, 918-924, 1141
 dilatation in Hirschsprung's disease, 894,
 900
 diverticula of, 900. *See also* Diverticulosis
 familial adenomatous polyposis of,
 917-918, 1012
 redundant, 896
 volvulus of, 943-944
 Colostomy, blind transverse, 935
 for imperforate anus, 981
 for rectal stricture, 1011
 in carcinoma of rectum, 1020
 in diverticulitis, 902
 Coma, 358
 hepatic, 792
 myxœdema, 541
 Commando operation, 504
 Complement fixation tests, 1207, 1228
 in liver hydatids, 804
 Concentration dilution test of urine, 1062
 Concussion, 356-357
 Condylomata, 29
 anal, 29, 996
 Cone formation stage, in cerebral tumour,
 383, 384
 Conn's syndrome, 579, 581
 Contraceptive 'pill', carcinoma and, 612
Contre-coup, 357, 428
 Contusion and compression, cerebral,
 357-362
 of muscle, 305
 of spinal cord, 405-406
 pulmonary, 649
 Conus medullaris, tumours of, 416
 Cooley's anæmia, 785-786
 Coomb's test, 74-75, 781, 782
 Corns, 98, 173
 Coronary artery stenosis, 137
 Corpora amylacea, 1172
 Corrigan's pulse, 703
 Cortisol, 576
 Cortisones, action of, 576, 784-785
 adrenalectomy and, 581-582
 dangers of, 906
 excess alkalosis and, 95
 ovarian radiation and, 614
 therapy, 31, 576, 581-582, 784-785,
 906, 947
 Costo-clavicular syndrome, 139, 442-443
 Costo-transversectomy (Pott's), 242
 Countryman's lip, 49, 457
 Courvoisier's law, 836
 Cowperitis, 1182
 Coxa plana, 250-251
 valga, 215
 vara, 215, 337-338
 causes of, 249, 337-338
 Crafoord's operation, 695
 Craniectomy, for extradural hæmorrhage,
 365
 posterior fossa, 391-392
 Cranio-cerebral injuries, 354-368
 Cranio-cleidodysostosis, 252
 Craniopharyngioma, 395
 Cranioplasty, in oxycephaly, 353
 Craniotabes, 235
 in rickets, 244
 Craniotomy, technique of, 389-390
 Crepitus, 177, 178, 270
 Cretinism, 494, 540, 541-542
 Crohn's disease, 910-913, 965
 Cross-infection, 4
 Crush syndrome, 70, 74, 77, 80-81
 anuria in, 1068
 Crutch palsy, ætiology of, 443
 Crystalluria, sulphonamide, 1073
 Cubitus valgus and varus, 330
 'Cul de sac' syndrome, following
 gastrectomy, 764
 Cunningham's penile clamp, 1133
 Curlings' ulcer, 738
 Cushings' syndrome, 579-581
 basophil adenoma and, 394-395
 Cut throat, 525-526
 C.V.P. measurement of, 69
 Cyanotic congenital heart disease, 696-
 697
 Cylindroma, 106, 512
 Cystectomy, for bladder carcinoma, 1155
 Cysticercosis, 63
 Cystitis, 1138, 1142-1146
 cystica, 1146
 relief of pain, 449
 Cystodiathermy, 1153-1154

- Cystography, 1065
 excretory and retrograde, 1125, 1139, 1141
 in ruptured urethra, 1188
 in vesico-intestinal fistula, 1141
 Cystosarcoma phylloides, 601-602
 Cystoscopy, 1064, 1092, 1097, 1110-1111,
 1135, 1138-1139, 1141, 1145, 1147, 1152
 Cystostomy, for retention in spinal fractures
 1187
 for rupture of bulbous urethra, 1187
 Cysto-urethroscopy, 1105, 1165
 in carcinoma of prostate, 1175
 Cyst solitary lymph, of neck, 521
 Cysts, 61-64, 100-101
 choledochus, 824
 congenital, 61, 62
 dental root, 473-474
 dentigerous, 473-474
 emphysematous, 671, 672-673
 false, 61
 foregut, 685
 implantation, 62-63
 infected facial, 459
 local bone, 248-249
 mediastinal, 685
 naso-palatine, 474
 of bile ducts, 824
 of bones, 248-249
 of brain, 382, 385, 388, 390, 391
 of breast, 589, 598-599
 of Cowper's gland, 1182
 of epididymis, 1226-1227
 of kidney, 1075-1078
 of liver, 802-805
 of lung, 671-674
 of mesentery, 888-890
 of mesocolon, 890
 of mouth, 491-493
 of orbital margin, 481-482
 of pancreas, 858-859
 of parotid gland, 511
 of scalp, 352
 of semilunar cartilage, 278
 of seminal vesicles, 1182
 of spleen, 779
 over root of nose, 482
 parasitic, 63
 pseudo-mesenteric, 888
 retention, 62, 491, 859
 of gland of Montgomery, 589
 retroperitoneal, 890
 sebaceous, 100, 352, 1238
 serosanguineous, 888
 thymic, 574
 thyroglossal, 566-567
 Cytology, exfoliative, colonic, 921
 in carcinoma of œsophagus, 725
 pancreas, 864
 prostate, 1174-1175
 stomach, 768
 Cytotoxic drugs, 110, 478, 631-632

D

- Dacron net, in inguinal hernia, 1040, 1041
 Dactylitis, tuberculous, 237
 Darrow's solution, 89
 Deafness, congenital, 463
 Débridement, 16-17

- Decompression disease, 133
 Decortication, in empyema, 658-659
 Deformities, 320-350
 of leprosy, 25-27
 Dehiscence, abdominal, 1028-1029
 Dehydration, 78, 83
 therapy, 360, 405
 Delhi boil, 35
 Dental cyst, 473-474
 Dentate line, 976
 Dentigerous cyst, 473-474
 Depression, surgical treatment of, 400
 de Quervain's disease, 312, 565
 Dercum's disease, 54
 Dermatitis around umbilicus, 1024-1025
 artifacta ulcer due to, 37
 chronic, 149
 of sheep, contagious pustular, 34
 radio, 105
 Dermatofibroma, 102
 Dermatomycoses, 34
 Dermoid, 50, 62-63
 external angular, 481
 implantation, 62-63
 of mesentery, 888
 of scalp, 352
 over root of nose, 482
 post-anal, 981
 sequestration, 62
 sublingual, 492
 tubulo-, 62
 Desmoid fibroma, 53, 1030
 Dextran, 76-77, 111
 Dextrose solutions, 89
 Diabetes, arthropathies, neuropathic, of,
 carbuncles and, 99
 carcinoma of pancreas and, 864
 Dialysis, peritoneal, in anuria, 1071
 Diaphragm, 682-684
 eventration of, 682
 Diaphyseal aclasis, 253
 Dickie's method of inserting posterior
 nasal tampon, 480
 Dimple, post-anal, 982
 Dindevan, use of, 131, 136
 Disc, intervertebral, prolapse of, 134,
 421-424, 442
 Dislocations, 265-266
See also under names of joints
 Diverticula, acquired, 898
 congenital, 622, 898, 1137
 intestinal, 898-900
 jejunal, 899
 Meckelian, 899-900
 œsophageal, 713
 of appendix, 973
 of bladder, 1137-1140, 1171
 of large intestine, 900
 of seminal vesicle, 1181
 of small intestine, 898-900
 Diverticulectomy, 1139
 Diverticulitis, colonic, 900-903, 1141
 intestinal, 898
 Diverticulosis, 900
 alimentary, 898
 intestinal, 900-903
 intramural, of gall-bladder, 833
 jejunal, 899
 of appendix, 973

- Diverticulum, bladder, 1137-1140, 1171
 duodenal, 898-899
 jejunal, 899
 Diverticulum, Meckel's, 899-900
 œsophageal, 713
 of urethra, 1198, 1202
 pharyngeal, 622-625
 Donovan's manœuvre, 71
 Dormia basket, 1099
 Dragstedt's night juice test, 743-744
 Drug resistance, 1-2
 Drugs, stomatitis due to, 490
 Ductus arteriosus, patent, 692-693
 Dugas' test for shoulder dislocation, 268
 Duhamel's operation, 896
 Dukes's staging, 52, 1015
 Dumb-bell tumours, 652, 685
 'Dumping syndrome', 762
 Duodenal intubation, in pancreatic disease, 855
 ulcer, chronic, 738-750
 diagnosis from œsophagitis, 714-715
 perforated, 751, 874
See also Peptic ulcer.
 Duodenum and stomach, 728-772
 atresia and stenosis of, congenital, 936
 diverticula of, 736-737, 898
 neoplasms, of, 772
 occlusion of, congenital, 733
 rupture of, 735
 Dupuytren's contracture, 331
 Dwarfism, renal, 571
 Dyschondroplasia, 253
 Dysentery, amœbic and bacillary, 1001-1002
 Dyskinesia, biliary, 843
 Dysphagia, 709
 lusoria, 707, 711
 Dysplasia, fibrous, monostotic, 474
 polyostic, 248, 474
 Dyspnoea, urgent, 552

E

- Ear, afflictions of, 459-463
 drum, ruptured, 462
 Ectopia vesicæ, 1122-1123
 Eczema, diagnosis from Paget's disease
 of nipple, 589
 varicose, 149
 Elbow-joint, dislocations, 271
 fractures, 201-203, 271
 nerve injuries in, median, 185, 445-446
 ulnar, 446
 tuberculosis, 295
 Electroencephalography, 387
 Electrolyte balance, 84-88
 in anuria, 1069
 salt regulating hormones and, 576
 Elephantiasis, 151-153, 1207, 1238
 chirurgicæns, 608
 filarial, scrotal, 1238
 neuromatosa, 57
 Embolectomy, 121, 130, 131, 681, 949
 Embolism, 51, 129-133, 700
 pulmonary, 79, 130, 680-681
 Embolus, mesenteric, 130, 948-949
 Emphysema, mediastinal, 636
 Empyema, 655-659, 670, 679
 antri, 479
 Empyema --*contd.*
 decortication, 658-659
 diagnosis from subphrenic abscess, 875
 necessitatis, 651
 of gall-bladder, 832
 treatment of, 657-659
 tuberculous, 659
 Encephalocele, 353
 Encephalography, 375, 388
 Encephalopathy, porta-systemic, 792-793
 Enchondromata, multiple, 253, 255
 Endocarditis, 125, 693
 Endometrioma of bladder, 1148
 of umbilicus, 1027
 Endometriosis, diagnosis from rectal
 carcinoma, 1013
 of appendix, 974
 Endothelioma, 60
 of bursa, 319
 of meninges, 381
 Enteric fever, 2, 3
 Enteritis, staphylococcal, 922
 Enterocoele, 1032, 1034
 Enterocolitis, acute, diagnosis from
 intussusception, 941
 staphylococcal, 5, 1103
 Enterostomy, spur, for ileal atresia, 937
 Enuresis, in children, 1132-1133
 Ependymoma, 419
 Epicondyle, medial, dislocation of, 271
 Epididymectomy, for chronic epididymo-
 orchitis, 1230-1231
 Epididymis, cysts of, 1226-11227
 tumours of, 1237
 Epididymitis, 1169, 1179, 1227-1229
 acute, 1228
 chronic, 1229-1231
 gonococcal, acute, 1228
 post-operative, acute, 1229
 tuberculous, 1228, 1229
 Epididymo-orchitis, 1216, 1227-1231
 Epilepsy, 383-384
 post-traumatic, 375-376
 Epipharynx, 625
 Epiphysectomy, unilateral, in scoliosis, 326
 Epiphysiolysis, 338-339
 Epiphysis, enlargement of, in rickets, 243
 lower, of femur, separation, 221-222
 of radius, separation, 207-208
 of head, of femur, separation, 218
 slipped, in coxa vara, 338-339
 Epiphysitis, Calve's, 251
 syphilitic, 236
 Epiplocele, 1032
 Epispadias, 1186
 Epistaxis, 70, 373, 480-481
 Epithelioma, 100, 102, 107, 352
 squamous celled, 107, 457
 Epulis, varieties and treatment, 59, 470
 Erb-Duchenne paralysis, 441
 Ergot, gangrene from, 43
 Erysipelas, 1, 6-7
 Erysipeloid, 7
 Erythema induratum, 141
 Erythroblastosis foetalis et infantium, 806
Escherichia coli, 2
 Esmarch's bandage, 234, 278
 Ethmoid, sarcoma of, 478
 Euphoria *mist.*, 61

Eve's tumour, 474
 Ewing's tumour, 262
 Exanthemata, acute, and osteomyelitis, 232
 Exomphalos, 1051-1052
 Exophthalmos, cause of, 538
 of thyrotoxicosis, 543, 547-549
 operative treatment, 548-549
 producing substance, 538
 Exostoses, bony, of deep meatus, 460
 multiple, 253
 Exostosis, subungual, 257
 Extension-thrombosis, 130
 Extra-corporeal circulation, 690, 703
 Eye, injuries, 485

F

Face, congenital abnormalities, 451-458
 infections of, 458-459
 neoplasms, 459
 wounds, 458
 Facial palsy, 431, 513, 514
 Facies, adenoidal, 618
 Hippocratic, 870
 in tetanus, 8
 Facio-maxillary fractures, 194-197
 Fæcal impaction, 952
 Fæcolith, 956
 Fæces, water loss by, 82
 Fallor's tetralogy, 697-698
 Fanconi's syndrome, 244-245
 Fasciotomy, subcutaneous, 331
 Fat embolism, 133
 necrosis of breast, traumatic, 591
 Fat necroses, 850
 Felon, 164-165
 Felty's syndrome, 788
 Femoro-popliteal obstruction, 136
 Femoral ring, anatomical relations of, 1047
 Femur, fractures of, 177, 192-193, 214-222
 Fenestration with interstitial radium, 643
 Fiberscope, 745
 Fibrinopænia, 76
 Fibroids, recurrent, 60, 318
 Fibrolipoma, 53, 54
 Fibroma, 53
 of muscles, 317
 Fibromyoma, 53
 Fibrosarcoma, 49, 59-60
 of abdominal wall, 1030
 of bursa, 319
 of muscles, 317
 periosteal, 259-260
 Fibrosis, nodular subepidermal, 102
 Fibrositis, 321
 Fibula and tibia, fractures of, 224
 Filariasis, 34, 152, 1238
 hydroceles and chyloceles, 1225-1226
 Finger-print sweat test, 848
 Fingers, congenital deformities of, 331
 enchondromas, 253, 255
 fractures of, 212
 terminal pulp, infection of 164-165
 Finney's pyloroplasty, 733, 749
 Finsterer-Devine operation, 760
 Fish-handler's disease, 7
 Fissure-in-ano, 985-988
 Fistulæ, 20-21
 arterio-venous, 128-129, 398

Fistulæ—*contd.*

 biliary, 840-841
 bladder, 1140-1141
 branchial, 519-520
 broncho-pleural, 680
 duodenal, 735, 759
 fæcal, 924-925
 gastro-jejunal-colic, 760-761
 hepato-bronchial, 674
 in cavernous sinus, 398
 milk, 594
 of Cowper's gland, 1182
 oro-antral, 465
 pancreatic, 859-860
 parotid, 449, 510-511
 pharyngeal, 625, 629
 recto-vaginal, 906
 salivary, 510-511
 thyroglossal, 567
 tracheo-oesophageal, 710-711
 umbilical, 1025-1026
 uretero-vaginal, 1141
 urethral, 1199
 vesico-intestinal, 1141
 vesico-vaginal, 1140-1141
 Fistula-in-ano, 1006-1010
 Flail chest, 648
 Flat-foot, 340-343
 Fluid and electrolyte balance, 1070
 therapy, contraindications, 91
 in acute dilatation of stomach, 734
 in intestinal obstruction, 936
 in pancreatitis, acute, 853
 in ulcerative colitis, 906
 parenteral, 89-93
 strictly-limited regimen, 1070
 Focal syndromes, 383
 Foot and mouth disease, human, 490
 infections of, 172-175
 strain, acute, 341
 Foreign bodies, 20
 causing appendicitis, 956
 causing gas abscess, 46
 in air passages, 633
 in bladder, 1137
 in bronchial tree, 659-660
 in ear, 460
 in fistula or sinus, 20
 in larynx, 633
 in oesophagus, 711-712
 in rectum, 985
 in stomach, 733-734
 in tissues, 18-20
 intra-bronchial, 659-660
 'Four-flap' repair of cleft-palate, 454
 Fournier's gangrene, 1237
 Fovea coccygea, 982
 Fowler's position, criticism of, 871
 in treatment of peritonitis, 871-872
 Fractional test meal, in duodenal ulcer, 743
 Fractures, 176-229
 blisters, 179
 blood loss, measurement of, 68
 bumper, 223
 closed, 176
 comminuted, 176
 complicated, 177, 191
 complications, 184-191
 compound, 176, 191-192

Fractures—contd.

- deformity in, 177, 193–194
- delayed union, 187–188, 193, 211
- ecchymosis in, 179
- fatigue, 177
- first-aid treatment, 180–181
- functional loss, 178
- greenstick, 176, 178, 193–194, 197
- impacted, 176, 178
- infection of, 185–186, 189
- internal fixation, 192–193, 215, 221
- malunion in, 190–191
- march, 177, 229
- non-union, 188–190, 211, 216
- of childhood, 193–194
- open, 176
- operation, indications for, 191–192
- pathological, 177
- pressure sores, in 191
- reduction of, 181
- rehabilitation after, 182–184
- signs, 177–179
- simple, 176, 181–184
- spontaneous, 232, 247
- stave, 211
- tight plasters, 187
- traction, 202–203, 219–222
- union of, 179–180, 187–191
- varieties of, 176–177
- See also under names of bones*
- Fragilitas ossium, 249
- Fragility test, 781
- Framboesia, 31
- Frei test, 1207
- Freiberg's disease, 251–252
- Freyer's prostatectomy, 1167
- Frey's syndrome, 514
- 'Frog-faced man', 249
- 'Frog hand', 167
- Fröhlich's syndrome, causes of, 395
- Froin syndrome, 396, 418
- Froment's sign for ulnar paresthesia, 447
- Frontal lobe tumours, 384
- Frostbite, 47
- 'Frozen shoulder', 295, 309–310
- Funnel chest, 650
- Furuncle, 99
 - of ear, 460
 - of face, 458

G

- Galactocoele, 599
- Gall bladder and bile ducts, 818–845
 - abnormalities of, congenital, 822–825
 - biliary stasis, 826
 - carcinoma of, 844–845
 - complications, in typhoid fever, 830, 832
 - disease, cardiac disorders and, 836
 - inflammation of, chronic, 832
 - injuries, 825
 - investigations, 819–822
 - mucocoele of, 829
 - neoplasms of, 844
 - perforation of, 830
 - strawberry, 833
 - torsion of, 825
 - typhoid, 832
- Gallie's graft, 1041
- Gallow's splint, use of, in fractured femur, 221
- Gall-stones, 825–829
 - causing intestinal obstruction, 947
 - chronic pancreatitis, in relation to, 855
 - colic, 828–829
 - fistulae caused by, 840–841
 - impaction of, 830
 - in bile ducts, 827, 836–840
 - silent, 827–828
- Ganglion, simple, 314
 - compound palmar, 312
- Ganglioneuroma, 56, 584
- Gangrene, 7, 38–48, 118, 119, 124, 130, 134, 136, 137, 138, 927, 942, 957, 960, 991, 993, 1033–1034
 - atherosclerotic, 39
 - chemical, 48
 - diabetic, 41, 43–44
 - dry, 40
 - embolic, 40, 948
 - ergot, 43, 78
 - gas, 3, 5, 44–46, 78, 186
 - in atherosclerotic obstruction, 134, 136
 - in thrombo-angiitis obliterans, 137, 138
 - infective, 44–45
 - moist, 40
 - progressive post-operative, 1029–1030
 - symptomatic, 39
 - traumatic, 39, 46–47
 - varieties, 39, 40
 - venous, 48
- Gas abscess, 46
- Gastrectomy, anaemia following, 764
 - for carcinoma of stomach, 770–771
 - partial, 747–748
 - complications following, 758, 852
 - for hour-glass stomach, 757
 - for peptic ulcer, 747–748, 752, 756
 - post, syndrome, 761–764
 - radical and total, 770–771
- Gastric aspiration, 752–753, 755
 - drainage procedures, 749–750
 - juice secretion, 728, 758, 871
 - mucosa, atrophy of, 729
 - polyposis of, 765
 - prolapsing, 737
 - operations, complications of, 758–764
 - tetany, 735
 - ulcer, chronic, 738, 739, 740–741, 746
 - See also Peptic ulcer*
- Gastritis, 736
- Gastro-enterostomy, complications, 760, 761
- Gastro-intestinal suction drainage for obstruction, 935–936, 979
- Gastro-jejuno-colic fistula, 760
- Gastro-jejunostomy, complications and, 750, 760
 - technique of, 749
 - for duodenal ulcer, 750
 - pyloric carcinoma, 771
 - stenosis, 757
 - vagotomy with, 748
- Gastro-photography, 745
- Gastroscopy, 744–745, 758, 768, 777
- Gaucher's disease, 788
- Gee's disease, 245

- Genu recurvatum, 340
 valgum, 339-340
 varum, 340
 Gigantism, 254, 394
 Gingivitis, 468-469
 Girdlestone's operation, 281
 Glandular fever, 155
 Glass splinters in tissues, localisation of, 19
 Glioblastoma multiforme (astrocytoma grade 4), 382, 388
 Gliomas, 382, 385, 388, 389, 482
 excision of, 390-391
 Glomangioma, 57
 Glomus tumour, 57, 462
 Glossectomy, 503
 Glossitis, 495, 496, 498
 Glossodynia, 500
 Glottis, acute œdema of, 633-634
 Glucocorticoids, 576
 Glucose tolerance test, in pancreatic disease, 856
 Goitre, 542-559
 adenoparenchymatous, 553-555
 carcinoma secondary to, 555, 561-562
 classification of, 542
 colloid, 553
 dyshomogenetic, 541
 endemic, 553-555
 exophthalmic, 543
 in children, 554-555
 intrathoracic, 557
 lymphadenoid, 564-565
 multinodular, 553-555
 carcinoma secondary to, 555, 562
 nodular, 553-557
 solitary, 555-557
 of puberty, 553
 plunging, 557
 pre-tibial, 541
 retrosternal, 557-559, 684
 substernal, 557
 thiouracil, 545
 toxic, primary, 543
 'Golf-hole' ureteric orifice, 1111
 Gonorrhœa, 3, 31-33, 1002
 Goodsall's rule, 1007
 Gout, 303-304
 Graham-Cole test, 820
 Gram negative shock, 78
 Granuloma, amœbic, 1002, 1017
 eosinophilic, 253
 giant celled reparative, 475
 inguinale, 1208-1209
 intubation, 639
 pyogenicum, 102
 umbilical, 1024
 venereum, 1208-1209
 Gravel in tissues, removal of, 19
 Grave's disease, 543
 Grawitz's tumour of kidney, 1115-1118
 Gregerson's benzidine test, 743
 Guest cannula, 90
 Gumma, 29, 37, 456
 endosteal, 236
 of breast, 595
 of lymph nodes, 155
 of muscle, 317
 of testis, 1231
 of tongue, 38, 498
 Gumma—*contd.*
 periosteal, 236
 synovial, local, 282-283
 Gummatous ulcer, 29, 37
 Gums, diseases of, 468-470
 'Gun-stock' deformity, 330
 Guttman's rehabilitation of paraplegics, 410
 Gynæcomazia, 615, 1176
- ## H
- Hæmangioma, 103-104
 of large intestine, 918
 of lip, 457
 of liver, 815
 of mouth, 494
 of neck, 532
 of rectum, 1013
 of tongue, 493, 494
 Hæmarthrosis, 76
 Hæmatemesis, in acute peptic ulcer, 738, 754
 in chronic peptic ulcer, 741, 753-754
 in portal hypertension, 809
 post-operative, 758, 777
 severe, causes and treatment of, 753-756
 Hæmatocele of testis, 1226
 Hæmatocrit, 68
 Hæmatoma, 63, 230
 of breast, 591
 of scalp, 351, 369
 perianal, 995
 perinephric, 1082
 pulsating, 124
 subdural, 365, 366
 tearing of epigastric artery causing, 1027
 Hæmatomyelia, 403, 404, 406
 Hæmatorachis, 403
 Hæmaturia, causes of, 130, 1061-1062, 1076, 1081-1082, 1091, 1096, 1109-1110, 1134, 1143, 1164
 delayed, 1081
 significance of, 1081, 1114
 Hæmochromatosis, 806
 Hæmoglobin level, 67-68
 Hæmo-lymphangioma, 104
 Hæmophilia, 76, 124, 494
 Hæmophilus ducreyi, 31
 influenzæ, 2
 Hæmorrhage, 65-72
 after gastric operations, 758
 after prostatectomy, 1169
 after thyroidectomy, 552
 after tonsillectomy, 619
 after tooth extraction, 464-465
 cerebral, 355, 362-368
 complicating fractures, 185
 complicating ulcerative colitis, 906
 complicating varicose veins, 71, 150
 extradural, 363-366, 403
 from hæmorrhoids, 991, 993
 from Meckel's diverticulum, 899
 from œsophageal varices, 810-811
 from ruptured varicose vein, 71, 150
 in spinal cord injuries, 403, 406
 intracranial, 355, 362, 399
 intramedullary, 404
 intraspinal, traumatic, 403
 intraventricular, 362

- Hæmorrhage—*contd.***
 meningeal, 361
 subarachnoid, 399
 subcortical, 362-363
 subdural, 366-368
 urethral, 1187-1188
- Hæmorrhoidectomy, 993-995**
- Hæmorrhoids, 809, 906, 989-996**
 sentinal, 987
 thrombotic, 991, 993, 995
 Hæmothorax, 648-649, 653-654
- Hair-ball of stomach, 734**
- Hallux rigidus, 347**
 valgus, 346-347
- Hamartoma, 57, 103, 663**
- Hamilton's ruler test, 268**
- Hammer-toe, 346, 347-348**
- Hand, infections of, 1, 160-172**
 proximal volar space, 166
 pulp space, terminal, 164-165
 'obstetrician's', 569
 paralysis of small muscles, 447
 'simian', 446
- Hansen's disease, 24**
- Harrington's repair of para-œsophageal hernia, 719**
- Harris's lines, 254**
- Harrison's sulcus, 243**
- Hartmann's operation, 1020**
 pouch, 818
- Harvey's sign, 119-120**
- Hashimoto's disease, 564-565**
- Hayward's treatment of breast cancer, 614**
- Head injuries, 354-376**
 closed, treatment of, 358-361
 due to missiles, 372
 late effects of, 374-375
- Heart and pericardium, 686-707**
 block, 703
 cholecystic, 836
 congenital disease of, 692-698
 diseased gall-bladder and, 836
 disorders of rhythm, 703
 failure, 693, 700, 881
See also Cardiac arrest
 fluid therapy and, 91
 left, by-pass, 691
 lung machines, 690
 massage of, 688-689
 special methods of investigation, 686-687
 tamponade, 691
 tumours of, 703
- Heinke-Miculicz pyloroplasty, 749**
- Heller's operation for cardiospasm, 722**
- Hemi-colectomy, 922, 934**
- Hemi-hepatectomy, 817**
- Henoch's purpura, 941**
- Heparin, 131**
- Hepatico-jejunostomy, 842**
- Hepatitis, amœbic, 798**
 infective, 796
 serum, 76, 796
 virus, 806
- Hepato-adenomata, 815**
- Hepato-lenticular degeneration, 808**
- Hepato-splenomegaly, endemic, 786-788**
- Hernia, 1031-1060**
 cerebri, 377
 diaphragmatic, 682-684
- Hernia—*contd.***
 divarication of recti abdominis, 1057-1058
 epigastric, 1056-1057
 fatty, of linea alba, 1056-1057
 femoral, 1047-1051
 differential diagnosis of, 1048-1049
 strangulated, 1049, 1051
 gluteal, 1060
 hiatus, œsophageal, 682, 715-720
 incarcerated, 1032, 1033
 incisional, 1054-1056
 inflamed, 1035
 inguinal, 1035-1045, 1048
 recurrent, 1045
 strangulated, 1043-1044
 treatment of, 1038, 1045
 internal, 946
 interparietal, 1058
 interstitial, 1058-1059
 in-W, 1044
 irreducible, 1033
 lumbar, 1059
 obstructed, 1033
 obturator, 1059
 of bladder, 1140
 of hydrocele, 1224
 pantaloons, 1043
 para-œsophageal, 719
 para-umbilical, 1053
 perineal, 1059
 phantom, 1059
 post-operative, 683-684, 1054-1056, 1059
 prevesical, 1043
 reducible, 1032
 saddle-bag, 1043
 sciatic, 1060
 scrotal, 1036
 sliding, 715, 1046-1047
 strangulated, 1033-1035, 1043-1045, 1054
 through foramen of Bochdalek, 683
 through foramen of Morgagni, 682-683
 through œsophageal hiatus, 682, 715
 through pelvic floor, 1059
 truss for, 1038, 1050
 umbilical, 1051-1056
 ventral, 1054-1056
- Hernie-en-glissade, 1046-1047**
- Hernioplasty, 1039**
- Herniorrhaphy, for indirect inguinal hernia, 1039**
 for umbilical hernia, 1052
 hiatal, 718
 hydrocele after, 1225
- Herniotomy, inguinal, for indirect hernia, 1038-1039**
 for strangulation, 1044
- Herpes, penile, 1206**
 of tongue, 495
- Hesselbach's triangle, 1042**
- Hetrazan, 34**
- Hewitt's airway, 495**
- Hey's prostatectomy, 1168**
- Hiccough, persistent, 777**
- Higgs' 'spike' operation for hammer toe, 348**
- Highmore, antrum of, infection of, 478-479**
- Hilton's method of treating abscess, 14**

- Hip-joint, congenital dislocation of 332-337
 dislocation of, 273-274
 snapping, 303
 tuberculosis, 292-294
 Hippocratic facies, 870
 method of traction, 269
 Hirschsprung's disease, 83, 893-896
 Hodgkin's disease, 156-157, 575, 789
 Hofmeister-Finsterer valve for peptic ulcer, 759
 Hollander's test, 744
 Homan's sign, 143
 Homografts, 121
 Honeycomb lung, 653
 Hordeolum, 99, 481
 Hormone, dependent carcinoma, 612
 for prostatic carcinoma, 1176-1177
 therapy, of breast cancer, 613, 614
 of male infertility, 1239
 Hormones, adrenocortical, 576
 Horner's syndrome, 128, 442
 Horse-shoe kidney, 1075
 Hospital infection, 1, 4-6
 Hot-cross bun head, 236
 Housemaid's knee, 318
 Houston's valves, 976
 Humerus, fracture-dislocation of, 200
 fractures of, 45, 199-204, 444
 Hunner's ulcer of bladder, 1144
 Hunterian chancre, 28
 of penis, 1206
 ligation for aneurysm, 126
 Hürthle-cell adenoma, 557
 Hutchinson's teeth, 30, 236, 464
 wart, 498
 Hutchison's neuroblastoma of adrenal medulla, 584
 Hyaluronidase, 94
 Hydatids, 63, 64
 of kidney, 1078
 of liver, 802-805
 of lung, 673-674
 of mesentery, 888
 of pancreas, 859
 of spleen, 780
 Hydrocele, 1222-1226
 congenital, 1224
 en bisac, 1224
 encysted, of cord, 1224
 infantile, 1223
 of hernial sac, 1049, 1224, 1225
 vaginal, 1223
 Hydrocephalus, 285, 388, 396-397
 Hydroencephalocœle, 353
 Hydronephrosis, 1084-1088, 1138
 aetiology, 1078, 1084, 1086, 1112
 diagnosis form retro-peritoneal cyst, 890
 Hygroma, cystic, 153, 520-521
 Hypercorticism, 578-582
 Hyperhidrosis, 449
 Hyperinsulinism, 860-861
 Hypernephroma, 354, 1115-1118
 Hyperparathyroidism, 570-572
 in aetiology of renal calculus, 570, 1089
 Hypersplenism, 789, 790
 Hypertension, congenital cystic kidneys
 and, 1076-1077
 in pyelonephritis, 1103
 in renal ischæmia, 1105
 Hypertension—*contd.*
 phæochromocytoma and, 585
 portal. *See* Portal hypertension, 808
 Hyperthyroidism, 543-550
 in children, 547
 Hypocorticism, 577-578
 Hypodermoclysis, 94
 Hypokalaemia, 87, 950
 Hypoparathyroidism, 568-570
 Hypophysectomy, for carcinoma of breast, 614
 Hyponatraemia, 85
 Hypoproteinaemia, 93, 899
 Hypospadias, 1184-1186
 Hypotassaemia, 950
 Hypothermia, in brain stem lesions, 360
 in cardiac surgery, 690-691
 in cytotoxic drug infusion, 632
 in giving blood, 75
 Hypothyroidism, 540-542, 548
 after radioiodine therapy, 547
 serum cholesterol test, 540
 Hysteria, joint complications, 285, 320
 torticollis in, 321

I

- ¹³¹I, tracer dose in diagnosis, 539
 treatment, 545-547
 Ileal loop conduit, 1159
 Ileitis, regional, 910-913, 961
 Ileocystoplasty, 1145
 Ileo-entrectomy, 882
 Ileostomy, for ulcerative colitis, 907-908
 Ileo-ureterostomy, 1133, 1159
 Ileum, atresia or stenosis of, congenital, 937
 tuberculosis of, 913
 Ileus, duodenal, chronic, 737
 gall-stone, 947
 meconium, 938
 paralytic, 777, 872, 949-951
 Iliac vein, compression and obstruction, 143
 Immunity, thymus and, 572
 Impalement of rectum, 984-985
 Impetigo, 98-99
 Implantation dermoid (cyst), 62-63
 Impotence, 1181, 1238-1239
 in prostatic enlargement, 1163
 Incontinence, anal, 1000-1001
 stress, 1131
 Induratio-penis-plastica, 1209
 Infantile paralysis, 350. *See also*
 Poliomyelitis
 Infertility, male, 1181, 1238-1239
 Infusion, continuous subcutaneous and
 rectal, 94
 Insect bites, 18
 Insulinoma, 860-861
 Insulin test meal, in peptic ulcer, 744
 Intermittent claudication, 117-118, 123,
 134, 137
 Intervertebral disc, prolapse of, 134, 421-
 424, 442
 Intestinal obstruction, 926-952, 961
 acute, 872, 926, 928-939
 in Hirschsprung's disease, 894
 of newborn, 936-939
 recurrent attacks, 951
 acute-on-chronic, 926, 930, 934

Intestinal obstruction—*contd.*

- adynamic, 926, 949–951
 - chronic, 926, 929–930, 951–952
 - closed-loop, 928
 - due to adhesions, 944–945
 - due to antibiotics, 971
 - due to bands, 900, 945–946
 - due to bolus, 764, 947
 - due to congenital atresia and stenosis in newborn, 936–937
 - due to faecal impaction, 952
 - due to gall-stones, 947
 - due to gastrectomy, 764
 - due to ingestion of dried fruit, 947
 - due to internal herniae, 946
 - due to mesenteric embolus and thrombosis, 948–949
 - due to stercolith, 948
 - due to stangulation, 927–928, 930, 933–934
 - due to strictures, 946–947
 - due to tuberculosis, 913
 - of mesenteric lymph nodes, 888
 - due to volvulus, 943–944
 - due to worms, 948
 - dynamic, 926
 - gastro-intestinal suction drainage, 935–936, 944
 - non-strangulating, 930
 - strangulating, 927–928, 930, 933–934
 - toxæmia, in, 927, 928
- Intestines, 892–925
- adenoma, of, 915, 916–917
 - adenomatous polypi of, 917–918
 - antibiotic therapy and, 1103
 - bacteria in, 2
 - blind loop of, 897–898
 - carcinoma of, 905, 916, 918–924, 934
 - congenital malformations of, 893
 - diverticula of, 898–903. *See also* Diverticulitis and Diverticulosis
 - fluid levels, 931–932
 - gas cysts of wall, 915
 - hæmangioma of, 918
 - inflammation of, 901–903
 - large, 893
 - diverticula of, 900
 - tumours of, 916–918, 934
 - lipomas of, 915
 - lympho-sarcoma of, 916
 - Meckel's diverticulum of, 899–900
 - obstruction of, 926. *See also* Intestinal obstruction
 - polyposis of, 915, 917–918
 - pseudo-polyposis of, 905
 - rupture, of, traumatic, 896–897
 - sarcoma of, 916
 - small, 761, 892, 893, 946
 - diverticula of, 898–900
 - obstruction of, sodium depletion and, 85
 - obturation and, 947–948
 - stricture of, 946–947
 - tumours of, 915–916
 - volvulus of, 943
 - strictures of, 905
 - tuberculosis of, 913–914
 - volvulus of, 943–944
- Intracardiac operations, closed, 690

- Intracranial pressure, 358, 383–384
 - tumours, 381–396
- Intragastric drip, transnasal, 94
- Intramedullary tumours, 415
- Intravenous infusions, 75
 - potassium, 88
 - pyelography, 1062–1063
- Intussusception, 939–943
 - ileo-cæcal, 999
 - jeuno-gastric, 759
 - Meckel's diverticulum and, 899
 - recto-sigmoid, 999
 - recurrent, 943
- Iodine, chemical protein-bound, test, 539
 - in goitre, 553–554
 - radioactive, 545–547
 - in investigation of thyroid function, 538–539
 - in thyroid carcinoma, 563
 - in thyrotoxicosis, 545, 549
 - therapy, 545–547
- Iron-deficiency anæmia, 489–490
- Irradiation, necrosis and, 472
- Ischemic contracture, 320
 - in fractures, 186
- Isotopes, radioactive, scanning of, for lesions of liver, 793
- I-triiodothyronine resin uptake test, 539–540
- I.V.P., 1062

J

- Jaboulay's operation for hydrocele, 1225
- Jacking systems, internal, 326
- Jacksonian epilepsy, 375, 384
- Jaundice, 823, 829, 836, 837, 858
 - acholuric familial, 780
 - in carcinoma of pancreas, 862, 863
 - in newborn, 1024
 - in pancreatitis, 851, 854, 855
 - syringe, 796
 - with cholelithiasis, treatment of, 838
- Jaws and face, 470–485
 - fractures of, 196–197
 - bone tumours of, 474–475
 - clicking, 303
 - lower, dislocation of, 266–267
 - multilocular cystic disease of, 474
 - necrosis of, 472
 - osteomyelitis of, 471–472
- Jejunum, diverticulum of, 899
- stenosis of, atresia of, congenital, 937
- Jod-Basedow thyrotoxicosis, 550
- Joints, acute suppurative arthritis, 279–281
 - barometric, 299
 - clicking, 303
 - deformities, 320
 - effusions in, painless, 282
 - flail, arthrodesis in, 350
 - gonococcal, 282
 - hæmophilic, 76
 - in hysteria, 285
 - in syringomyelia, 284
 - injuries to, 265–278
 - loose bodies in, 301–302
 - mimetic, 285
 - neuropathic, 265
 - osteoarthritis, 297–302

Joints—*contd.*

- penetrating wounds of, 281–282
- snapping, 303
- stiffness after fracture, 191, 193
- syphilitic, 282–283
- tuberculous, 285–295

K

- Kahler's disease, 259
- Kahn test, 27
- Kanavel's sign, 169
- Kantor's 'string sign', 912
- Kaposi's pigmented sarcoma, 110
- Kay's augmented histamine test, 743
- 'Keel' operation for incisional hernia, 1055
- Keetley-Torek's operation, 1217
- Kehr's sign in ruptured spleen, 776
- Keller's operation, 347
- Keloid, 53, 116, 159
- Keratin horn, 102
- Keratitis, interstitial, 30–31
 - neuropathic, 430
- Kerato-acanthoma, 102, 457
- Keratosis, senile (or solar), 105
- Kernohan's classification of astrocytic gliomas, 382
- Kidneys, 1074
 - aberrant renal vessels, 1078
 - abnormalities of, congenital, 1074–1079
 - angiography of, 1066
 - artificial, 81, 1071, 1105
 - calculus, 1088. *See* Calculus, renal
 - carbuncle of, 1106–1107
 - carcinoma of, 1118–1119
 - secondary deposits in bones, 263
 - congenital cystic, 1075–1077
 - crossed dystrophia, 1075
 - cyst of, solitary, 1077
 - denervation, 1088
 - dialysis, recurrent, 1105
 - ectopic, 1074
 - embolism of, 130
 - exposure of, 1119
 - function, poor, fluid therapy and, 91
 - tests, 1062
 - hydatid cyst of, 1078
 - infections of, 1088, 1099–1105
 - injuries of, 1081–1083
 - ischæmia of, hypertension in, 1105
 - neoplasms of, 1113–1120
 - operative exposure of, 1119–1120
 - pelvis, duplication of, 1078–1079
 - papilloma of, 1118
 - polycystic, 1075–1077
 - transplantation of, 1105, 1120
 - tuberculosis of, 1108–1113
 - unilateral fusion, 1075
- Kienböck's disease, 252
- Killian's dehiscence, in pharyngeal pouch, 622
- Kinmonth and Taylors' lymphangiography, 152
- Kirschner's wire, 18, 504
- 'Kiss cancer', 51, 1149
- 'Kite' method of correction in talipes, 345
- Klinefelter's syndrome, gynæcomazia in, 615
- Klumpké's paralysis, 441–442

- Knee-joint, dislocation of, 274–275
- internal derangement of, 275–278
- snapping, 303
- tuberculosis of, 294
- Knight's restricted orbital undercutting, 400
- stereotactic tractotomy, 400
- Knock-knee, 339
- Kocher's incision, 834
 - manœuvre, 817
 - method for shoulder dislocation, 268–269
- Köhler's disease, 251
- Koilonychia, 720
- Kondoleon's operation, 153
- Krukenberg's tumour, 52, 766
- Kümmel's disease, 403
- Küntschner intramedullary nail, 193
- Kveim's test, 159
- Kypho-scoliosis, 322, 650
- Kyphosis, 326–328
 - in acromegaly, 254
 - in osteitis deformans, 247
 - in Paget's disease, 328
 - in Pott's disease, 240, 327
 - in rickets, 244
 - in Scheuermann's disease, 327
 - senile, 328
- Lachrymal gland, mixed tumour of, 482
 - sac, mucocele of, 482
- Laminectomy, 411, 419–420
- Langer's lines, 165, 518, 520, 530, 599
- Larag, 776
- Laryngectomy, total, for carcinoma, 642–643
- Laryngoceles, 639–640
- Laryngo-fissure, 643
- Laryngopharynx, 625
 - neoplasms of, 629–631
- Larynx, affections of, 633–643
 - carcinoma of, 641–643
 - foreign body in, 633
 - neoplasms, 640
 - obstruction of, 634
 - papilloma of, 640
 - stenosis, 526
 - syphilis of, 639
 - tuberculosis, 638–639
- Laugier's femoral hernia, 1049
- Lead poisoning, teeth in, 468
- Le Fort's classification of fractures of maxilla, 194–195
- Lentigo, 107
- Leontiasis ossea, 249, 475
- Leprosy, 24–27, 1231
 - gynæcomazia and, 615
 - of toes, 27, 174
- Leriche's syndrome, 134, 136, 706
- Letterer-Siwe disease, 253
- Leucotomy, 400
- Leukæmia, lymphatic, 155, 789
- Leukoplakia of penis, 1210
 - of tongue, 496–497
 - superficial glossitis and, 496–497
- Ligation and sieve ('lattice') operations, 143
 - for varicose veins, 148
 - subfascial, for venous ulcer, 150
- Linea alba, fatty hernia of, 1056–1057

- Linitis plastica, 768-769
- Lip, 451-455
 carcinoma of, 37, 57, 457-458
 chancre of, 28, 456, 457
 cracked, 456
 pigmented, buccal mucous membrane and, 456
 tumours, ectopic salivary, 457
- Lipoma, 53-55
 arborescens, 55, 298
 in sucking—pad of infant, 493
 of cord, 1037
 of intestine, 915
 of neck, 532
 of scalp, 352
 of tongue, 493, 500
 retroperitoneal, 891
- Lipo-sarcoma, 54
 retroperitoneal, 891
- Lister's metal bougies, 1195
- Litholapaxy, 1135-1136, 1200
- Lithotomy, suprapubic, 1136
- Little's area, 480
 disease, 265, 320
- Littre's hernia, 900, 1032
- Liver, 791-817
 abscess of, 797-801
 actinomycosis of, 801
 biopsy, 794
 in portal cirrhosis, 808
 carcinoid, secondary, 816
 carcinoma of, primary, 815-816
 secondary, 816-817
 cholangioma, 815
 cirrhosis of, 805-808, 881
 cysts of, 802-805
 enlargements of, table of 795-796
 failure, 792
 'foaming', 45
 function tests, 793
 hepatoma of, 815
 'hob-nail', 806
 hydatid disease of, 802-805
 melanoma of, secondary, 816
 neoplasms of, 815-817
 rupture of, 794-795
 sarcoma of, secondary, 816
 syphilis, 802
 tuberculosis, 801-802
 tumour of, resection of, 816-817
 wounds of, 795
- Lobectomy, for bronchiectasis, 662
 for gliomata, 390
 for lung abscess, 671
 cancer, 667
- Lockhart-Mummery perineal operation for carcinoma of rectum, 1020
- Lockwood, 'gubernacular tails' of, 1218
 operation, 1050
- Long-acting thyroid stimulator, 538
- Lordosis, 328-329
- Lotheisen's operation, 1050-1051
- Lowsley's operation, 1123, 1159
- Ludwig's angina, 15, 467, 495, 527
- Lumbar disc protrusion, 422-424
 paravertebral injection, 122
 puncture, 425-427
 in brain injuries, 359-360, 367
 in brain tumours, 384, 387
 s.p.s.—80
- Lumbar disc protrusion—*contd.*
 in intracerebral abscess, 379
 in meningitis, 376
 in spinal canal tumours, 417
- Lungs, abscess of, 669-671
 and bronchi, tumours of, 662-669
 carcinoma of, 57, 664-669
 collapse therapy, 678
 complications, post-operative, 678-681, 777
 cysts, 671-674
 embolism of, 79, 130, 680-681
 hydatids of, 673-674
 laceration of, 649
 radiotherapy, 668-669
 complications, 1283
 resection, for tuberculosis, 677-678
 tumours of, 662-669
- Lupus vulgaris, 100
- Luschka, crypts of, 818
- Luxatio erecta, 268
- Lymphadenitis, 153-155
 cervical, 528-530
 iliac, suppurative, 965
 mesenteric, acute non-specific, 885-886, 961
 tuberculous, 154-155, 886-888
 treatment of, 530-531
- Lymphadenoma, 156-157, 575
 and leukaemia, 155
- Lymphangiography, 151-152, 1236
- Lymphangioma, 153, 457, 494
- Lymphangiosarcoma, 608
- Lymphangitis, 151, 160, 161
- Lymphatics, 151-153
 inguinal, enlarged, 1207
 of arm, 161
 of breast, 587-588
 of gall-bladder, 819
 of rectum and anal canal, 977-978
 of stomach, 769-770
 of testis, 1231
 of thyroid, 537
 of tongue, 502
 of umbilicus, 1023
 tuberculosis of, 154, 528-531
- Lymph nodes, 38, 153-155, 1049
 excision of, 110
 tuberculous mesenteric, 886-888
- Lymphoedema, 151-153
- Lympho-granuloma inguinale, 1002, 1046, 1206-1208
 venereum, 1206, 1229
- Lymphoma, malignant, of Africa, 158
- Lymphosarcoma, 60, 620, 772

M

- McBurney's point, 953
- McCarthy's resectoscope, 1168
- McEvedy's high operation for femoral hernia, 1050
- MacMurray's osteotomy, 300
- Macrocheilia, 153, 456, 457
- Macroductyly, 332
- Macroglossia, 153, 494
- Madelung's deformity, 232, 330
- Madura foot, 174
- Maingot's operation for inguinal hernia, 1040

- Malgaigne's bulgings, 1042
 Mallet finger, 306, 307-308
 Mallory-Weiss syndrome, 754, 755, 756
 Mandelic acid, in urinary infections, 1104
 Mandible, adamantinoma of, 474, 475
 dislocation of, 266-267
 fractures of, 196-197
 giant-celled reparative granuloma of, 475
 neoplasms, malignant, of, 478
 osteomyelitis of, 471-472
 Mannitol, 77, 81
 Manus valga, 232, 330
 Marble bones, 253
 Marie-Strümpell, spondylose rhizolelique, 296-297
 Marion's disease, 1170-1171, 1184
 sign, 1165
 Marjolin's ulcer, 49, 105
 Marsupialisation of mesenteric cyst, 890
 with partial excision in ranula, 492
 Martin's pump, 75
 Massage, cardiac, 688, 689
 Mastectomy, 609-611
 Mastitis, 591-594
 carcinomatosa, 603-604
 from local irritation, 591, 615
 of puberty, 591
 Mastoiditis, acute, 461
 Matas's operation for aneurysm, 127, 705
 Mathé's sign, 1108
 Maxilla, carcinoma of, 476
 excision of, 477-478
 fractures of, 194-195
 neoplasms of, malignant, 476-478
 osteomyelitis of, 471
 Maydl's hernia, 1044
 Mayo's operation for umbilical hernia, 1054
 Meatotomy, ureteric, 1079, 1099
 Meatus, pin-hole, 1183
 urethral, ulceration of, 1191
 Meckel's diverticulum, 899-900, 1026
 in ætiology of intussusception, 899, 939
 Meconium ileus, 938
 Mediastinum, tumours of, 684-685
 Mediterranean anæmia, 785-786
 Medulloblastoma, 383
 Megacolon, acquired, 896
 congenital aganglionic, 893-896
 primary, 893-896
 secondary, 896
 Megalo-ureter, congenital, 1080
 Meibomian cyst, 481
 Melanoma, 352, 1022
 amelanotic, 109
 intraocular, 483-484
 malignant, 51, 108-110, 352
 Meleney, chronic undermining ulcer of, 164
 'Melon-seed' bodies, in joints, 286, 301, 312
 Ménière's syndrome, 432, 463
 Meningioma, 381-382, 383, 385, 386, 388, 389, 390, 414-415, 419
 Meningitis, infective, acute, 375-376
 in spinal injuries, 411
 Meningocele, 353, 411
 Meningo-myoele, 411-412
 Mental disorders, surgical treatment of, 400
 Meralgia paræsthetica, 448
 Mercury weighted tube, in gastro-intestinal drainage, 935-936
 Mermaid splint for knock-knee, 340
 Mesenteric artery stenosis, 137
 embolism, 130, 948-949
 embolus and thrombosis, causing intestinal obstruction, 948-949
 Mesenteric nodes, 886-888
 Mesentery, lesions of, 884-890
 wounds of, 884-885, 897
 Mesopharynx, 625
 Mesothelioma, 662, 1237
 Metacarpals, fractures of, 211
 Meteorism, 1081-1082
 Meulengracht diet, 756
 Michel's clips, use of, in localisation of foreign bodies in tissues, 18
 Microcephaly, 353
 Microcurie, 539
 Microdochectomy, 600
 Micrognathia, 455
 Microliths, 1089
 Mikulicz's disease, 487, 514-515
 Milli-equivalent, (mEq), 84
 Millicurie, 539
 Millin's operation for incontinence, 1133
 retropubic prostatectomy, 1168, 1177
 Milroy's disease, 152
 Milwaukee brace, 326
 Miner's elbow, 318
 Minerva collar, 407
 Mitral regurgitation, 702
 stenosis, 699-702
 Moles, pigmented and hairy, 57
 Molluscum fibrosum, 102
 sebaceum, 102, 457
 Moloney's operation for inguinal hernia, 1040
 Mondor's disease, 595-596
 Moniliaiasis, 12
 Mononucleosis, infective, 155
 Monteggia's fracture, 208, 272
 Montgomery, gland of, retention cyst of, 589
 Moon's turret teeth, 30, 236, 464
 Moore prosthesis, 216
 Morgagni, columns of, 976
 crypts of, 976
 foramen of, hernia through, 682-683
 hydatid of, cyst of, 1226
 Morphine provocation test, in pancreatic disease, 856
 Mouth and tongue, 486-506
 carcinoma of, 493
 to mouth ventilation, 688
 Mucoviscidosis, 847, 939
 Mule-spinner's cancer, 1239
 Mumps, acute epididymo-orchitis of, 1228
 mastitis of, 591
 Murphy's sign, in gallstones, 828, 961
 Muscles, arm, innervation of, 441
 hernia of, 308
 inflammation of, 316-317
 injuries of, 305
 ischæmia of, 117
 nerve injuries affecting, 433, 441
 rupture of, 305-306
 sling operation for incontinence, 1133

Myalgia, epidemic, 1229
 Myasthenia gravis, 572-573, 574
 Mycetoma pedis, 174
 Myelocoele, 353, 412
 Myeloma, multiple, 259, 652
 Myer's vein stripper, 148
 Myocardium, revascularisation of, 704
 Myositis, acute suppurative, 316
 clostridial, 44
 ossificans, 317
 complicating dislocation, 271
 'rheumatic', 316-317
 syphilitic, 317
 Myringotomy, 461
 Myxœdema, 540-541
 pretibial, 549
 Myxomatous degeneration, 138

N

Nævolipoma, 54, 104
 Nævus, 103-104, 107
 lymphatic, 153
 spider, 104, 807
 tardes, 104
 verrucosus, 101
 Naffziger's operation, 549
 Nasopharynx, 625
 tumours of, 625-628
 Naughton Dunn's arthrodesis, 350
 Neck, 518-536
 abscess of, 13
 actinomycosis of, 531-532
 block dissection of, methods, 535-536
 carcinoma of, cytotoxic drugs, 631-632
 secondary, 534-535
 cellulitis of, 15, 526-527
 hæmangioma of, cavernous, 532
 hygroma of, cystic, 520-521
 injuries of, 525-526
 lymphadenitis of, 528-531
 malignant tumours of, primary, 532-534
 neoplasms of, benign, 532
 solitary lymph cyst of, 521
 tuberculous nodes of, 528
 webbing, 522
 Necrosis, avascular, in fractures, 186
 of jaws, 472
 Nelaton's line, 218
 Nephrectomy, 1119-1120
 for calculus, 1094
 for hydronephrosis, 1087
 for hypertension in renal disease, 1106
 for renal neoplasms, 1078, 1115, 1118,
 1119-1120
 partial, 1094
 Nephroblastoma, 1114
 Nephrocalcinosis, 570
 Nephrolithotomy, 1093-1094
 Nephrostomy for calculous anuria, 1073
 Nephro-ureterectomy, in renal
 tuberculosis, 1112
 Nerves, 428-450
 anastomosis of, 439
 auditory, neurinoma of, 382
 autonomic, 449-450
 cervical, cutaneous distribution of, 442
 circumflex, injuries of, 443
 closed injuries, treatment of, 435
 Nerves—*contd.*
 compression of, 428, 430, 431, 436, 448
 concussion of, 434
 cranial, 428-433
 injuries of, 428-433
 in skull fracture, 370
 cutaneous, external, compression of, 448
 division of sensory root, 429-430
 dorsal, twelfth, injuries of, 448
 eighth, injuries of, 374, 432
 tumours of, 382, 391, 463
 eleventh, injuries of, 374, 433
 facial, 506, 513, 514
 injuries of, in skull fracture, 374
 fifth, injuries of, 374, 428-430
 fourth, injuries of, 374, 428
 friction of, results of, 446
 grafting of, 439
 ilio-inguinal, injuries of, 448
 indication for operations, autonomic
 nervous system, 449-450
 injection into Gasserian ganglion, 429
 injuries of, 185, 428-449
 closed, 435
 due to injections, 440
 incomplete division of, 440
 irremediable, treatment of, 439
 interosseous, posterior, injuries of, 445
 laryngeal, recurrent, 537
 injuries of, 432, 552
 paralysis of, 637-638
 median, compression of, 446
 injuries of, 185, 445-446
 paralysis of, 170
 musculospiral, injuries of, 443
 ninth, injuries of, 374, 432
 olfactory, injuries of, 374, 428
 optic, injuries of, 374, 428
 peripheral, injuries of, 434-440, 443-
 449
 phrenic, 440
 popliteal, injuries of, 448-449
 radial, injuries of, 443
 regeneration of, rates of, 436
 rupture of, 434
 sciatic, injuries of, 439, 448
 seventh, injuries of, 430-432
 sheaths, neurinoma on, 382
 sixth, injuries of, 374, 430
 spinal, injuries of, 440-442
 suture of, 438-440
 tenth, injuries of, 374, 432-433
 third, injuries of, 374, 428
 twelfth, injuries of, 433-434
 ulnar, anterior transposition of, 446
 injuries of, 446
 vagus, 728
 wounds, open, treatment of, 437-438
 Neuralgia, brachial, 442-443
 glosso-pharyngeal, 432
 lingual, 499
 trigeminal, 428-429
 Neurapraxia, 185, 434
 Neurectomy, presacral, 450
 Neurilemmoma, 532
 Neurinoma, 382
 Neuritis, brachial, 442-443
 diabetic, 285
 Neuroblastoma, 56, 584, 1114

Neurofibroma, 53, 57, 765
 macroglossia and, 494
 of chest wall, 652
 spinal, 414, 418
 Neurofibromatosis, generalised, 56
 plexiform, 56
 Neurogenic bladder, 1184
 Neurolipomatosis, 54
 Neuroma, 56-57
 acoustic, 385
 Neurotmesis, 185, 437-438
 Nitrogen metabolism, 1069
 Noble's plication operation, 951
 Noradrenalin, 76, 79, 583
 Nück, canal of, hydrocele of, 1037, 1224
 Nystatin, 162

O

Obsession, surgical treatment of, 400
 Occipital arteritis, 138
 lobe tumours of, 384
 Oddi, sphincter of, surgery of, 844, 857
 Odontomes, 473-474
 Oedema, lymphatic, 7, 152
 of arm, 608
 of hand, 160
 pulmonary, 674-675
 Oesophageal stenosis, 711
 transection, 814
 Oesophagectomy, for carcinoma, 725-726
 Oesophagitis, 714-715
 Oesophagocardiomyotomy, 722
 Oesophagoscopy, 709
 in achalasia, 722
 in carcinoma, 725
 in hiatus hernia, 716
 in oesophagitis, 714
 in peptic ulcer, 755
 in pharyngeal pouch, 624
 in varices, 810
 Oesophagus, 708-727
 abnormalities of, congenital, 710-711
 achalasia of, 721-723
 burns and scalds of, 712-713
 carcinoma of, 723-727
 cardiochalasia of, 731
 diverticulum of, 713
 foreign bodies in, 711-712
 hiatus hernia, 682, 714, 715-720
 injuries of, 712-713
 obstruction of, 720
 paralysis of, 713
 perforation of, 712
 ring contractions of, abnormal, 723
 rupture of, spontaneous, 713
 'short', 710, 715
 spasm of, muscular, localised, 720
 stricture of, 723
 tumours of, benign, 723
 ulceration of, peptic, 714
 varices of, 713, 810
 Oestrogen, 576, 1161
 stimulated carcinoma, 612, 613
 therapy, in carcinoma of breast, 613
 Oligodendro-glioma, 382
 Oligospermia, 1238
 Oliguria, 1067, 1068, 1169
 Ollier's disease, 253, 255
 Ombredanne's operation for maldescended testes, 1217
 Omentocoele, 1032, 1034-1035
 Omentum, greater, 883-884
 cyst of, 884, 890
 torsion of, 884
 Omphalitis, 1024
 Omphalocele, 1051
 Onychogryphosis, 175
 Oophorectomy, bilateral, for carcinoma of breast, 612-613
 Operation, prevention of infection, 6
 Ophthalmia neonatorum, 34
 Ophthalmoplegia, 548
 Orbit, cellulitis of, 14-15, 484-485
 decompression of, 548
 tumours of, 482-483
 Orbital undercutting, restricted, 400
 Orchiectomy, 1235
 for carcinoma of testis, 1235
 for epididymitis, 1231
 for incompletely descended testis, 1217-1218
 for neoplasm of testis, 1235
 Orchiopexy, technique of, 1216-1217
 Orchitis, leprosy and syphilitic, 31, 1231
 Oriental sore, 35
 Oropharynx, 625, 628-629
 Ortolani's sign, 334
 Os calcis, apophysitis of, 251
 fracture of, 227-228
 Osgood Schlatter's disease, 251
 Osteitis deformans, 246-248. *See also* Paget's disease of bones
 Osteitis fibrosa cystica, 248, 320, 570
 Osteitis pubis, 1169-1170
 Osteitis, tuberculous, 237-238
 Osteoarthritis, 297-302, 571
 of big toe joint, 346
 of cervical spine, 442
 of hip, 134, 300-301
 of knee, 300-301
 of Paget's disease, 247
 Osteochondritis, 235, 249-252, 327-328
 dissecans, 302
 Osteochondromas, 256, 651
 Osteoclasts in rickets, 244
 Osteoclastoma, 257-259, 354, 475
 Osteogenesis imperfecta, 249
 Osteoma, 256-259
 ivory, 256, 354, 474
 of orbit, 482
 of skull, 256
 osteoid, 264
 Osteomalacia, 245
 Osteomyelitis, acute pyogenic, 230-234
 acute, of vertebra, 413
 chronic pyogenic, 234
 in fractures, 185, 186
 of jaws, 471-472
 of skull, 353-354
 of terminal phalanx, 165
 syphilitic, 236-237
 typhoid, 232
 Osteopetrosis, 253
 Osteopokily, 253
 Osteoporosis, in Cushing's syndrome, 580
 tuberculous arthritis and, 287
 Osteosarcoma, 255, 260-262

- Osteotomy, derotation and innominate, for congenital dislocation of hip, 337
 for ankylosing spondylitis, 297
 for coxa vara, 339
 for genu varum, 340
 for hallux valgus, 347
 for osteitis deformans, 248
 for osteoarthritis, 299, 300
 for renal rickets, 244
 for rheumatoid arthritis, 297, 300-301
 subtrochanteric, 216
 Otitis, external, 460
 media, 461-462
 secretory, 462
 Otosclerosis, 462
 Ovary, arrhenoblastoma of, 581
 Oxycephaly, 353
 Oxygen therapy, 79-80

P

- Pacemakers, 704
 Pad sign, in carcinoma of pancreas, 864
 Paget's disease of bones, 246-248, 320, 328
 of jaws, 475
 of nipple, 589, 603, 606-607
 of penis, 1210
 Paget's quiet necrosis, 302
 recurrent fibroid, 60
 Pain, arteries, veins and, 117-118
 ischæmic, relief of, 123
 lumbar puncture in relief of, 426
 surgical relief of, 449-450
 visceral, 892
 Palate, lesions of, 456
 Pancoast tumours, 665
 Pancreas, 846-866
 aberrant, 765
 adenoma of, non-beta-cell, 861
 annular, 737, 847
 beta-cell tumour of, 860
 calculi of, 858
 carcinoma of, 856, 861-866
 congenital abnormalities of, 847-848
 cysts of, 858-859
 ectopic, 847
 fibro-cystic disease of, 847-848
 injuries to, 848-849
 insufficiency of, tests for, 848
 islet-cell tumours of, 860
 pseudo-cyst of, 858
 ulcerogenic tumour, 861
 Pancreatectomy, total, 866
 Pancreatitis, 849
 acute, 508, 849-854
 chronic, 854-858
 post-operative, 759-760, 854
 relapsing, chronic, 854-855
 Pancreato-duodenectomy, 857, 865-866
 Pancreato-jejunostomy, retrograde, 857
 Papilloma, 53, 101-102, 352, 492-493, 500, 1149-1150, 1209
 Paracentesis abdominis, 882
 thoracis, 713
 Paralysis agitans, 401
 facial, 431, 513, 514
 infantile, 350. *See also* Poliomyelitis
 laryngeal, 637-638
 spastic. *See* Little's disease, 265
 in spinal cord lesions, 409
 arthritis, 965
 araphimosis, 1205
 Parasyphilis, 283
 arathormone, in parathyroid tetany, 570
 arathyroidectomy, 571-572
 Parathyroids, 568
 adenoma of, 570
 carcinoma of, 572
 hyperplasia of, 570-572
 Paratyphoid fever, 910
 Paravertebral injections, 122-123
 Parietal lobe tumours, 384
 Parkinsonism, surgical treatment of, 401
 Paronychia, 161-162
 of great toe, 172
 Parotid, 506-517
 cysts of, 511
 fistula of, 449, 510-511
 neoplasms, 511-514
 sarcomoid, 515
 Parotidectomy, 507
 for parotid tumour, 513-514
 Parotitis, 508-510
 Parot's nodes, 236
 Patella, dislocation of, 275
 fractures of, 192, 222-223
 Paul-Bunnell reaction, 155
 Paul-Mikulicz operation, 937, 944
 PBI test, 539
 Peau d'orange, 605, 607-608
 Pectus excavatum, 650
 Pellagra, 489
 Pelvic abscess, complicating peritonitis, 873
 Pelvis, cellulitis of cellular tissue, 15
 chondroma of, 256
 deformities of, in rickets, 244
 fractures of, 213-214
 Pemphigus, syphilitic, 30
 Penicillin resistant organisms, 1, 2, 3, 33
 Penis, 1203-1212
 abnormalities of, congenital, 1203-1204
 amputation of, complete or partial, 1211-1212
 carcinoma of, 1210-1212
 inflammations of, 1205-1206
 injuries of, 1205
 neoplasms of, 1209-1210
 straightening of, in hypospadias, 1185
 strangulation of, by rings, 1205
 Pepper's neuroblastoma of adrenal medulla, 584
 Peptic ulcer, acute, 737-738, 754
 chronic, 738-742, 753-754
 complications of, 750-758
 diagnosis of, 740-745
 malignancy and, 740, 744, 752, 758
 pyloric stenosis, 756-757
 treatment of, 746-750
 complicating hyperparathyroidism, 571
 in Zollinger-Ellison syndrome, 739, 744, 860, 861
 of Meckel's diverticulum, 900
 of œsophagus, 714
 perforated, 751-753, 870, 961
 recurrent, 749, 750, 760
 Pericardectomy, 692
 Pericarditis, 691-692, 881
 Pericardium, 691-692
 Pericranitis, syphilitic, 354

- Peridental suppuration, 472
 Perineal operation for cancer, 1020
 Perineo-abdominal operation, 1018-1020
 Perineum, split, 984
 'watering can', 1199
 Perionychia, chronic, 162
 Perioritis, complicating venous ulcer, 150
 Perisplenitis, 779
 Peritheliomata, 60
 Peritoneal adhesions and bands, 880
 dialysis, 1071
 loose bodies, 882
 pain, 892
 Peritoneum, 867-883
 carcinoma involving, 52, 882-883
 neoplasms of, 882-883
 Peritonitis, 867-880
 acute, 867
 bile, 878
 complications of, 872-873
 diffuse, 830, 869, 870
 encysted, 880
 fibrous, 880
 generalised, 870-871
 localised, 869-870
 loculated, 880
 meconium, 875-876
 periodic, 878
 plastic, 880
 pneumococcal, 876-877
 post-operative, 877-878
 purulent, 880
 streptococcal, 261
 tuberculous, 878-880
 Perlèche, 490
 Perniosis, sympathectomy for, 140
 Perthes' disease, 250-251, 338
 test, 148
 Pes cavus, 343-344, 348
 planus, 340-343
 Peutz-Jeger's syndrome, 915-916, 917
 Peyronie's disease, 1209
 Pfannenstiel's incision, 614
 Phæochromocytoma, 583, 584-586
 Phagedena, 29
 Phantom pain, 43
 Pharyngeal pouch, 622-624
 Pharyngo-maxillary space infection, 527
 Pharyngotomy, lateral, with partial
 pharyngectomy, technique of, 628-629
 Pharynx, 625
 diverticulum of, 622-625
 neoplasma of, 625
 Phenindione, 131
 Phimosis, 1203
 Phlebothrombosis, 142, 143, 681, 767
 Phlegmon, peri-appendicular, 964
 Phrenic crush, 440
 Phrygian cap, 822
 Phytomendione, use of, 131
 Pick's disease, 691
 ascites in, 881
 perisplenitis in, 779
Pied de jeune soldat, 229
Pied forcé, 229
 Piles, 989. *See also* Hæmorrhoids
 'Pill', contraceptive, carcinoma and, 612
 Pilonidal sinus, 163, 982-984, 1025
 Pineal gland, tumours of, 385
 Pin-hole, urethral meatus, 1183
 Pinna, carcinoma of, 460
 Pituitary, 392-394
 stalk, section of, for exophthalmos, 549
 Pituitary-thyroid axis, 538
 Plasma, normal electrolyte values, 88
 prothrombin index as liver function
 test, 793
 treatment of burns, 111
 Plastic bags, for intravenous therapy, 89
 Platt's, classification of bone tumours, 255
 diagnostic triad, 288
 Pleura, diseases of, 652-659
 Plombage, 678
 Plummer's hydrostatic bag, 722
 Plummer-Vinson syndrome, 720-721
 Pneumatosis cystoides intestinales, 915
 Pneumaturia, 1141
 Pneumococci, 2
 Pneumonectomy, radical, technique of,
 667-668
 Pneumonia, aspiration, 679
 complicating cut throat, 526
 hypostatic, in fractures, 184
 in spinal injuries, 410
 Pneumothorax, 652-653
 artificial, 652
 spontaneous, 653
 traumatic, 648
 Poker man, 317
 Poliomyelitis, 140, 320, 343, 346, 348-350,
 618
 Polya operation, 747-748
 complications following, 760
 Polymazia, 590
 Polyorchism, 1213
 Polyp, fibro-epithelial, 493
 nasal, 480
 Polyposis familial, adenomatous, 917, 1012
 multiple colonic, 1012
 Porphyria, 786, 964
 Portal and systemic venous systems,
 anastomoses between, 809, 814
 blood pressure, measurement of, 812
 hypertension, 808-809
 au froid, 811
 gynæcomazia in, 615
 operation for, 813-815
 Porta-systemic encephalopathy, 792
 Porto-azygos disconnection with gastric
 transection, 815
 Portovenography, operative, 813
 Port wine stain, 103
 Proctitis, 1205
 Post-gastrectomy syndromes, 761-764
 Postural drainage, 359
 Potassium balance, 86-88
 Potato nose, 103
 tumour, 532
 Pott's disease, 238-241, 327
 abscess formation in, 239, 240
 fracture, 225-227
 operation for Fallor's tetralogy, 698
 paraplegia, 240, 242
 puffy 'tumour', 49, 376
 Prednisolone, in Hodgkin's disease, 158
 in pruritus ani, 997
 Pregnancy, after radical mastectomy, 611
 Presbycusis, 462

- Priapism, persistent, 1209
 Procidentia, 988-1000
 Proctalgia fugax, 987, 988
 Proctitis, 1001-1003
 Proctoclysis, 94
 Procto-colectomy, for ulcerative colitis, 908
 Procto-colitis, 1001
 Prognathism, mandibular, 455-456
 Prolapse, anus and rectum, 997-1000
 Proptosis, pulsating, 484
 Prostate, 1160
 abscess of, 1178, 1181
 calculi, 1171-1172
 carcinoma of, 263, 1161, 1172-1178
 treatment of, 264, 582-583, 1175-1178
 'decoy', 1164
 enlargement of, benign, 1161-1170
 fibrous, 1168, 1170-1171
 median bar obstruction, 1168
 tuberculosis of, 1180
 Prostatectomy, 1167-1170
 transurethral resection, 1168, 1171, 1172
Prostatism sans prostate, 1170-1171
 Prostatitis, 32, 1178
 prostatic calculi and, 1171
 tuberculous, 1180
 Protein-bound iodine test, 539
 Prothrombin, plasma index, 793
 time, 76
 Pruritus ani, 987, 996-997
 Pseudo-coxalgia, 250-251, 338
 Pseudo-hydronephrosis, pararenal, 1083
Pseudomonas pyocyanea, 2, 4
 Pseudo-myxoma peritonei, 883
 Psoas bursa, 1049
 Pulmonary embolism, 79, 130, 680-681
 oedema, 674-675
 stenosis, 695
 ventilation, in cardiac arrest, 681
 Pulseless disease, 138
 Pupils, in cranio-cerebral injuries, 359, 367
 Purpura, 782-783
 thrombocytopenic, 783-785
 with intestinal symptoms, diagnosis
 from intussusception, 941
 Pustule, malignant, 11
 Putti-Platt operation for recurrent
 dislocation of shoulder, 270
 Pyæmia, 15-16, 232
 portal, 15, 796-797, 992
 Pyelitis, 1100
 Pyelography, excretory, 1077, 1086, 1092,
 1097, 1105, 1110, 1117, 1139, 1152
 infusion, 1063
 instrumental, 1064-1065
 intravenous, 1062-1063
 retrograde, 1064-1065, 1077, 1087,
 1092, 1097, 1111, 1117
 Pyelolithotomy, 1093
 Pyelonephritis, 1100
 acute, 963, 1062, 1100, 1103, 1169
 chronic, 1103-1105
 Pyelonephrolithotomy, 1094
 Pylephlebitis, 142, 796-797, 992
 diagnosis from subphrenic abscess, 875
 Pyloric stenosis, hypertrophic, of adults, 733
 complicating peptic ulcer, 756
 of infants, 730-733
 Pyloroplasty, 749
 with vagotomy, for perforated peptic
 ulcer, 752, 756
 Pylorus, function of, 728-729
 Pyonephrosis, 1106
 Pyorrhœa alveolaris, 468-469
 Pyuria, abacterial, 1113
 in cystitis, 1143
 renal calculus and, 1091
 tuberculosis and, 1109
- O
- Quadriceps extensor, rupture of, 305
 Queckenstedt's test, 417, 442
 Quercat, erythroplasia of, 1210
 Quinsy, 620-621
- R
- Radial bursa, infection of, 169
 groove, injury in, 444
 Radiation, necrosis, 472
 Radioactive cobalt therapy, 1155
 chromic phosphate, 883
 gold, 883, 1154, 1203
 tantalum wire, 1154, 1211
 Radio-iodine studies of thyroid function, 538
 treatment of carcinoma of thyroid, 563
 See also Iodine, radioactive
 Radiotherapy, carcinoma of thyroid from, 560
 of acromegaly, 394
 of ankylosing spondylitis, 297
 of basophil adenoma, 395
 of anus, 1022
 of bladder, 1154-1155
 of carcinomatous epulis, 470
 of breast, 611-612, 614
 of bronchus, 668-669
 of chromophobe tumours, 393
 of epithelioma, 102
 of glomus jugulare tumours, 462
 of Kaposi's pigmented sarcoma, 110
 of kidney, 1115, 1118
 of larynx, 642, 643
 of lung, 668-669
 of maxilla, 477
 of œsophagus, 726-727
 of osteoclastoma, 258
 of Paget's disease of penis, 1210
 of penis, 1210, 1211
 of reticulosos, 157
 of rodent ulcer, 106
 of testis, 1236
 of tongue, 497, 505
 of urethra, 1203
 of Wilms's tumour, 1115
 Radius, congenital absence of, 330
 dislocation of head, 272
 fractures of, 204-210
 Ramstedt's operation, 732-733
 Randall's plaque, 1089
 Ranula, 491-492
 Rathke pouch tumour, 395
 Raynaud's disease, 139
 phenomena, 139
 von Recklinghausen's disease of bone,
 248, 324, 570
 of nerves, 56, 494

- Rectal infusion, continuous, 94
 Recto-sigmoid, strawberry lesion of, 1002
 Rectum, 975-1022
 abnormalities of, congenital, 979-982
 abscesses of, 1003-1006
 carcinoid of, 1021
 carcinoma of, 1008, 1014-1021
 examination of, 978
 foreign bodies in, 985
 incontinence of, 1000-1001
 inflammation of, 1001-1003
 injuries of, 984-985
 papilloma of, 1013, 1014, 1017
 prolapse of, 997-1000
 diagnosis from intussusception, 941
 stricture of, 971, 1010-1011
 tuberculosis of, 1007-1008
 tumours of, benign, 1011-1013
 malignant, 1014-1021
 Rectus femoris, rupture of, 305-306
 'Red currant jelly' stool, 940
 Reed, Dorothy, giant cells, 156, 575
 Reflex heating, 122
 Reiter protein complement-fixation test, 28
 Reiter's disease, 1192-1193
 Renal arteriography, 119, 1066-1067
 artery stenosis, 137
 dwarfism, 571
 function tests, 1062
 pain, 450, 1061
 Resin-uptake test, 539-540
 Rest pain, 118, 134
 Resuscitation, cardio-respiratory, 687-689
 Reticulosarcoma, 60, 157, 563
 Reticuloses, 155-159
 Retinal embolism, 129
 Retinoblastoma, 483
 Retroperitoneal cyst, 890-891
 fibrosis, idiopathic, 1099
 lipoma and sarcoma, 891
 lymph nodes, block dissection of, 1236-1237
 neoplasms of, 891
 space infection, 890
 Rheomacrodex, 81
 Rh factor, 73-74
 Rhinophyma, 103
 Rib, cervical, 522-524
 fractures of, 212-213, 647
 tumours of, 651-652
 Richter's hernia, 929, 1032, 1034, 1049
 Rickets, 242-244, 320
 renal, 244, 320, 340, 571
 Rickety rosary, 243
 'Rider's bone', 317
 Riedel's thyroiditis, 565
 RISA, 69
 Risser jacket, 326
 Risus sardonius, 8
 Rodent ulcer, 37, 105-107, 115
 Rogitine, 585
 von Rosen's sign, 334
 Rosenthal's syndrome, 76
 Rovsing's operation, 1077
 Rowbotham's operation, 548-549
 Rutherford Morison's kidney pouch, 874
 Rutherford's operation, 1190
- S
- Sabin vaccine, 348
 Sacral puncture, 427
 Sacro-coccygeal teratoma, 981
 Sacro-iliac tuberculosis, 292
 St. Peter's Hospital suprapubic drainage apparatus, 1157
 Saint's triad, 716, 836
 Saline solutions, 89
 Salivary glands, 493, 506-517
 tumour, ectopic, 457, 516-517
 Salk vaccine, 348
 Salmon patch, 103
 Saphena varix, 1048
 Sarcoid, 103, 158, 397
 diagnosis from enchondromas, 256
 of parotid, 515
 of stomach, 769
 Sarcoma, 51, 58-60, 259-262, 354, 475
 osteogenic, 255, 260-262, 476
 See also under names of organs and regions
 Scalds and burns, 110-114
 Scalene node biopsy, 666
 syndrome and cervical rib, 139, 522-524
 Scalp, 351-352
 cellulitis of, 14
 Scaphoid, fractures of, 211
 Scapula, winging of, 443
 fractures of, 199
 Scars, chronic, 105
 Scheuermann's disease, 251, 327-328
 Schmorl's node, 422
 Schneeberg cancer, 664
 Schüller-Christian's disease, 253
 Schwannoma, 532
 Scleroderma, 139
 Sclerosis, diffuse, in syphilis, 237
 Scoliosis, 244, 322-326
 Scrotum, 1213, 1214, 1237-1240
 carcinoma of, 1239-1240
 elephantiasis of, 1238
 idiopathic gangrene of, 1237
 Scrum-pox, 99
 Scurvy, 240, 245, 489
 Sebaceous cysts, 100-101, 1238
 horn, 101
 Secretin-pancreozymin test, 855-856
 Seldinger needle, 120, 1067
 Seminal vesicles, 1160-1161, 1181-1182
 Seminoma, 1232, 1237
 Sengstaken tube, 70
 Septicæmia, 1, 15, 232
 parotitis as complication, 508
 Septum, nasal, deflected, 481
 Serum, acid phosphatase, 1174
 albumin estimation, 793
 alkaline phosphatase, 793
 amylase estimation, in pancreatitis, 851, 855
 raised, abdominal pain with, 852
 anti-gas gangrene, 45
 anti-tetanic, 9
 bilirubin estimation, 793
 cholesterol test, 540
 reactions, 10-11, 78
 transaminase, 793
 Sever's disease, 251

- 'Sheepskin', in protection of pressure areas, 42, 46
- Shenton's line, 334, 336
- Shock, 77-79, 86, 91
 - in burns, 110, 111-112
 - in fractures, 179, 184
 - oligæmic, 66-67
 - spinal, 405
- Shoulder, congenital elevation of, 330-331
 - joint, dislocation of, 267-270
 - fracture-dislocation of, 270-271
 - tuberculosis of, 294
 - periarthrititis of, 309-310
 - snapping, 303
- Shoveller's fracture, 403
- Sialectasis, 507
- Sialography, 507, 508, 509, 510, 514
- Sigmoid, carcinoma of, 920
- Signe de Dance, in intussusception, 940
- Simmond's disease, 396
- Singapore ear, 460
- Singer's nodules, 640
- Sinus, 20-21
 - frontal, compound fractures of, 371-372
 - median mental, 472-473
 - pilonidal, 982-984
 - piriformis, neoplasms of, 629
- Sinusitis, maxillary, 478-479
- Sister Joseph's nodule, 1027
- Sistrunk's operation, 567
- Sjoqvist intramedullary tractotomy, 430
- Skin, carcinoma of, 105-107
 - effects of irradiation, 105
 - grafting, 114-116
 - infections of, 98-101
 - malignant disease of, 105-110
 - premalignant conditions of, 105
- 'Skip' areas, in gangrene, 119
- Skull, 353
 - carcinoma of, secondary, 263, 354
 - fractures of, 368-373
 - inflammation of, 353-354
 - 'map like', 253
 - new growths of, 354
 - osteomyelitis of, 353-354
 - Paget's disease of, 247
 - tuberculous disease of, 238, 354
 - 'worm eaten', 236
- Smith's fracture, 205-206
- Smith-Petersen's 'cup' arthroplasty, 300
- nail, 215-216
- Smoking, in ætiology of bronchial carcinoma, 57, 664
 - of duodenal ulcer, 739
 - of glossitis and leukoplakia, 496
 - in arterial disease, 122, 134, 138
 - pulmonary post-operative complications and, 678
 - tongue and, 495, 496
- 'Snail-track' ulcers, 498
- Snake bites, 18
- Snapping hip and tendon, 303
- Snuffles, in syphilis, 30, 235
- Socia parotidis, 507
- Sodium balance, 84-86
- Soft sore, 31
- Souttar's tube in œsophageal carcinoma, 727
- Spasm, peroneal, 342
- Speckled bones, 253
- Spermatic cord, encysted hydrocele of, 1224
 - torsion of, 1218-1220
- Spermatocele, 1227
- Spherocytosis, hereditary, 780-781
- Sphincterotomy, for anal fissure, 988
 - transduodenal, 856-857
- Spider nævus, 104
- Spigelian hernia, 1028, 1058
- Spike operation for hammer-toe, 348
- Spina bifida, 411-413, 1184
- Spinal canal, tumours of, 414-419
 - concussion, 405
 - cord injuries, 404-411
 - rehabilitation, 410
 - shock, 405
- Spine, 402-427
 - bamboo, 296
 - deformities of, 322-329
 - dislocations of, 402-403
 - fractures of, 403-404
 - gunshot wounds of, 411
 - osteomyelitis of, acute, 413
 - osteoporosis of, acute, 328
 - sarcoma of, primary, 413
 - secondary deposits in, 327, 413
 - sprains of, 402
 - tuberculous disease of, 238. *See also* Port's disease
 - tumours of, 413-414, 442
- Spinotalamic tractotomy, 420-421
- Splanchnicectomy, in pancreatitis, 857
- Spleen, 773-790
 - abscess of, 789
 - congenital absence of, 775
 - cysts of, 779
 - embolism of, 130
 - enlargements of, 778-779
 - functions of, 773
 - hypertrophy, following splenectomy, 773
 - infarction of, 778
 - malarial, rupture of, 777
 - methods of investigating, 774-775
 - movable, 775
 - neoplasms of, 789
 - rupture of, 775-777
 - tuberculosis of, 789
 - wandering, 775
- Splenectomy, 779, 790
 - in Egyptian splenomegaly, 787
 - in Felty's syndrome, 788
 - in hæmolytic ænæmia, 781-782
 - in hypersplenism, 789
 - in portal hypertension, 813
 - in purpura, 785
 - physiological effects of, 773-774
- Splenic artery, aneurysm of, 777-778
 - pedicle, torsion of, 775
 - puncture, 774
 - vein, occlusion of, 788
- Splenomegaly, Egyptian, 786-788
- Splenoportography, 774-775, 811-812
- Spleno-renal anastomosis, technique of, 814
- Splenosis, 774
- Splenunculi, 774
- Spondylitis, 442
 - ankylosing, 296-297, 328
 - tuberculous, 238-241

- Spondylolisthesis, 329
 Spondylosis, cervical, 425
 Spongioblastoma polare, 383
 Sprains, 265, 402
 Sprengel's shoulder, 330-331
 Stamm's osteotomy for hallux valgus, 347
 Stapedectomy, in otosclerosis, 462
 'Stave' fracture, 211
 Steatorrhoea, 571, 763, 855
 in chronic pancreatitis, 854
 in Crohn's disease, 911
 in mucoviscidosis, 847
 jejunal diverticulosis and, 899
 Steeple head, 353
 Steindler operation, for pes cavus, 343
 Stenosis-anastomosis loop syndrome, 897-898
 Stenosis, oesophageal, 711
 pulmonary, pure, 695
 pyloric, hypertrophic, 733
 renal artery, 119
 valvular, 695
 Stensen's duct, 506, 508
 Stercolith, causing intestinal obstruction, 948
 Sterilisation, hospital infection and, 6
 Sterility, male, causes of, 1181, 1216
 Sternberg-Reed giant cells, 156
 Sternal-clavicular joint, dislocation of, 267
 Sternomastoid 'tumour', 321-322
 Sternum, fracture of, 213
 Steroid therapy, infection and, 1
 Stilboestrol, 1126
 Still's disease, 296
 Stomach and duodenum, 728-772
 Boeck's sarcoid of, 769
 carcinoma of, 765-768
 following ulcer, 765
 dilation of, acute, 734, 777
 foreign bodies in, 733-734
 giant rugal hypertrophy of, 737
 handbag, 757
 hour-glass, 742, 757-758
 leather-bottle, 768-769
 neoplasms of, benign, 764-765
 photography of, colour, 745
 sarcoma of, 772
 syphilis of, 737, 768
 'teapot', 757
 ulcer of, 737. *See also* Peptic ulcer
 volvulus of, 735-736
 Stomas, cutaneous, 1157
 Stomatitis, 486-489
 monilial, 12, 487
 'Stork bites', 103
 Stove-in-chest, 212, 646, 647-648
 Strangury, 1091, 1109
 'Strawberry angioma', 103-104
 Strawberry gallbladder, 833
 Struma fibrosa, 565
 lymphomatosa, 564
 ovarii, 566
 Student's elbow, 318
 Stryes, 1, 99, 481
 Subacromial bursitis, 310
 Subclavian artery stenosis, 137
 steal syndrome, 137
 Subglottic carcinoma, 641
 Submandibular gland, tumours of, 516
 Subphrenic abscess, 753, 873-874
 Subtentorial tumours, 385
 Sudeck's osteoporosis, 207
 Sulkowitch test, 571
 Sulphonamides, 3, 1102-1103
 crystalluria, anuria due to,
 use of, as intestinal disinfectant, 3, 5
 Supraglottic carcinoma, 641
 Supraspinatus tendinitis, 310
 Surgery, arterial, 117-141
 cardiac, 689-691
 Swab weighing, in estimation of blood
 loss, 68
 Swimmer's itch, 1146
 Sympathectomy, 121, 136, 139, 140
 cervico-dorsal, 704
 chemical, 123
 lumbar, 140-141
 pre-ganglionic cervico-dorsal, 140
 renal pedicle, 1088
 Syndactyly, 331
 Synovioma, 60, 314
 Synovitis, syphilitic, 282-283
 traumatic, 279
 Syphilis, 27-31
 ano-rectal, 987, 1002
 congenital, 30-31
 of bones, 235-237
 of hard palate, 472
 of lymph nodes, 155
 of tongue, 28, 494, 497-499
 specific serum test in, 27-28
 treatment of, 3, 30
 *See also under names of organs and
 regions*
 Syringomyelia, 162, 284
 Syringo-myelecele, 412

T

- T₃ test, 539-540
 Tabes, crises of, 963
 Takayasu's arteriopathy, 138
 Talc granuloma, 880
 Talipes, 150, 344-346
 Tamponade, cardiac, 691
 for oesophageal varices, 810-811
 Tanner's operation for peptic ulcer, 749
 slide, 1042, 1051
 Tantalum gauze, in inguinal hernia, 1040,
 1041
 Tantalum-wire, radioactive, 1154, 1211
 Tarsals, fractures of, 227-229
 Tarsorrhaphy, in exophthalmos, 548
 Teeth, 464-468
 Telephonist's ear, 470
 Temporal arteritis, 138
 Temporal lobe tumours, 384
 Temporomandibular joint, dislocation of,
 266-267
 Tendon sheaths, infections of, 311
 tumours of, 314
 Tendons, collagen degeneration in, 309
 cut, 314-315
 displacement of, 309
 grafting of, 315
 injuries of, 305-319
 lengthening, 315-316
 operations on, 314-316

- Tendons—*contd.***
 rupture of, 207, 306–308
 shortening, 316
 snapping, 303
 supraspinatus, calcification of, 311
 rupture of, 310–311
 suture of, 315
 transplantation, 316
 in infantile paralysis, 350
 in nerve injuries, 439
- Tendovaginitis, stenosing, 312**
- Tennis elbow, 308**
- Tennison's operation for cleft-lip, 453**
- Tenosynovitis, acute suppurative,**
 168–170, 311
 simple, 311
 stenosing, 312
 tuberculous, 311–312
- Tenotomy, 315–316**
 in infantile paralysis, 350
- Teratoma, 50, 57, 62, 685**
 of testis, 1233–1237
 sacrococcygeal, 981
- Testis, abnormalities of, congenital, 1213**
 ectopic, 1213, 1218
 imperfect descent of, 1213–1214
 incompletely descended, 1214–1218
 inflammation of, 1227
 injuries of, 1218
 inversion of, 1213, 1218
 maldescended, 1213, 1218
 neoplasm of, 1231–1237
 and old clotted hæmatocele, 1226
 retractile, 1216
 supernumerary, 1213
 syphilis of, 1231
 torsion of, 1218–1220
- Tetanus, 5, 7–10**
- Tetany, 569**
 gastric, 735
 parathyroid, 568–570
- Tetracycline resistant organisms, 1, 3**
- Thalassæmia, 785–786**
- Thiersch's method of skin grafting, 114**
 operation for prolapse of rectum, 998, 999, 1000
- Thiopentone, intra-arterial injection of,**
 causing gangrene, 48
- Thiouracil, in goitre, complications, 545**
- Thomas's knee splint, 221**
 test for fixed flexion deformity, 293
- Thompson prosthesis, 216**
 punch, 1169
- Thomson's adenoid curette, 620**
- Thomson-Walker's urethrotome, 1196**
- Thoracic duct, wounds of, 526**
- Thoraco-abdominal wounds, 649–650**
- Thoracoplasty, technique of, 676–677**
- Thoracotomy, for carcinoma, 727**
 for congenital oesophageal atresia, 711
- Thornburn's gravitation paraplegia, 403**
- 'Three slings' method for treating fracture**
 of clavicle, 198–199
- Thrombectomy, 131, 143**
- Thrombo-angiitis obliterans, 121, 122,**
 134, 137–138
- Thrombo-endarterectomy (rebore), 136**
- Thrombo-phlebitis, 142, 680**
 complicating varicose veins, 149
- Thrombo-phlebitis—*contd.***
 migrans, 142, 864
 of cavernous sinus, 458, 485
 of superficial veins of breast, 595–596
- Thrombosis, 129–132**
 causing gangrene, 47
 of aortic bifurcation, 706–707
 of axillary artery, 131
 vein, 144
 of mesenteric vessels, 948–949
 of popliteal artery, 124
 of portal vein, 808
 pulmonary, 680
 venous, 141–144, 910
- Thrush, 12, 487**
- Thyrectomy, in myasthenia gravis, 574**
- Thymoma, 575**
- Thymus, 572–575**
 cysts of, 574
 hyperplasia of, 572
 tumours of, 574, 685
- Thyroglossal tract, anomalies of, 566–567**
- Thyroid gland, 537–567**
 adenoma of, 549–550, 555
 carcinoma of, 559–563
 secondary, 264, 561
 ectopic and aberrant, 565
 enlargements of, 542–559
 function tests, 538–540, 541
 histological surprise, 562
 inflammations of, 565
 intrathoracic aberrant, 566
 lateral aberrant, 560, 566
 lingual, 565
 long acting stimulator, 538
 median ectopic, 565
 neoplasms of, secondary, 563
 physiological hyperplasia of, 553
 reticulosarcoma, primary, of, 563–564
 scanning of, 539
 thyroglossal ectopic, 565
- Thyroidectomy, 550–553**
 subtotal, technique of, 551–552
 pretibial myxoedema after, 549
 tetany after, 568–570
 total, 562
- Thyroiditis, 564–565**
- Thyrototoxic crisis, postoperative, 549**
- Thyrototoxicosis, 543–550**
 ætiology of, 538
 diagnostic test, 539
 factitia, 538
 multinodular goitre, 550
 primary, 543–549
 radioactive iodine therapy and, 549
 secondary, 549–550
 due to iodine, 550
- Tibia, curvature of, 236, 244**
 fractures of, 68, 223–224
 overgrowth and curvature of, syphilitic, 236
- Tic douloureux, 428–429**
- Tieberg-Wiesebach syndrome, 139**
- Tietze's disease, 650**
- Tinea pedis, 34**
- Tinel's sign, 436**
- TNM classification of carcinoma of**
 breast, 52–53
- Toe-nail, ingrowing, 175**

- Tomography, 821
 renal, 1067
- Tongue, abnormalities, 493, 565
 angioma of, 500
 black hairy, 495-496
 burning, 500
 carcinoma of, 57, 499, 501-506
 'caviare' lesion of, 500
 congenital fissure or furrowing, 494
 excision of, operations for, 503-505
 furring of, 495
 geographical, 498
 hairy, 495-496
 hemiatrophy of, 433, 500
 inflammations of, 495-499
 injuries of, 494
 lipoma of, 493, 500
 lymphangioma of, 494
 myoblastoma of, 500
 neoplasms of, benign, 500
 malignant, 501-506
 nerve lesions of, 499-500
 osteoma of, 500
 sarcoma of, 506
 smooth sore, 489
 syphilis of, 28, 494, 497-499
 tuberculosis of, 498
 ulcers of, 38, 499
- Tongue-tie, 493
- Tonsils, 617
 tumours of, malignant, 620
- Torkildsen's operation, 396
- Torticollis, 321-322
- Tourniquet, test, in purpura, 782-783
 use of, 70-71, 81
- Towne's view of cerebral abscess, 380
- Trachea, compression of, 545, 555
 scabbard, 555
 wounds of, 525
- Tracheostomy, 634-636, 646-647
- Traction and counter-traction, in treatment
 of fractures, 219-222
- Tractotomy, stereotactic, 400
- Transurethral resection of prostate, 1168,
 1171, 1172
- Trench foot, 47
- Trendelenburg's position, 71, 132
 sign, 335-336
 test in varicose veins, 146
- Treponema immobilisation test, 28
- Trichiniasis, causing myositis, 63, 317
- Trichobezoar, 734
- Trichomonas vaginalis*, urethritis due to,
 1192
- Trigger finger, 312
- Trismus, impacted tooth causing, 464
- Troissier's sign, 767
- Trotter's incision, 477
 triad, 627
- Trousseau's sign, 142, 569, 767
- Trusses, 1038, 1046, 1050
- Tuberculosis, 23-24, 151
 ano-rectal, 987, 1002, 1007-1008
 chemotherapy, 23, 291
 cutaneous, 100
 empyema in, 659
 lingual, 498
 of bones and palate, 237-242, 472
 periosteal, 238
- Tuberculosis—*contd.*
 pulmonary, surgical treatment, 675-678
 gastrectomy and, 764
 senile, 295
See also under names of organs and regions
- Tumours, 49-61
 benign, 50, 53
 malignant changes in, 60-61
 brown, 570
 giant-cell, 257-259
 malignant, 50-53, 57-61
 methods of spread, 50-52
 melanocytic, 107-108
 mixed parotid, 512, 513
 pulsating, 126
 raspberry, 1026-1027
 teratoma, 50-52
 'turban', 106
See also under names of organs and regions
- Turner's syndrome, 522
- Tympanoplasty, 462
- Typhlitis, amœbic, 909
- Typhoid fever, complications of, surgical, 910
 osteomyelitis and, 232
 treatment of carriers, 832

U

- Ulcer, anastomotic, 760
 clinical examination, 37-38
 decubitus, 46
 differential diagnosis of, 37-38
 gastro-jejunal, 760
 indolent, treatment of, 37
 jejunal, 760
 non-specific, 36
 perforating, 36
 post-thrombotic, 149
 rodent, 37. *See Rodent ulcer*
 tongue, 38, 499
 trophic, 36
 tuberculous, of ileum, 913
 of tongue, 38, 498
 venous, 29, 149
- Ulceration, 36-38
- Ulna, fractures of, 208-210
- Ulnar bursa, infection of, 168-169
- Umbilicus, 1023-1027
- Urachus, patent, 1026
- Uræmia, 1072
 in cystic kidneys, 1077
 paralytic ileus in, 950
- Ureter, abnormalities of, 1079-1080
 accidental ligation of, 1073
 calculus in, 570, 1059-1099
 injuries to, 1083-1084
 neoplasms of, primary, 1119
 transplantation of, 1073, 1123
- Ureteroceles, 1080
- Uretero-colic anastomosis, 1158-1159
- Uretero-lithotomy, 1098
- Ureterostomy, cutaneous, 1157
 intubated, 1088
- Uretero-trigonitis, 1143-1144
- Urethra, 1160, 1183-1203
 abnormalities of, congenital, 1183-1186
 angioma of, 1200

Urethra—*contd.*

- calculus of, 1199–1200, 1202
 - carcinoma of, 1200–1201, 1203
 - caruncle of, 1202–1203
 - diverticula of, 1198, 1202
 - false passages, 1194
 - fistula of, 1199
 - injuries of, 1186–1191
 - intrapelvic, rupture of, 1187–1189
 - meatus of, ulceration, 1191
 - neoplasms of, 1200
 - papilloma, 1200
 - papillomas acuminata, 1203
 - prolapse of, 1202
 - prostatic, calculi in, 1199–1200
 - reconstruction of, 1185–1186
 - rupture of, 1186–1191
 - strictures of, 1193–1198, 1202
 - congenital, 1183
 - post-prostatectomy, 1197
 - valves of, congenital, 1183–1184
- Uretero-sigmoidostomy, 1157–1158**
- Urethritis, 32, 1179, 1191–1192, 1201–1202, 1228**
- Urethrocele, 1198, 1202**
- Urethrography, 1066–1067, 1194**
- Urethroscopy, anterior and posterior, 1065–1066**
- in diagnosis of prostatitis, 1180
 - of stricture, 1194
- Urethroplasty for stricture, 1196–1197**
- Urethrostomy, perineal, 1185**
- Urethrotomy, 1196, 1200**
- Urinary affections, symptoms of, 1061–1062**
- instrumental examination, 1063–1064
 - investigation, 1062–1067
- Urine, alkalinisation of, 1094, 1102**
- antibiotics, 3, 1103
 - concentration dilution test, 1062
 - diversion of, operative, 1123, 1155–1159
 - examination of, 1062, 1100, 1110, 1135, 1147, 1151, 1179
 - extravasation of, following rupture of urethra, 1190–1191
 - 17-hydroxycorticosteroids excretion test of Addison's disease, 578
 - incontinence of, 1131–1132, 1169
 - in porphyria, 786
 - normal output, 82
 - reflux of, 1158
 - residual, 1130
 - retention of, 1126–1128
 - due to drugs, 1129
 - in prostatic enlargement, 1163
 - in spinal cord lesion, 408, 1129
 - postoperative, 1129
 - pyelonephritis with, 1101
 - with overflow, 1128, 1164
 - suppression of, 1067–1073
 - water loss by, 82–83

V

- Vaccinia, accidental, and anthrax, 12**
- Vaginitis, monilial, 12**
- Vagotomy, for peptic ulcer, 747, 748, 752, 756**
- Valgus and varus, meaning of, 329–330**

- Valvotomy, in Fallot's tetralogy, 698, 700–702**
- Varicocele, 1220–1222**
- Varicose veins, 144–150**
- Vascular rings (Dysphagia lusoria), 707**
- Vasectomy, 1167**
- Vasodilator drugs, 122**
 - methods, 122–123
- Vaso-epididymal anastomosis, 1239**
- Vasospastic conditions, 139–141**
- Veins, injuries to, 185**
 - thrombosis of, 141–144, 910
 - varicose, 144–150
 - ruptured, treatment of, 71, 150
- Venodysplasia, 90**
- Venous pressure, 69**
- Ventricle, third, tumours of, 385**
 - intraventricular tumours blocking, 395–396
- Ventricular failure, right, in blood transfusion, 75**
- Ventricular septal defect, 696**
- Ventriculography, in cerebral abscess, 380**
 - tumour, 387
 - in hydrocephalus, 388
- Vermiform appendix, 953–974. *See also* Appendix**
- Verruca vulgaris, 102**
- Vesiculitis, seminal, 963, 1181**
- Vincent's angina and gingivitis, 468, 512**
- Vitamin deficiency in aetiology of renal calculus, 1088**
 - of stomatitis, 486
 - teeth and, 465
- B, 489**
 - deficiency, after gastrectomy, 764
- C and scurvy, 245, 489**
- D treatment of celiac rickets, 245**
 - of fibrocystic disease of pancreas, 848
 - of hypoparathyroidism, 570
 - of osteomalacia, 245
 - of resistant rickets, 244
- K₁ treatment, 131**
 - requirements, 93
- Vitello-intestinal duct, 1023, 1025–1026**
- Vocal cord, carcinoma of, 641**
 - polyp of, 639
- Volemetron apparatus, 69**
- Volkman's contracture, 186–187**
- Volvulus, 943–944**
 - neonatorum, 937
 - of midgut, 937
 - of pelvic colon, 943–944
 - of stomach, 735–736
 - of small intestine, 943
- Von Rosen sign, 334**
- V-Y repair of cleft-palate, 453–454**

W

- Waldcyer's rings, 617**
- Warthin's tumour, 511–512**
- Warts, 98, 102**
 - anal, 996, 1021
 - 'gonorrhœal', 32–33
 - venereal, 1209
- Wasp stings, 17, 18**
- Wassermann reaction, 27**

Water balance, 82-84
 in anuria, 1069
 in burns, 112
 cold, treatment of burns, 113-114
 intoxication, 83-84
 Waterhouse-Friderichsen syndrome, 577
 Wax in ear, removal of, 460
 Weaver's bottom, 318
 Wens, 100, 352
 Werner's test, 539
 Wharton's duct, 515, 516
 Wheelhouse's operation for stricture, 1196
 Whipple's triad, 860
 'White bile', 838
 Wilm's tumour of kidney, 584,
 1114-1115
 Wilson's disease, 808
 Wolfe skin graft, 114-115
 Woollsorter's disease, 12
 Wounds, 1, 4, 16-18, 437

Wrist drop, 200
 joint, dislocation of, 272-273
 injuries of, 210, 446, 447
 tuberculosis of, 288, 295
 Wry neck, 321-322

X

X-ray diagnosis. *See various conditions*
 treatment. *See various diseases*

Y

Yaws, 31
 Young's perineal prostatectomy, 1168, 1177

Z

Zollinger-Ellison syndrome, 739, 744,
 860, 861
 Zygomatic arch, fracture of, 195